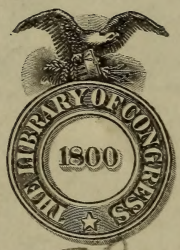

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A TREATISE

ON THE

PRINCIPLES AND PRACTICE OF MEDICINE

BY

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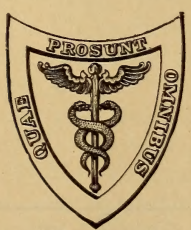
SECOND AND THOROUGHLY REVISED EDITION

Illustrated with 100 Engravings and 21 Plates



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PREFACE TO THE SECOND EDITION.

The demand for a new edition of a work in so highly developed a field of medical literature is both a commendation and a summons to improvement. The author has spared no effort in complying. The real advances throughout this enormous and active domain have been incorporated, and the whole work has moreover been virtually rewritten to secure increased brevity and clearness. The result is indicated by the fact that a vastly greater amount of information is furnished in a space decreased by seventy pages.

A few of the additions and changes may be mentioned. Particular attention has been given to therapeutic details in accordance with the recent awakening of the profession to the importance of logical treatment; numerous new preparations, and modified names and dosages, particularly for children, are explicitly specified. There are practically new chapters on the arrhythmias and other cardiac neuroses, tropical splenomegaly and various other tropical affections. Due consideration has been given to the meningitis serum of Flexner and Jobling, Strong's work on amœbic dysentery, blood cultures in typhoid and other bacteriæmias, the "carriers of infection," the recent epidemics of meningitis and poliomyelitis, the accumulating evidence in favor of the spirochæte as the cause of syphilis and the recent status of tuberculin in its therapeutic application and in its various diagnostic uses, as the ophthalmo-reaction, and von Pirquet's test. New plates are introduced, illustrating the diphtheria bacillus and its cultural appearance and also the spirochæte pallida and refringens.

The chapters on tuberculosis and syphilis are designedly amplified, as these diseases touch every organ, enter every specialty, and attain as great sociological as medical importance; indeed, an understanding of these maladies is almost an understanding of medicine. Recent statistics of case reports have been incorporated. Attention has been given to numerous criticisms and many minor alterations have been made in the interest of logic, clarity and conciseness.

So wide is the range of Modern Practice that an author who would cover it in a single volume, and a reader who would grasp it intelligently, must approach their respective tasks with the aid of *thorough system*. An effort has been made to deal with the subject in this manner, from its main divisions, which follow the most rational classification, down to the subordinate paragraphs. Careful use has been made of types of various prominence to facilitate the finding of a topic and the appreciation of its importance. With the vast mass of material, an attempt has been made so to arrange the facts that the

reader may grasp the process of reasoning. Causative pathology has been blended with the consecutive clinical features of disease, reasons have been given for facts, exceptions have been subordinated to what is usually found at the bedside, and the allurements of typical clinical pictures and dogmatic generalizations have been avoided because they hold neither in practice nor at the postmortem table.

The author also conceives that a book on Practice should be *well-directed,* which implies that it should deal adequately with scientific theories and principles, but that it should recognize that the final object of its existence is the *application* of knowledge *to the cure or alleviation of disease.* Accordingly, an unusual amount of space has been devoted to treatment, to the detailed consideration of drugs and to numerous formulæ and prescriptions ready for the student to use or improve upon. The physiological action of drugs has been dwelt upon carefully because, in the writer's experience, the symptoms of disease may be confounded with those of the remedies exhibited for its cure. The index embraces the chief references to the remedies and the formulæ for their exhibition.

The reader will also find a large number of tables giving the differential diagnosis of diseases likely to be confused, and in many instances, of entire subjects, such as those of the liver and kidney, the chief eruptive diseases and those of the typhoid group. They are practical though necessarily schematic. Nowadays the border-lines of surgery and medicine overlap, and the practitioner, internist and surgeon must be familiar with the province of each other. For this reason, as far as the scope of the treatise admits, surgical indications and results are introduced.

Names and dates are employed to give credit, where it is possible, to the great workers in the history of Medicine.

The writer wishes to acknowledge the extremely careful attention bestowed by the Publishers upon every literary and typographical detail, and the assistance of Dr. Milton Mandel in the proof reading, and of Dr. G. B. Dyche in many valuable suggestions.

A. R. E.

CHICAGO, 1909.

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SECTION I.

THE SPECIFIC INFECTIONS

TYPHOID FEVER.

The name (*Typhos, Stupor*) refers to the clouded mentality, and dates from Hippocrates. Bright, Louis and others recognized that ulceration of the bowels occurred in certain continuous fevers but typhoid was first described as a disease separate from typhus by Gerhard and Pennock (1837).

Definition.—Typhoid fever is a *general infection,* characterized—

1. *Etiologically* by the Bacillus typhosus.

2. *Anatomically* by hyperplasia and ulceration of the lymphatic structures of the intestine, hyperplasia of the spleen and mesenteric glands, and by parenchymatous change in other organs.

3. *Clinically* by a characteristic fever, roseolous eruption, enlargement of the spleen, slow pulse, a peculiar serum reaction, typhoid bacilli in the blood, and often by intestinal symptoms.

Etiology.—The *typhoid bacillus* was first seen by Eberth and Koch (1880), and first cultivated by Graffky (1884).

1. Characters.—It has short, thick, rounded ends, is three times as long as wide, in length is one-third the diameter of a red blood-corpuscle, and its numbers are usually, but not always, proportionate to the degree of infection. It is polymorphous, sometimes thread-like, varies in different media, contains no spores, is flagellated, and actively motile. It grows best at body heat, and is a facultative anaërobe growing on various solid and fluid media, especially on agar, gelatin, and, characteristically, on the potato. It does not produce acid, ferment lactose, produce indol, or coagulate milk. It stains with aniline dyes, but not by Gram's method; it produces an *endo-toxin* only by the disintegration of the bacillus itself.

Recently typical lesions have been produced in the chimpanzee.

Its tenacity of life is great, the germ persisting for years in bone lesions and from three weeks to three months after death. It resists drying, except in thin layers, for months, and lives some time in water, seldom longer than three weeks, although its multiplication is prevented by saprophytes. The germ may live for months in ice, sour milk, fæces, or in the upper, but not in the deeper, layers of the soil. It may live a year in soiled clothes, which explains contagion

in washerwomen and nurses. It is killed in a few hours by direct sunlight.

2. Location.—It is found in the *typhoid ulcers,* in the lymphatics of the mesentery, in the *spleen,* in the intestines at autopsy (chiefly in the upper small intestine), in the *fæces,* in the *blood,* in the *gall bladder,* in the urine, in the roseolæ, less frequently in the lungs, muscles, œsophagus and tongue, and in various secondary foci, as in pleurisy, pneumonia (sputum), endocarditis, meningitis, parotitis, osteomyelitis, and abscesses. According to Lynch, it has also been isolated sixteen times from the blood of the fœtus in maternal typhoid infections. In curious cases typhoid bacilli may occur in the stools of individuals, particularly children, not suffering from typhoid fever.

3. Entrance.—It resists gastric juice fairly well, yet its chief and probably sole *atrium* is the digestive tract, the infection being carried in water (its main medium), milk, butter, ice, vegetables, or oysters. As shown in the Spanish-American and Boer wars, dried fæces containing the bacilli may be carried by flies and sand. Alice Hamilton found the bacilli in flies in the Hull House region of Chicago. Cockroaches and other insects may also convey infection.

4. Exit.—The *germ leaves the body* chiefly in the fæces and urine, which are dangerous even far into convalescence; they are the chief means of dissemination, the disease being less often conveyed directly from one individual to another, although direct infection occurs in hospitals and camps. Lentz found that after a typhoid attack four per cent. of persons become "chronic carriers of typhoid bacilli," the germs remaining in their dejecta for one, fifteen or even forty-two years. Soper describes a cook who, though perfectly well herself, was a "bacillen-träger" and caused 26 cases of typhoid fever in five years.

Predisposing Etiology.—1. Reduced Physiological Resistance.—Reduced physiological resistance, as from overwork or mental depression, is a frequent predisposing cause; yet typhoid very often occurs in the robust. It is claimed that the weak or emaciated are immune,—*e. g.,* those with syphilis, tuberculosis, cancer, endocarditis, anæmia, or convalescents from other diseases. Some immunity is claimed in pregnancy, lactation, and the puerperium. Alimentary affections, according to Bouchard, such as decreased hydrochloric acid formation, or dilated stomach, favor infection. Most cases occur in time of war, or among the poorer classes, or among recent residents in typhoid localities (Trousseau).

2. Season.—For unknown reasons most cases develop in the late summer and fall ("autumnal fever").

3. Age.—The years from fifteen to twenty-five include most cases (56 per cent.), yet fœtal cases are recorded, and a few cases occur in the first year of life. Their number increases from the first to the fifth year. Between the fifth and fifteenth years as many are affected as after thirty-five. The disease is relatively rare after fifty.

4. Geographical Distribution.—Typhoid is endemic rather than epidemic. Although it is the most common continued fever in temperate climates, it is a world-wide disease and sustains a most intimate relation to the *water supply,* the disposal of *sewage,* the density of population, and personal hygiene.

Immunity.—Congenital immunity is not proved. Immunity has no relation to the severity of the disease, though it is usually conferred after one attack, but is not life-long nor as frequent as in the case of scarlatina. In the Hamburg epidemic (1887), $2\frac{4}{10}$ per cent. of the cases were second attacks; and in Eichhorst's series there were over 4 per cent. of such attacks. These second attacks equal or even exceed the original in severity.

Symptoms.—The general clinical picture offers more variability than that of any other infection. *Typhoid is not an intestinal disease, but a general infection.* Its forms are:

1. The typical enteric, frequently with diarrhœa, tympany, hemorrhage, and perforation.
2. The septicæmic, without any, or with very slight, anatomical intestinal lesions, though responding to the Widal test and accompanied by bacillæmia.
3. Other localizations than enteric; *e. g.,* in the lung, larynx, bone or gall bladder.
4. Mixed infections—malaria, streptococcus, colon bacillus, etc.
5. Paratyphoid, with close clinical resemblance to typhoid but with organisms differing culturally and in their agglutinating reactions. (See Diagnosis.)

The *incubation* lasts one to two weeks, with symptoms more vague than in other acute infections,—*e. g.,* depression, pains in the head, back, or limbs, dulness of hearing, disturbed sleep, or perhaps chilliness, anorexia, epigastric oppression, constipation, or even diarrhœa. The incubation lasted only three days in a girl who took a pure typhoid culture with suicidal intent.

The *first week* corresponds anatomically to the intestinal catarrh and the beginning of medullary infiltration of Peyer's plaques. The disease dates from the fever, which each evening is from 1° to 1.5° higher than on the previous day, until a temperature of 103° to 104° is reached. The pulse is full, dicrotic, and rapid, but not high in proportion to the fever. The tongue is coated white. Thirst, anorexia, dry cough, enlarged spleen, eruption of rose spots, and typhoid bacilli in the blood, are found. The abdomen shows slight distention and pain or tenderness over the ileocæcal region or epigastrium. Delirium is infrequent, and headache, constipation, dulness and apathy are usually present.

The *second week* corresponds to the end of medullary infiltration and the beginning of eschar-formation. The fever is higher and usually continuous; the morning remission is less. The pulse is full, faster, and less dicrotic. The blood responds to the Widal test. The tongue is dry and glazed, as are also the lips, pharynx, and

mouth, to which the mucus adheres. The voice is weak. The bronchitis increases, and the urine is febrile. Abdominal symptoms are often aggravated. Meteorism appears. Yellow pea-soup dejecta, which are involuntary in severe cases, occur. Roseolæ usually develop during this week. The face is apathetic, its lines obliterated, the mouth half open, and the upper lip retracted, showing sordes. Nervous symptoms increase with higher fever; delirium follows; the patient becomes less querulous; and euphoria from narcosis by the toxins is the rule, while pain suggests complications. The patient may begin to recover, through the increased bactericidal powers of the body fluids, or may die at this stage from hemorrhage, nervous toxæmia, etc.

The *third week* is the stage of ulceration. The fever gradually declines with marked morning remissions and unaccountable variations. The pulse is smaller, and faster (100 to 120), and loses its dicrotism; it is sometimes slower. The tongue clears and the roseolæ disappear, being replaced by miliaria from sweating. In exceptional cases diarrhœa may be seen for the first time in this week. Emaciation is marked. When the fever falls, the patient complains of hunger, weakness, and pain. Death may occur in the typhoid state from heart weakness, pulmonary inflammation, paralysis of the nerve-centers, hemorrhage, or perforation.

The *fourth week* (cicatrization) usually marks convalescence, as the average duration of typhoid is from three to six weeks; in severe cases the symptoms assigned to the third week may continue. Convalescence may be uneventful, or marred by slight temperature, rapid pulse, palpitation, nausea, vomiting, muscular weakness, etc.

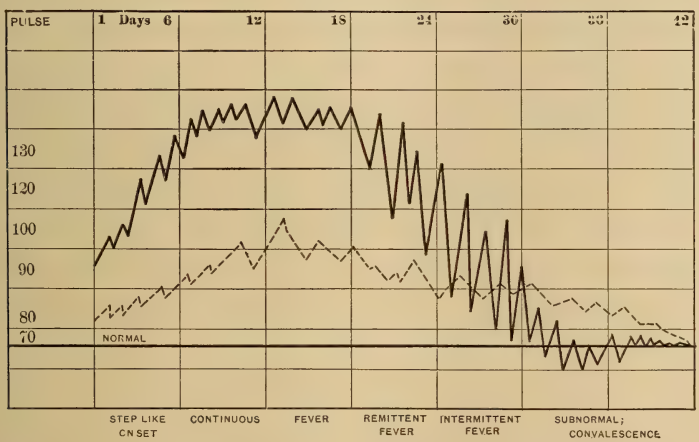
Symptoms in Detail.—To avoid repetition, and to explain anatomically the clinical signs and symptoms, the typhoid pathology will be incorporated under the appropriate organs, and complications and sequels will be classified with the more common typhoid manifestations.

1. Fever.—Fever is an expression of toxæmia, and is a cardinal symptom of typhoid. It is subacute, averages three to four weeks in duration, usually comes on without chill, and resolves by lysis. It is the best studied chapter in the disease since the time of Wunderlich, who diagnosticated typhoid from the fever chart alone. In the *first week,* corresponding to the catarrh and medullary infiltration, the evening temperature is $\frac{6}{10}°$ to 1° more than in the morning, and *the ascension is ladder-like.* In the *second week,* corresponding to the end of infiltration and to the formation of the slough, the *fever is continuous from one-half to three weeks.* In the *third week* (ulceration), the fever is remittent, the amphibolic stage of Wunderlich, and the *stadium hecticum* of Traube. The cause of fever variations is not known. The *fourth week* (cicatrization) is the stage of " steep curves," lasting from three to eight days. When a sudden drop occurs, recurrences and relapses are not infrequent.

Convalescence.—In its first week the fever-curve is usually subnormal; when not so, some irregularity must be looked for. Later there is a gradual rise to normal.

Atypical Fever Course.—As to *onset,* two variations may be observed, a sudden rise of the fever or its incidence with a chill. Chomel (1834) described cases with sudden onset and De Mussy (1884) spoke of an explosive onset; a sudden onset is observed in 10 per cent. of typhoid cases. The writer saw, with Dr. Korssell, a child whose temperature rose suddenly to 106° on the second day. When there is an initial chill every other possible disease should first be excluded, yet Osler observed chills in 22 per cent. of his cases; (*a*) at the onset, especially in children, and in light forms; (*b*) throughout the course, and accompanied by sweats, the *sudoral*

FIG. 1.



Typical typhoid-fever curve.—The heavy line marks the temperature curve, and the broken line the pulse curve.

type of the French; (*c*) in complications—pneumonia, pleurisy, otitis media; (*d*) after antipyretics, tubbing, or (*e*) in defervescence from secondary sepsis.

In the *second week* especially high temperature suggests complications; if the fever becomes very high, the course is apt to be severe. Sometimes a pseudo-collapse may occur, particularly in subjects between twenty-five and thirty years of age.

In the *third or fourth week,* the fever exceptionally may decline by crisis; Jaccoud observed a brusque defervescence in 29 per cent. of his cases.

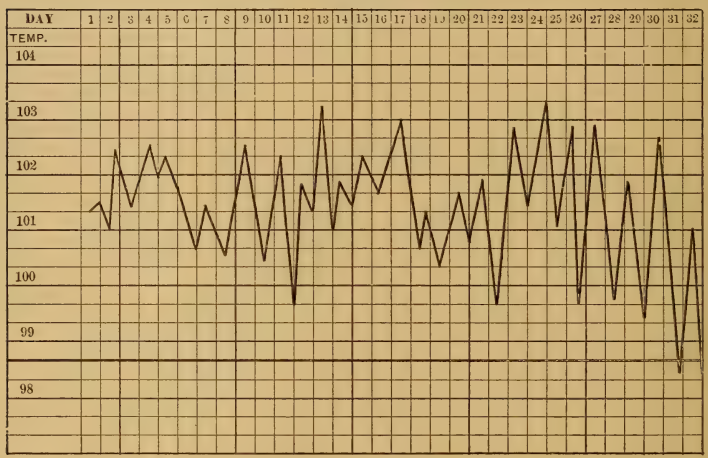
The fever may be intermittent or remittent throughout the disease, especially in children, the aged, and in severe cases. Irregularity and variations, especially when protracted, cause difficulty in diagnosis, suggesting, perhaps, miliary tuberculosis or sepsis.

In the *typus inversus,* seen chiefly in children and the aged, the morning temperature is higher than the evening record. Afebrile

typhoid is exceptional. Modification of temperature may be caused by visits from friends, excitement, entrance to the hospital, diet, hemorrhage, abortion, peritonitis, and other complications, as well as by relapse.

2. The Splenic Tumor.—The splenic tumor is a cardinal symptom occurring early and frequently, persisting in exacerbations and relapses. In importance and size, it ranks second to the splenic enlargement of malaria and sepsis. The cause of this tumor is the bacillus or its toxin (not the fever) which produces splenic hyperæmia and hyperplasia—changes closely analogous to those in the intestinal lymph-structures. Massive endothelial proliferation is said by

Fig. 2.



Typhoid fever; irregular fever curve throughout the course.

Mallory to occur in the splenic, lymphatic, and medullary vessels. In the first week the spleen is hyperæmic and swollen to two or three times its normal size. Its markings are indistinct. In the second week it is more pulpy and darker. By the fourth week it has usually disappeared, but *as long as we find the splenic tumor, the disease has not ended.*

Frequency.—(*a*) *Anatomically* the splenic tumor is present in 98.4 per cent. of the cases. Its rare absence is explained by thickening of the capsule, old induration or infarction of the organ, or extreme emaciation. (*b*) *Clinically,* it is present in 75 per cent. (Curschmann) or 90 per cent. (Leube). The percentage varies with the skill of the observer, the method of examination, the epidemic, and age of the subject (the sign being often absent in children, in adults older than forty-five years, or after hemorrhage). It should be palpated with the patient in the right diagonal position. The examiner should stand or sit on the right side of the patient, palpating with the right hand pressing evenly on the abdomen, and the

left hand hooked beneath and lifting up the lower left ribs. Tympany may obscure the splenic tumor. The author has twice seen the spleen ruptured by deep or rough palpation. Percussion of the spleen is worthless. It should be remembered that the spleen may be ectopic. Relatively rare splenic complications are abscess, infarct and rupture.

3. Dermal Symptoms.—(*a*) *Rose Spots* (roseola typhosa, *tâche rosée lenticulaire* of Louis) constitute the third cardinal symptom. They are *round spots,* varying from the size of a pin-head to that of a lentil (lenticular), circumscribed, *rose-colored,* slightly elevated to the touch, purely hyperæmic, and therefore *disappearing on pressure* and *reappearing on its release,* not often fusing, and later becoming darker, but leaving no mark, with sometimes a vesicle at their apex, and rarely desquamating, except in a few infantile or juvenile cases. They are rarely hemorrhagic, and therefore the commonly used term, petechia, is inappropriate. Being purely hyperæmic, they are not found at autopsy. They scarcely ever pustulate. They may be seen as early as the fourth day, especially in women and children, but *usually appear from the seventh to the tenth day.* They occur *in crops,* during two weeks, each crop lasting from three to five days. Occasionally, they outlive the fever. Their location is on the lower chest and abdomen, though they sometimes appear twenty-four hours earlier on the back, because of its warmth. They rarely occur on the neck or face, and generally are more frequent on the proximal than on the distal parts of the extremities. If there are many spots on the lower parts of the limbs, they are probably not roseolæ. They number five to ten, or even thirty. They are more abundant in women, less so in children and the aged, and seldom so abundant as to resemble measles closely. Their frequency varies somewhat with the epidemic. Eichhorst found them constantly in more than 1,200 cases, Leube in 80 per cent., Osler in 93 per cent., and Murchison in 33 per cent.

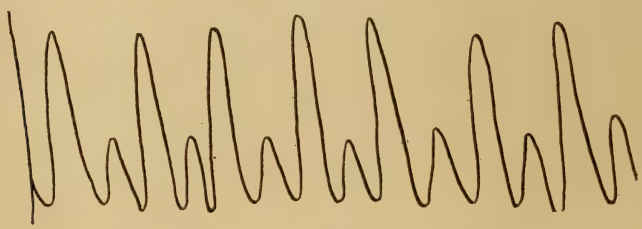
Typhoid bacilli in the roseolæ may establish a doubtful diagnosis, but rarely an early one; Polacco found bacilli in 100 per cent. and Krause in 94 per cent. of their cases. In other diseases, as miliary tuberculosis, meningitis, and pneumonia, roseolæ are very rarely encountered.

(*b*) *Other Cutaneous Manifestations.*—The typhoid odor is musty or semi-cadaveric. Miliaria are frequent, though less than in sepsis. They usually appear in the third or fourth week. Herpes is rare, occurring in less than 1 per cent. of the cases. The author has observed it only ten times in upwards of 1,800 cases. It is far less frequent than in pneumonia, malaria, typhus, meningitis, and influenza. Decubitus occurs in 1 per cent., mostly in severe and hospital cases. Noma is a rare complication. In 204 cases of gangrene collected by Keen, 50 per cent. occurred in the leg, 25 per cent. in the face, neck, and trunk, and the balance in other locations. Sweats and chills occur in the "typhus sudoralis," especially in the latter half of the fever course, described in Naples by Borelli and in Paris

by Jaccoud. Œdema is due most frequently to phlebitis, sometimes to anæmia, and rarely to nephritis. The skin sometimes shows atrophic striæ like those of pregnancy. Desquamation may occur, particularly in children. Other cutaneous manifestations are: Urticaria; erythema, even of the nodose or exudative forms; pelioma typhosum, or the *tâche bleuâtre*—pale blue spots measuring 4 to 10 millimeters, due to pediculosis, and therefore seen in parts nearest the pubic or axillary hair, etc.; erysipelas, phlegmon and furuncles (increased by hydrotherapy); alopecia after the typhoid attack, rarely followed by permanent baldness.

4. Circulation.—(*a*) *The Slow Pulse.—The slow pulse is the fourth cardinal symptom.* In uncomplicated cases it follows the temperature in the early stages, but is lower than the fever would justify, especially in men under forty years; *e. g.,* pulse 90 with the fever 103°. It is rather faster in women, in children, and in the nervous. Its slowness is due to *an inhibitory action of the toxins* on the medulla or the heart, a fact not recognized either by the older or by some modern writers. The author has seen four typhoid cases in which the pulse ranged from fifty-six to sixty-four, with the fever above 103° and the respirations above forty. Slowness is of

Fig. 3.



Pulse tracing in typhoid, showing dicrotism.

good import. At first a little tense in volume, it soon becomes *dicrotic,* which, with slowness, suggests typhoid. Dicrotism, evidenced by a double shock, occurs in 80 per cent. of cases, but is not prognostic. It is due to the shortened systole and to decreased peripheral resistance. It is usual in adults, and is absent in children and often in adults at the fastigium in very severe cases. Since it indicates lack of arterial tone, it is also absent in arteriosclerosis, in rigidity of the aorta or in vaso-motor paralysis. In Osler's Clinic, the *blood-pressure* was found to be 115 to 125 millimeters of mercury in the systole, and 85 to 100 millimeters in diastole. A fast pulse early in the typhoid course is a poor prognostic, especially in men. Irregularity or crossing of the pulse and temperature-curves, is also ominous. The pulse may become faster from heart or lung inflammation, perforative peritonitis, hemorrhage, and severe intoxication. It may also run up on visiting days, in women, children, emaciated and nervous individuals, or in the stage of great temperature variations. In the first stage of convalescence, when there is subnormal

temperature, the pulse is usually normal in rate, or slow, which is explained by disturbed conductivity in the heart muscle or by myocarditis. In the second stage it is usually higher than normal, especially after the patient gets out of bed, after eating, or a movement of the bowels. On the whole, bradycardia in convalescence is more common than tachycardia.

(*b*) *Heart Muscle.*—Early pathological changes are rare. *Parenchymatous degeneration,* evidenced by albuminous granules, nuclear swelling, and pigmentation, are found later, the heart as a whole being flabby, soft, pale, and even waxy. Fatty and hyaline degeneration, vacuole formation, and segmentary myocarditis are the usual findings in fatal cases. *Interstitial myocarditis,* like that of diphtheria or scarlatina, occurs in over 50 per cent. of typhoid cases (Rhomberg). It usually regresses without functional or organic disturbance, and chronic myocarditis rarely develops. Its clinical signs may appear at the end of the second week, lasting into the fourth week, or may develop first in convalescence. The symptoms are *gradual dilatation* of the left ventricle, weak tones (especially the first tone at the apex), accentuation of the second pulmonic, a systolic bruit, and a fast, irregular pulse with lessened tension but without dicrotism. Tachycardia, without myocarditis, may result from the toxins acting on the medulla oblongata. Recovery is usual, even if the myocarditis lasts two or three months.

Dilatation of the right heart may be observed in severe cases. Less frequent are the following complications: Cardiac thrombosis and embolism; pericarditis ([once in 1,000 typhoid autopsies], mostly fibrinous, and mostly in complicating sepsis); and endocarditis ([once in 1,000], mostly mural over myocarditic patches), sometimes due to Eberth's bacillus. *Collapse,* with its usual signs, may cause death from heart degeneration, or *from toxic vasomotor paralysis* (as in diphtheria, pneumonia, or pyocyaneus infections); myocarditis is the most frequent cause, while pulmonary embolism, uræmia, cerebral hemorrhage, etc., are far less common. Sometimes no clear cause is discovered at autopsy. Collapse develops mostly in strong men (80 per cent. of Dewèvre's 140 cases) and most often during convalescence. (See PROGNOSIS.)

(*c*) *Vessels.*—*Phlebitis typhosa* occurs in 1 per cent. of the cases, being considered marantic, or infective due to Eberth's bacillus or pus cocci, when pain and fever are present; it occurs chiefly in males, is unilateral and is usually left-sided, since the left iliac vein has a slower current, being crossed by the right iliac artery. The symptoms are pain, coldness, œdema, and sometimes a tender, palpable cord, which should be manipulated most carefully, if at all, lest embolism be caused. Thrombosis usually occurs in the saphenous vein where it empties into the femoral, sometimes in the popliteal vein, and rarely in the arms. It may extend from the leg to the cava, when the other leg may be involved. There is some danger of detachment, pulmonary embolism, and sudden death; but relative

recovery is usual after two or three months. Arteritis typhosa, most often found in the femoral artery, is rare, W. W. Keen collecting only forty-four cases. It is a specific inflammation caused by the typhoid bacillus. Spontaneous gangrene may result. Arteriosclerosis may be initiated by typhoid.

(*d*) *The Blood.*—The *red blood-cells* average 4,000,000. Their reduction is always slow, and anæmia is especially frequent during the third week or in relapses. An apparent increase of the red disks, analogous to that observed in cholera, may result from loss of fluids, as from diarrhœa (Hayem). The hæmoglobin decreases proportionately to, or slightly in excess of the oligocythæmia, especially in convalescence. Marked post-febrile anæmia is not common. The *leukocytes* decrease from 8,000 to 3,000, or even less, but average 5,000 per cubic millimeter. *Leukopenia* is more common in typhoid than in any other febrile condition and helps to differentiate it from pneumonia, sepsis, meningitis, etc. It is possibly due to destruction of the lymphatic elements by the endothelial phagocytes. Increase of the white cells occurs only in complicating inflammations or in convalescence. The polymorphonuclear neutrophiles are absolutely and relatively decreased to below 60 or 50 per cent., while they are increased in other infections. The large mononuclear and transitional forms are relatively increased. The eosinophiles are greatly decreased; their return is a good prognostic (Türck).

Typhoid is *a primary bacillæmia or septicæmia.* Fränkel and Simmonds, in 1885 first found the bacilli in the blood. The bacilli reach the blood from the lymphopoietic organs, and from the blood reach the fæces secondarily by way of the bile or by ulceration of Peyer's patches. A *positive blood culture* is the safest and surest means of diagnosis *in the first week of typhoid.* Cultures are positive in 100 per cent. in the first week, in 71 per cent. in the second and in 36 per cent. in the third week (Silverberg). Convalescence is rapidly established as soon as the bacillæmia ceases.

The Gruber-Widal Serum Test.—The "agglutination" and "immobilization" of typhoid bacilli brought in contact with a typhoid serum, were found by Durham and Gruber, and also independently by Pfeiffer and Kolle. Gruber pointed out the value of the test in proving the existence of a previous typhoid, while Widal applied the laboratory facts at the bedside, and found the reaction during the disease. The agglutinins are produced by the tissues reacting to the bacilli.

Technique.—A bouillon culture, not more than twelve to eighteen hours old, is used, taking forty parts of the bouillon and one of the blood (dilution of 1 to 40). The test may be made macroscopically in the test-tube, or on the microscope slide, where immobilization, clumping, and agglutination of the bacilli, may be observed sometimes in a very short time—an hour is the usual time-limit for the test. Ficker and Ruediger have recently employed dead cultures with equally accurate results, and this method is certainly more con-

PLATE I

FIG. 1

Bouillon Culture of Typhoid Bacilli before the Addition of Diluted Typhoid Serum. × 500. (After Cabot.) Serum diagnosis.

FIG. 2

The Same, Five Minutes after the Addition of Typhoid Serum (dilution 1 to 10), showing Typical Clump Reaction. × 400. (Cabot.)

venient, safe, and capable of wider use than the use of living bacilli, especially in country practice. (Plate I.)

Significance.—The Gruber-Widal reaction may be found thirty years after a typhoid attack. It is sometimes absent in typhoid, particularly in severe cases and it often develops late, *e. g.,* on the seventeenth or the fortieth day. Cabot found the reaction in 97 per cent. of 5,978 collected typhoid cases. In Osler's series it appeared in 93 per cent. before the eighth day. When it appears in the first week, it is due to a "silently developing" infection (Widal); *i. e.,* the typhoid is more advanced than we appreciate. Leube considers the test significant only when it is absent in the early stages and develops later, care being necessary in all other cases whether the reaction be positive or negative. Undue importance is sometimes attached to this as well as to many other laboratory tests, invaluable though they may be in connection with the physical findings. Typhoid bacillæmia decreases as the Widal reaction develops. Kasel and Mann believe the reaction is not one of infection, but one of immunity, agglutination becoming most marked toward the end of the disease.

5. Nervous Symptoms.—Typhoid was once called "nervous fever" (*febris nervosa, nervenfieber*). The nervous system may be conspicuously involved even in the incubation stage.

(*a*) *Headache.*—Toxæmic headache is almost invariable, being most often frontal or occipital, constricting, throbbing, or a pressure sensation. It usually disappears in the second week, and, if it lasts or develops late, it may indicate meningitis. Insomnia may be both early and lasting. The early excitation passes into the later euphoria of the typhoid state.

(*b*) *The Typhoid State.*—The typhoid state begins in the second week (*v. s.*) with apathy and delirium, which is usually mild. The patient may attempt to get out of bed, and, in alcoholics, delirium tremens may be simulated or actually develop. In mild cases delirium lasts into the third week. In very irritable subjects, as in women and children, or in overwhelming intoxication, we may have photophobia, stupor, coma, loud breathing with an open mouth, absence of the pharyngeal reflex, involuntaries, waving of the hands (floctitation), leaping of the tendons (*subsultus tendinum*), picking of the bedclothes (*carphologia*), trismus, contractures, grinding of the teeth, rigid neck, and convulsions (in children especially), which symptoms are not due to the fever, since they all may occur with low temperature, but to toxæmia. In coma-vigil the patient is semicomatose subjectively, but the open, unseeing eyes give a "watchful" appearance objectively. Anatomically a "wet-brain" is found. Murchison observed convulsions but six times in 2,960 cases. They may develop in children, in severe intoxication, or from such cerebral complications as meningitis, encephalitis, thrombosis, or embolism.

(*c*) *Nervous Complications.*—Typhoid may begin as mania. The

author has seen five typhoid patients taken to the detention hospital in the third week, or even in convalescence, under a mistaken diagnosis. Mania may be a later symptom.

Psychoses are due to the exhausting inanition of protracted toxæmia, or sometimes an hereditary predisposition. They are generally *post-febrile,* are of the depressive variety such as melancholia, and usually give a favorable prognosis, though alienists assert that one-third to one-fifth do not recover. Edsall (1905) found that two thirds of juvenile cases recover. Typhoid may cure a previous insanity.

Meningitis occurs in 0.5 per cent. of cases, mostly in women and children, generally as a complication and late in the typhoid course. Meningitis may be due to the pneumococcus, the pyogenic cocci, or to Eberth's bacilli, which have been found on lumbar puncture in over twenty-six cases (Cole), of which half were serous and half suppurative. There are four recorded cases of tuberculous meningitis complicating typhoid. Actual meningitis must not be confounded with pseudomeningitis or "meningismus," in which toxæmia produces irritative meningeal symptoms, as described under typhoid state (*v. s.*).

In the *brain,* œdema, degenerated and pigmented ganglionic cells, and nuclear multiplication, are the most common findings at the autopsy. Hemorrhage, embolism, arterial and sinus thrombosis, and abscess (sepsis), are rare pathological complications. Aphasia, monoplegia, hemiplegia, and paralysis of the cerebral nerves, are rare clinical complications. The author has twice noted hemiplegia developing suddenly in convalescence, probably embolic from detached heartclots. In both cases the patients were allowed to leave the bed too soon. Forty cases of hemiplegia in typhoid are on record.

In the *spine,* myelitis, multiple sclerosis, infantile paralysis, and Landry's paralysis (Eberth's bacillus), are rare complications or sequels. The knee-jerks are sometimes increased, especially in emaciated subjects.

Neuroses may complicate convalescence, such as chorea, hysteria, neurasthenia, Basedow's disease, tetany, paralysis agitans, etc.

In the *nerves,* neuritis in the single or multiple form (*q. v.*) with anæsthesia, paralysis, and atrophy, may follow typhoid as it does other infections. Neuralgia occurs late. In the feet it constitutes the "tender toes" of Hanford, and is seemingly increased by bath treatment. It always results in recovery.

6. Digestive Tract.—The upper lip is retracted and bleeds easily. Sordes of dried saliva, food, and epithelium, are observed on the teeth and gums. Though the tongue shows nothing absolutely characteristic, Leube attaches special importance to its appearance. At first it is white on the dorsum, moist, flabby, and therefore indented by the teeth, and its edges are red. It may be moist throughout the disease. In the second week it becomes dry (from the fever, lack of saliva, and mouth-breathing), small and fissured; it bleeds,

and a brown or even black coating replaces the white. In the third week or later, a triangular patch clears at its tip. Typhoid bacilli are not uncommon on the tongue. Acute glossitis is a severe complication. Aphthæ are rare.

Angina may usher in the disease, or, occurring in the first week, lead to a wrong diagnosis. The throat is swollen, granular, and eroded. The angina, usually of the ordinary type, may be due to the typhoid bacillus (Wagner and French). The phlegmonous type is very dangerous. An angina cachectica of white plaques or epithelium and bacteria is occasionally observed. Letulle has seen 60 cases with yellowish or pinkish gray ulcers on the uvula or anterior pillars.

Parotitis, occurring in ½ to 1 per cent. of the cases, has become less frequent since special care of the mouth has lessened infection ascending Steno's duct. It is usually one-sided, is due to the typhoid bacillus, or pyogenic cocci, and is usually seen on the fifteenth day. The prognosis is poor, especially when due to metastasis. Two personal cases, caused by the typhoid bacillus, recovered.

Ulceration of the œsophagus is very rare; the cicatrized ulcer may produce later stenosis (twelve cases were collected by J. E. Thompson, 1904).

Specific typhoid lesions are rarely observed in the *stomach,* although hæmatemesis occurs (Leube and Schlesinger). Early and severe *epigastric pain* rather contraindicates typhoid. Incipient *nausea* and *vomiting* are unusual, occurring mostly in women or children. If observed at the height of the disease these symptoms suggest meningitis, *overfeeding,* or *perforation,* and (in convalescence) *dietetic errors.*

Anorexia is observed at the onset and during the fastigium, though great hunger develops in convalescence.

Specific changes in the intestines: (*a*) *Hyperæmia, catarrh,* and beginning medullary infiltration in the lymphoid structures during the first week, most changes being observed in the lower ileum, whence the name ileo-typhoid. (*b*) *Medullary infiltration* or *hyperplasia,* during the second week, occurring first in Peyer's patches, and then in the follicles. The patches are oval and parallel with the long axis of the gut; they lie opposite to the mesenteric attachment, are 3 to 5 millimeters high, and are at first red, then paler from compression of the bloodvessels. The infiltration is hard or soft. It may be uneven and reach beyond the plaque. The solitary follicles, even to the sigmoid flexure (colotyphoid), may be principally enlarged. These changes are not absolutely distinctive of typhoid, but are more significant in adults than in children, in whom they are frequent in digestive diseases, the exanthemata, and diphtheria. Mallory believes that the lymphoid swelling is wholly explained by the proliferation of the endothelial cells of the vessels to act as phagocytes; when they degenerate, fibrinous thrombi develop. Medullary infiltration need not advance to ulceration. Regression may occur

and the lymph-cells degenerate and disappear. The lymphatic structures may then assume a reticulated, or "shaven beard," appearance. (*c*) *Ulceration* occurs in the third week, from anæmic necrosis, slough formation, and exfoliation of small particles. The ulcers thus formed by the typhoid bacilli are usually easily seen without opening the gut. They have an irregular, oval outline, sharp, steep, often undermined edges, with a red floor generally composed of the muscular coat and often extending deeper than the eschar. The lower ileum is most affected and may present almost no normal surface in extreme cases. The solitary follicles, both in the large and small gut, ulcerate at their apices. Baer has studied the localization of the typhoid ulcer and states that it may occur in any part of the digestive tract except the mouth. The pharynx is affected in 2 per cent. of the cases, the œsophagus in 4 per cent., the stomach in 2 per cent.; 97 per cent. of the ulcers develop in the small intestine, 89 per cent. in the ileum, 2 per cent. in the appendix and 33 per cent. in the large intestine. (*d*) *Cicatrization* occurs by granulation tissue and growth inward of the epithelium. The patch is depressed and slightly pigmented. The typhoid cicatrix does not narrow the intestinal lumen.

There may be few or no anatomical changes in the intestine. Slight bowel lesions are usually present in most of the cases reported "without intestinal lesions."

The *mesenteric glands* are swollen in the second week, contain typhoid bacilli, are altered like the intestinal lymphatics, and may be palpable. The swollen glands usually regress, but may soften, become tuberculous, or suppurate, sometimes causing peritonitis, or simulating perforation. Only six cases are recorded of suppuration in the mesenteric glands; Le Conte reports a recovery after operation.

No parallelism exists between the frequency, constancy, or severity, of the clinical symptoms, and the degree of anatomical involvement; *e. g.,* in ambulant cases there may be perforation or hemorrhage, or lethal cases may occur with little intestinal change.

Meteorism is rather less common than usually described. It occurs mostly in the duodenum or jejunum above the ulceration, but sometimes involves the colon. It is less frequent and less intense with early treatment and careful dieting. If much tympany develops under these circumstances, the prognosis is poor, since meteorism *is due to toxæmic paresis of the intestinal musculature* rather than to local ulceration. The heart and lungs may be crowded upward and dyspnœa result. It may occur with hemorrhage or perforation.

A thickened ileum may be palpated. Abdominal *tenderness and pain* occurred in 60 per cent. of Osler's series and in 30 per cent. at the onset, where particular attention was paid to these points. They are due to: (*a*) Local bowel conditions, as gas, constipation, inflammation in the ileocæcal region, or perforative and peritonitic complications; (*b*) to splenic or hepatic tumefactions; (*c*) pleurisy, distended bladder, phlebitis, etc.; (*d*) muscle changes or cutaneous hyperæsthesia, so frequently observed in fever of any variety.

Typhoid *appendicitis* may simulate ordinary appendicitis, which is differentiated only by the blood-count, sero-reaction, or, most surely, by the clinical evolution; *e. g.,* the fever, rose spots, etc., of typhoid, precede local pain and tenderness. Gurgling has no diagnostic value whatever in typhoid, although it occurs in most cases.

The *stools* present no pathognomonic chemical nor physical characteristics; the bacillus typhosus, present in 20 to 40 per cent. of cases, reaches the bowel by the bile or through the ulcers. The dejecta are thin, offensive, pea-soup-like, alkaline, and resemble the contents of the small intestine (Addison). Because the fæces are poor in mucus, two layers form on standing, an upper cloudy, and a lower, yellow-gray, granular and flocculent layer. The stools contain red corpuscles and triple phosphates, the latter more abundantly than in any other disease. Shreds or sloughs may also be found.

Diarrhœa in the febrile stage occurs in 25 to 33 per cent. of cases. Three to six stools daily are the average. If this is exceeded, the type of the disease is severe; prodromal diarrhœa rarely occurs. Diarrhœa is a *measure of the degree of toxæmia* or of *colonic involvement,* and is not a result of ulceration of the small intestine.

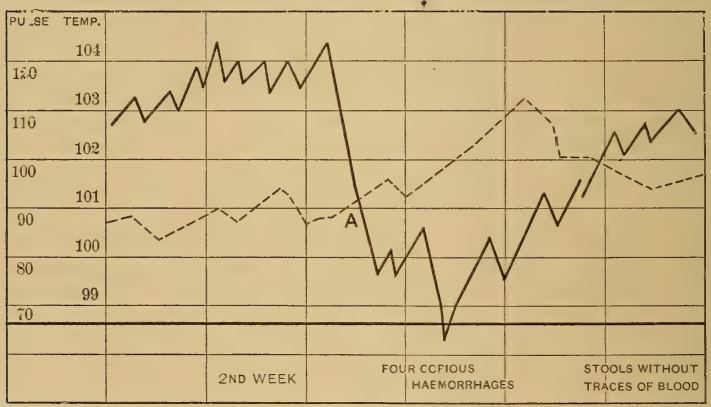
Hemorrhage is seen in 4 to 6 per cent. of the cases. In time it is usually late, though some assert that in 30 per cent. it occurs in the first two weeks. It is less frequent in relapses and in children (1 per cent.). *Ulceration into the muscularis* is its chief cause. Oozing, from simple hyperæmia, bleeding in the hemorrhagic forms (blood dissolution), and in patients inheriting the bleeding tendency, are exceptional. Bleeding is more common in ambulatory, delirious, and carelessly fed patients. There may be no symptoms if the hemorrhage is small (occult hemorrhage); but if it be large, acute anæmia suddenly develops, with collapse, which is evidenced by a fast pulse, lowered temperature, and pallid skin. The intellect often clears, the spleen becomes smaller, and fresh blood, or, later, black, tar-like movements, are voided (manifest hemorrhage). The abdomen is often distended. There may be dulness on percussion from blood retained in the bowel, if the peristalsis is weak and the hemorrhage is very profuse. In two rapidly fatal cases the entire abdomen became flat and one and two pints of blood were vomited. Confusion with bleeding piles and bismuth stools is possible. The average number of hemorrhages is four.

Some patients improve at once after hemorrhage, as was remarked at an early date by Graves and Trousseau; yet 20 to 50 per cent. die. Large blood-clots are ominous, indicating erosion of a large artery. Hemorrhage may cause death within an hour (Trousseau, Leymaire).

Perforation occurs mostly at the time of eschar-formation, at the end of the second, or in the third week; but it may also occur later,—in the third to fifth week (Leube). Scott found that 92 per cent. occurred between the second and fifth weeks. It develops in 9 to 12 per cent. of fatal cases and *in 3 per cent. of all cases,* but it very rarely occurs in children. Scott's figures, including 9,713 English

and American cases, show perforation in one-third of all fatal cases. Perforation is usually low in the ileum (81 per cent.); or in the colon or sigmoid (12 per cent.); in the appendix (3 per cent.); in Meckel's diverticulum (2.4 per cent.); or in the jejunum (1.2 per cent.), according to Fitz's figures. In 83 per cent. but one perforation is found; several may occur, twenty-five being observed in one patient. It is promoted primarily by *deep ulceration,* and secondarily by severe infection, movement, strains, coughing, constipation, tympanites, or solid diet which increases peristalsis. It is claimed that in 75 per cent. of cases perforation occurs in severe infections. Three types are encountered: (1) Least frequently, undiscovered latent perforation; the typhoid state masks the perforation, which affords an unpleasant postmortem surprise. (2) More

FIG. 4.



Typhoid-fever curve, with fall of temperature (solid line) after hemorrhage and rise of pulse (broken line). Note crossing of curves at A.

frequently, cases with gradual onset, and therefore ambiguous symptoms. (3) Most frequently, cases with sudden symptoms and more or less clear diagnosis. The *symptoms of perforation* are: (*a*) *Pain* (in 75 per cent.) due to the perforation itself, usually localized in the hypogastrium or right lower quadrant of the abdomen and followed later by the pains of peritonitis. In 70 per cent., the pain is sudden and severe; in 20 per cent., gradual in onset; and in 10 per cent., absent. (*b*) *Tenderness* on palpation (75 per cent.), evidenced by the board-like rigidity of the abdominal muscles. (*c*) *Limitation of the respiratory movements* of the abdomen, due to pain and tenderness. (*d*) *Vomiting* of gastric contents or fæcal matter. (*e*) *Intestinal obstruction.* (*f*) *Abdominal distention,* appearing possibly for the first time—in which event its significance is enhanced—or increasing, if already present. There are cases in which the abdomen is flat or even retracted; in one typhoid patient, seen first in a dying condition, the scaphoid abdomen contained three pints of pus. Abdominal distention crowds upward the diaphragm, heart, and lungs,

and causes hurried breathing. (*g*) Disappearance of the flatness of the liver by reason of free gas in the peritoneum. This is most significant if the abdomen was not previously distended. Perihepatitic adhesions naturally prevent obliteration of the liver dulness, and it must always be remembered that a gas-distended colon may overlie the liver, and that in some cases the colon normally overlaps the liver. (*h*) Other occasional abdominal findings are flatness in the flanks, caused by fluid; the Beatty-Bright friction-rub; absence of peristalsis; rectal or vesical pain or tenderness. (*i*) Marked leukocytosis is not always found, and special emphasis on its absence is most misleading, as the leukocytes are occasionally decreased. (*j*) Sudden fall of temperature, rapid rise in pulse-rate, and other signs of shock, may occur at the time of perforation, though less frequently than the usual descriptions indicate. The writer has seen cases in which the pulse-rate did not increase. Dieulafoy mentions a case, successfully operated upon, for perforation, in which two later perforations resulted fatally. The sudden clearing of the patient's intellect may deceive the inexperienced physician. In the advancing generalized peritonitis, the fever, if it has fallen, rises; the pulse becomes rapid, small, and thready; the skin clammy and dusky, and the features pinched (facies Hippocratica). Hiccough is common. The peritonitis rarely remains local. In six of the author's cases, the local abscess ruptured later. Ninety-eight per cent. of the unoperated cases die.

Peritonitis occurs, frequently in ambulatory cases, from rupture of the bowel, gall-bladder, spleen, or lymph-glands; occasionally from post-typhoid appendicitis or without rupture of any organ or tissue. It is found in 2 per cent. of fatal cases.

The *liver* in the early stages is anatomically hyperæmic, large and firm; later its lobular markings become indistinct from cloudy swelling and fatty change. The organ is softer, and Wagner's lymphomata (and some focal necrosis) occur. Clinically, some tenderness is not unusual and at times may lead to an incorrect diagnosis of liver abscess or cholecystitis.

Icterus is very rare (0.5 per cent.), and its absence is of diagnostic value, as the duodenal catarrh common in other infections is usually absent. Icterus may occur from gall-stones, cholecystitis, abscess, etc. The author has but once seen icterus in typhoid fever. Single liver abscess is rare, as are suppurative pylephlebitis and suppurative cholangitis. Twenty-six cases of liver abscess are on record (Sheldon, Swain, Sennert).

Typhoid favors the *formation of gall-stones,* which may develop months, years, or decades later.

Cholecystitis has been reported by Chiari and others; thirty cases with perforation are on record. Tenderness, pain, muscular rigidity, and enlarged gall-bladder, are found in most cases. Typhoid bacilli are found in the gall-bladder in nearly all autopsies, although seldom having produced symptoms, and probably occur from hæmatogenous

infection. Dehler published a case in which the gall-bladder was drained to rid a patient of the persistent bacilli.

7. The Respiratory Tract.—This is the seat of frequent and manifold symptoms and complications.

(*a*) *The Nose.*—Epistaxis occurs in 20 per cent. of cases, usually early, and in young subjects; it is often profuse, sometimes dangerous, but rarely lethal. Croup and diphtheria are rare. The author has seen but two cases begin with profuse coryza.

(*b*) *Larynx.—Catarrh* is common. *Decubitus,* with erosions of the swollen hyperplastic lymph-follicles (like the intestinal changes), occur in the posterior wall, followed often by œdema glottidis. These bed-sores are seen at the height of the fever; occur rarely in children; and are found in 5 to 20 per cent. of the lethal cases. *Perichondritis* and *necrosis* are attended by pain, aphonia, stenosis, mediastinitis, cervical cellulitis, and diffuse emphysema. Tracheotomy may be indicated. The author has seen but three cases of perichondritis. Dupuy (1903) collected 255 cases of perichondritis and submucous laryngitis, and believes they explain 3 to 10 per cent. of typhoid fatalities. Without operation, the mortality is 98 per cent.

(*c*) *Air Tubes.—A dry catarrh* of the trachea and bronchi is almost invariable, while ulceration or fibrinous inflammation is rare. Rales, retrosternal dryness, rawness, and pain, are present in the *bronchitis* of typhoid. At autopsy the bronchial glands are often swollen, and Eberth's bacillus is sometimes found. *Bronchiolitis* is regular and specific. It is found everywhere in the lungs, but especially in the lower lobes. Coughing is not invariable but its presence in early typhoid is of value in differentiation from simple intestinal catarrh. The outlook is less favorable when severe and early coughing occurs.

(*d*) *Lungs.—Hypostasis,* from a dorsal decubitus and weak heart, is often relieved by change of posture. It is very common, and occurs in the first half of the disease, or at its height, mostly in weak adults and in cases of severe infection. Lesser degrees of hypostasis may be overlooked. The percussion-note is tympanitic or dull; the fremitus is increased, if the patient can talk, or decreased from stagnation of secretion; the breathing, and especially the expiration, is distant (or sometimes bronchial). Rales, moist, crepitant, and subcrepitant, are heard.

Lobar pneumonia may, in rare instances, dominate the early clinical picture (pneumo-typhoid) and may be of two types. The first type, (*a*) in which the pneumonia, usually seen at the height of the disease, is a true complication, due to the pneumococcus, though sometimes due to the colon bacillus and streptococcus, especially in sepsis. The pneumonic symptoms are variable, atypical (as in most secondary pneumonias), often without chill, with a slower course, and often ending by lysis, or, less often, with such complications as abscess or gangrene. More than one-half of these cases die. The second type (*b*) is due to Eberth's bacillus. The course is that of an initial pneumonia, which does not end typically, but shows later a

typhoid temperature, rose spots, Widal reaction, etc. These cases are naturally confusing, especially in their incipiency, and the typhoid is more apt to be overlooked than in the first type. *Lobular pneumonia,* from inhalation, pus cocci, and Eberth's bacillus, is a true complication. *Abscess* follows pneumonia or sepsis; it occurs in 0.7 per cent. of the fatal cases. *Infarct* occurs in 6 per cent. of typhoid fatalities. Marantic thrombi from the leg may reach the lungs by way of the right heart. This is indicated by hæmoptysis, chill, temperature, pain, etc. *Gangrene* (1 to 2 per cent. of typhoid autopsies) occurs more often than abscess, is seen late in the disease, and is usually metapneumonic, but may be caused by perichondritis or aspiration of food.

(*e*) *Pleura.—Serofibrinous pleurisy* is usually rare unless the lungs are diseased. Sears observed 18 cases in 1,065 typhoid patients. Postmortem statistics show it in 6 per cent. It is most common as empyema in convalescence, and is due oftenest to Eberth's bacillus. In 1903, fifty-five cases of pleurisy were collected in which the typhoid bacillus was found. Pneumothorax and hæmoptysis are very uncommon.

(*f*) *Tuberculosis.*—Old latent tuberculosis may be aroused, usually late in the disease. *Acute caseous pneumonia, subacute tuberculous peribronchitis* with a few bacilli, fever, emphysema and aggravation of the bronchial symptoms, and occasionally miliary tuberculosis may occur. The sooner tuberculosis begins after typhoid the more unfavorable is its course.

8. Genito-Urinary Tract.—(*a*) The *urine is febrile, i. e.,* decreased, acid, concentrated, with a specific gravity of 1,030 or more. The amount of urine depends considerably upon the amount of water ingested. Typhoid bacilli are present in 25 to 50 per cent. of the cases. The bacilli may give the urine a glistening appearance. Bacilli may persist from four months to several years after recovery, probably multiplying in the bladder. In convalescence, the urine is increased, is neutral in reaction, and has a lower specific gravity. Sometimes polyuria may occur during the fever. (*b*) Serum albumin, globulin, and rarely peptones, are found in *febrile albuminuria.* Serum albumin is found in quantities varying from traces to considerable amounts, in 20 per cent. of the cases; cylindruria is approximately half as frequent. The prognosis is three times as unfavorable when albumin is found. Albuminuria appears from the seventh to the tenth day, and its average duration is twelve days. It corresponds anatomically to parenchymatous and fatty degeneration of the kidneys. (*c*) *Nephritis* in typhoid is of the acute parenchymatous type, with the interstitium of the organ but little involved. It is often hemorrhagic, and occurs usually in the first three weeks but occasionally in convalescence. It is found in 1 per cent. of cases, mostly in men, rarely in children. Nephritis makes the prognosis less favorable, as it indicates a more severe infection, from which 50 per cent. of the cases die. Uræmia and suppression of urine are rare.

If not fatal, nephritis usually clears up entirely and rarely remains in chronic form. Early and marked renal symptoms constitute the nephrotyphoid of French writers. Miliary typhoid abscesses in the kidney may keep up the bacilluria. Lymph-nodes are found in some cases, as in the liver (Wagner). (*d*) The urine of typhoid exhibits the *diazo reaction* of Ehrlich (1882), which is due to the incomplete oxidation of the proteid metabolism of the aromatic sulphates. The author has found it in 98 per cent. of his cases. It occurs in the early stages of the disease, also at the acme and during relapses. It is a valuable sign, though of secondary rank. Since it is found in numerous other acute and chronic conditions, it has no differential or pathognomonic value. It rather closely parallels the bacillæmia. Its absence, together with the absence of other findings, contraindicates a florid typhoid. (*e*) *Cystitis* and *pyelitis* occur, due chiefly to pus organisms, or typhoid or colon bacilli. Simple retention of urine is not uncommon. Hæmoglobinuria is quite rare. Typhoid bacilli may form the nuclei of renal stones. (*f*) The *genitalia* are more often implicated in women than in men. Menstruation is disturbed. It often appears early in the onset of typhoid, and is suppressed in 60 per cent. of the cases. Hæmatocele or hæmatometra may develop. Endometritis, vaginal erosions, ulcers, inflammation, or gangrene and mastitis, are rare complications. *Pregnancy* is of grave import, the outlook being worse in its later stages than in the earlier (*e. g.*, eight to ten weeks). It is most dangerous at the acme of the fever, abortion and premature delivery being very frequent (in 65 per cent.). The maternal mortality is increased at least two fold. Blumer and Dobbin have reported puerperal infection from the typhoid bacillus. *Orchitis* usually develops late, or in convalescence, with chills and fever. J. G. Beardsley, in 1907, collected 102 cases which were chiefly due to the typhoid bacillus. The patient almost always recovers in ten to fourteen days, although suppuration and atrophy have occurred. It is frequently associated with urethritis. Emissions are common in convalescence.

9. Special Senses.—The *eye* is rarely involved. Muscle paralysis (neuritis) and various inflammations are very uncommon. In convalescence, mydriasis and paralysis of accommodation often result from exhaustion. Uhtoft, in 253 cases of optic neuritis, found 17 cases following typhoid.

Involvement of the *ear* includes functional toxæmic disturbance of hearing, abscess, and otitis media (2 per cent.). Mastoid disease and meningitis are very exceptional.

10. Muscles, Thyroid Gland, and Bones.—The *muscles* anatomically are dry, of a smoked-meat appearance, and may be the seat of hemorrhages, rupture, granulo-fatty and waxy degenerations, and abscess, due to Eberth's bacilli and pus cocci. Myositis may explain some cases of muscular tenderness with cramps.

In the *thyroid gland,* a strumitis typhosa, with suppuration or regression, has been observed. It is due to the typhoid bacillus. Recovery is the rule.

Though *bone disease* was observed in 237 cases by Keen, it is much more frequent than the figures indicate. *Periostitis* is more common than bone disease proper. These complications are post-typhoid and are due to the typhoid bacillus (75 per cent.), or pus cocci (25 per cent.). They attack the tibia (38 per cent.), the ribs (13 per cent.), femur, ulna, temporal bones, etc., and occur chiefly in young individuals. "Their most striking features are chronicity, indolence, and a remarkable tendency to recurrence" (Osler). *Osteomyelitis* may resemble syphilitic or tuberculous osteopathies. Typhoid bacilli have been found seven and ten years after typhoid in osteomyelitic fistulæ. Longcope has found marrow changes which resemble lymphoid hyperplasia, and Fränkel isolated typhoid bacilli in the vertebral marrow in ordinary cases of typhoid.

Arthritis may be typhoid or gonorrhœal, rheumatic, septic, etc. Keen has noted eighty-four cases. It may be poly- or mono-articular, serous or purulent. The hip is most frequently affected. The "typhoid spine," of Gibney, is characterized by pain, tenderness, and stiffness in the back; these seem to be root symptoms. Once classified as a neurosis, the *x*-ray findings of McCrae seem to indicate an actual spondylitis.

Anomalous Courses.—According to Curschmann, the following aberrant courses are distinguished:

1. Malignant.—The *malignant,* hyperpyretic or foudroyant form is rare. It is severe even in the earliest stages. The temperature rises rapidly and there may be hyperpyrexia. The pulse is soft, weak, and rapid. The status typhosus is profound. Meteorism, diarrhœa, and albuminuria are present, and death from deep intoxication usually occurs.

2. The Slow Severe Form.—The *slow severe* form lasts from four weeks to four months. The fever is often remittent with frequent exacerbations. Defervescence may occur or death intervene, marasmus alone being found at autopsy. The severe types of typhoid have grown rare, even during the last decade.

3. Typhus Abortivus.—*Typhus levissimus, typhulus, typhoidette* of Brouardel, exhibits a short and mild course, temperature with a short initial stage, or often a crisis, lasting six to twenty days. Bronchitis is frequent, the spleen enlargement is constant, and meteorism, diarrhœa, roseolæ, hemorrhage, and perforation, are rare. Relapses are, however, more frequent.

4. Typhus Ambulatorius.—*Typhus ambulatorius* (of the German, or latent typhoid of French writers—walking typhoid), occurs in adults, especially in men, who often endure it by the help of alcohol. Some malaise may exist. There are two types: (*a*) the lighter and atypical, and (*b*) the usual course, very often ending in hemorrhage, perforation, etc. The temperature is often not marked, but is usually less at night than during the diurnal activity. The pulse is faster because of exertion. The spleen is often enlarged and the roseolæ are frequent.

5. Typhus Afebrilis.—*Typhus afebrilis* (Liebermeister and Gerhardt) is most often met with in family practice. It may assume the ambulatory form, and is diagnosticated by exclusion and by the pulse, spleen, roseolæ, diazo reaction, Widal test, etc. Atypical cases have apparently increased in recent years.

6. Hemorrhagic.—The hemorrhagic form is characterized by nasal, subcutaneous, and particularly intestinal hemorrhage, constituting the blood dissolution of the older writers. It occurs especially in children and alcoholics, and constitutes less than one-tenth of 1 per cent. of typhoid cases. The prognosis is usually but not invariably bad. The four cases which the author has seen occurred in 1900, in St. Luke's Hospital, where a severe house-epidemic prevailed.

7. Visceral Forms.—The so-called *visceral forms* should seldom cause confusion, if watched with care. They include the gastric or bilious types, nervous fever, meningo-typhoid, cerebro-typhoid (mania, psychoses), laryngo- or tonsillo-typhoid, nephro-typhoid, pneumo-typhoid with pneumococcus mixed infection or Eberth's bacillus (Lépine, Chantemesse, Widal), which is pneumo-typhoid in the narrowest sense.

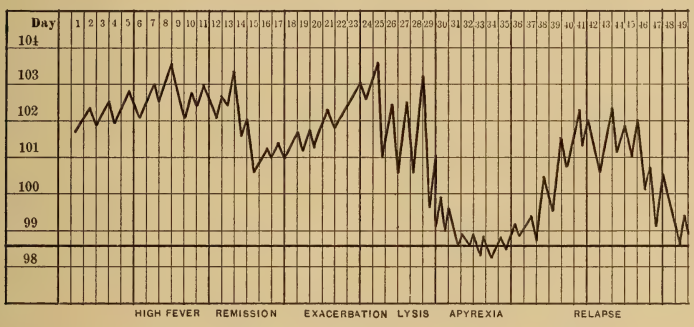
8. In Children.—In children but few cases occur in hospitals. The temperature course is shorter and milder than in adults. The younger the patient, the less is the degree of fever. It may begin with a chill. It is often remittent, the "infantile remittent fever" of Abercrombie being really typhoid. Epistaxis is less common. The pulse in children is faster, and more often irregular, than in adults, though after the twelfth year, it is equally slow. A weak and irregular pulse is very rare, and a fast, irregular pulse generally indicates meningitis. Opinions differ as to the frequency of roseolæ, but it is probable that they occur as often in children as in adults, and in some cases are abundant and confluent (in children between the fifth and tenth year). Bedsores are very rare, but noma is more frequent than in adults. The nervous system is generally less frequently involved in children, though if the infection is severe, convulsions, irregular pupils, rigidity of the neck, headache, etc., may occur. (See Typhoid State.) Neuralgias and psychoses are rare. The frequency of aphasia has not yet been explained. Bronchitis occurs as often as in adults, but atelectasis, hypostasis, and lobular pneumonia are more frequent. Children exhibit less meteorism and hemorrhage (1 per cent.), and much less frequent perforation. There are fewer anatomical changes. Splenic enlargement is less frequently palpable (in 50 per cent.), and occurs somewhat later. Initial vomiting, pain, and perhaps diarrhœa, are more frequent. Albuminuria and nephritis are relatively rare. *In infants.* Griffith and Ostheimer has assembled 394 cases in infants, of which 325 are beyond doubt. Of these, 139 occurred under one year, 187 in the second, and 68 between two and two and one-half years. The mortality under one year (*v. i.*) was 73 per cent., and of the entire group, 50 per cent. Typhoid is rare in the first two years of life.

It is sometimes overlooked, being very atypical. The course is short. Bronchitis, splenic tumor, and roseolæ are infrequent. If the disease is severe, a very high temperature, meningeal symptoms, and initial vomiting occur, and also 50 per cent. mortality caused by toxæmia, pneumonia, perforation, or ulceration of the larynx.

9. In the Aged.—Typhoid is infrequent after the fortieth year. The fever is lower, atypical, and often absent. The pulse is faster, irregular, and without dicrotism. There is greater lung congestion owing to weakness of the right heart, and lung complications are more frequent. The nervous symptoms are dominant, often occurring with great depression. Roseolæ are fewer and bedsores are more frequent. The spleen is less often enlarged because of old infarcts, induration, and a thicker capsule. Tympany and hemorrhage occur more often. Convalescence is naturally slower. In 1903, the author had under his care, in Wesley Hospital, a woman, aged fifty-seven, and her mother, aged seventy-two, both with typhoid, ending in recovery.

Relapses and Exacerbations.—Relapses and exacerbations differ in degree only. Their explanation is still obscure but is probably in-

Fig. 5.



Showing remission, exacerbation, lysis, and relapse.

volved in the immunity problem. In the *relapse,* the temperature falls to normal, or nearly to normal, for five days on the average, and then fever and other signs of typhoid reappear. It occurs in 9 per cent. and varies with the epidemic, sometimes occurring in as much as 35 per cent. Relapses are more common in young (in one-sixth of Henoch's cases) than in old subjects. They are more likely to occur (*a*) in light or moderately severe cases (75 per cent.) than in severe ones (25 per cent.); (*b*) when the splenic tumor does not disappear (Gerhardt, but the question may arise whether the spleen was enlarged from some previous disease, such as malaria); (*c*) when the temperature in convalescence does not become subnormal (Curschmann); or (*d*) when the pulse is variable and rapid while the patient is quiet (Curschmann).

After an average defervescence of five days, the fever runs the same course as in the first attack, though it is shorter by ten days (Murchison). The pulse is faster (110 to 130 in women, children,

and weak men), labile and less dicrotic. There are less roseolæ, nervous symptoms, respiratory symptoms, hemorrhage (4 per cent.), and perforation (0.7 per cent.), whence, as a rule, the prognosis is relatively good. The duration of the relapse is six to fifteen (even twenty-one days), and there are seldom more than two,—three or four being very rare. The author has seen five relapses, covering, with the original attack, as many months. It is said that they may be afebrile, with a return of the roseolæ, spleen, etc. Relapses should not be confused with Biermer's "after fever," due to inanition or possibly, sepsis. *Recrudescence* is renewal of the fever after it has lessened, but not entirely subsided. In 50 per cent. recrudescence is followed by more severe symptoms.

Convalescence.—Convalescence requires fourteen to twenty-one days, but its duration is variable, and it is not certainly established till subnormal temperature, at least in moderate or severe infections, is observed. The pulse tends to run high on exertion or excitement. Anæmia, emaciation, œdema, phlebitis, tuberculosis, neurasthenia, psychoses, bone, muscle, and other complications, may intervene. If the appetite is poor in convalescence, complications are to be expected.

Diagnosis.—General Rules.—1. The bacteriological diagnosis is not usually practicable for the inexperienced physician. Splenic puncture is never permissible, though positive in 95 per cent. of cases tested. The bacilli may be found in the blood in nearly all cases, and practically always in the roseolæ.

2. Observe long and carefully. Say that "a diagnosis is impossible in the first few days."

3. No single sign is absolutely diagnostic, except bacilli in the blood, etc., and the characteristic Widal reaction.

4. Diagnosticate (*a*) from positive findings, and (*b*) by careful exclusion.

5. The "typhoid state" only indicates toxæmia, not necessarily typhoid.

6. In doubtful cases remember the great statistical frequency of typhoid.

7. Remember its manifold forms; *e. g.*, pneumo-typhoid, etc.

8. Be on the safe side in light, ambulatory, or abortive cases as to the diagnosis and the still more important matter of treatment.

The Cardinal Signs.—These are: (*a*) The Widal test; (*b*) bacilli in the blood, urine, roseolæ, etc.; (*c*) the roseolæ; (*d*) the slow pulse; (*e*) the fever-curve; and (*f*) the splenic tumor.

(*a*) The sero-test, successful in 95 to 97 per cent. of cases, is important. As stated above, its absence does not exclude typhoid.

(*b*) The bacilli in the blood are found in practically all cases.

(*c*) The eruption has a diagnostic value in its location, occurrence in crops, time of appearance, and short duration.

(*d*) The slow pulse, and to a much less extent its dicrotism, are suggestive. It is rarely to be confused with the slow pulse of tuberculous meningitis.

(*e*) The fever curve shows a gradual, step-like onset, continuous fastigium, steep curves in the breaking stage, and subnormal register in convalescence.

(*f*) While the splenic tumor occurs in many other infections, it is *per se* suggestive after the first week.

Secondary Signs.—These are, in order of importance, the typhoid tongue, the stools, bronchitis (especially after the first week), the typhoid state, leukopenia, intestinal symptoms and complications, and the diazo reaction.

Symptoms Contra-indicating Typhoid.—The symptoms contra-indicating typhoid are, according to Leube, coryza, early vomiting and sweating, herpes, endocarditis, retraction of the abdomen, persistent constipation, and constantly negative diazo test. To these may be added leukocytosis (unless some complication has occurred), icterus and rigors. Any of the above symptoms may, exceptionally, complicate typhoid.

Differential Diagnosis.—Differentiation from acute miliary tuberculosis may be most difficult. In both diseases protracted fever, splenic tumor, brain symptoms, bronchitis and bronchopneumonia, rapid pulse, etc., may be observed, and miliary tuberculosis may develop from typhoid (Birsch-Hirschfeld, and in two personal observations). Typhoid may be complicated with septicæmia, very rarely with malaria in this climate, or with meningitis. The table on pages 42 and 43 illustrates the differential diagnosis of the diseases most likely to be confused with typhoid.

Exanthematous typhus (*q. v.*) and recurrent fever (*q. v.*) are easily distinguished. The *exanthematous diseases,* such as scarlatina and measles are rarely confused, except, perhaps in the initial stage. In 75 instances typhoid and scarlatina co-existed (Fournier). An extensive eruption of roseolæ resembling measles is easily distinguished by the course, temperature curve, and catarrhal symptoms.

Fibrinous pneumonia rarely resembles typhoid, and still more rarely occurs with typhoid in its earliest stages by the localization of typhoid bacilli in the lungs. Though typhoid may begin with severe bronchitis or pneumonic symptoms, doubt usually exists only during the first few days. The intestinal symptoms and cardinal findings of typhoid usually appear after a few days when all doubt is dispelled. Pneumonia undoubtedly may occur without local findings, yet the diagnosis of central pneumonia is usually incorrect. Sudden onset with chill, pain in the side, sudden rise of pulse and temperature, rapid respiration, bloody, viscid sputum, herpes, leukocytosis, and signs of local infiltration or a tympanitic note and a few crepitant rales over one lobe, followed later by some typical pneumonic infiltration as the disease advances from within toward the periphery of the lung, establish the diagnosis of pneumonia on the second or third day.

Intestinal anthrax is accompanied by fever, splenic tumor, and nervous symptoms. Hemorrhagic vesicles in the mouth, vomiting, colic, hemorrhage from the intestines and kidney, cyanosis and

Pathognomonic Signs		Pulse and Circulation	Fever	Splenic Enlargement	Blood
Typhoid.	Typhoid bacilli in the blood, spleen (by puncture), urine, roseolæ, or stools. Widal test.	Relatively slow dicrotic pulse early in the disease, especially in men, and in not too severe infections. Endocarditis and pericarditis very rare.	Typical rise, acme, defervescence, and subnormal in convalescence. Remissions and intermissions following continuous fever speak for typhoid. Responds to hydrotherapy as a rule. Sometimes chills at onset or in course.	Very frequent; larger than in miliary tuberculosis; appears in first week.	Bacilli in blood, early; Widal test later. No leukocytosis unless complications, but leukopenia. Polymorphonuclears decreased. Late anæmia.
Miliary Tuberculosis.	Tubercle bacilli in the blood or splenic pulp; tubercles in the choroid.	Rapid, 120 or more, high even during remissions; slowed in tuberculous meningitis and in the aged. Dicrotism uncommon. Sometimes pericardial rub.	No cycle; irregular or remittent. Seldom long continued, and if so, longer than in typhoid. Exacerbations with renewed dissemination of tubercle bacilli. Brain involvement may lower fever or make it variable, as inversed type.	Less frequent; smaller and usually appears later.	Sometimes tubercle bacilli in blood. No Widal. Leukocytosis not often, but more than in typhoid.
Malaria.	Plasmodium in the blood (or splenic pulp).	Pulse moderate in rate; sometimes dicrotic; very rarely endocarditis.	Intermittent, beginning very often with chills; type varies (quotidian, tertian, etc.); often rises high at once; if continuous (subcontinuous), at least made irregular by quinine or usually wholly abolished (for a time at least).	Much enlarged; more than in typhoid; and persists frequently as ague-cake.	Plasmodium, melanæmia, no leukocytosis save in pernicious forms. Great and early anæmia. No Widal.
Sepsis (including Endocarditis).	Cultivation of pyogenic organisms, pneumococcus, from the blood or various foci.	Usually fast and irregular; cardiac localizations very frequent in sepsis, puerperal especially, and in endocarditis (a cardiac form of sepsis). Cardiac enlargement, thrill, bruit, accentuated pulmonic tone, etc.	Chills, sweats, steep elevations, and sudden depressions almost always at some time, and usually repeated with each discharge into the blood of infective material.	Especially enlarged from sepsis and infarcts. Also often the lymph-nodes.	Various organisms. Marked polymorphonuclear leukocytosis very frequent; sometimes absent in endocarditis. No Widal.
Meningitis.	Lumbar puncture (*a*) meningococci, tubercle bacilli, pyogenic organisms, etc.; (*b*) increased, turbid fluid, etc.	Rate differs with type; variable.	Differs with variety, but seldom as high as typhoid; no cycle.	Rare.	Leukocytosis in epidemic and purulent types; rarer in tuberculous. No Widal.

Skin	Lungs	Nervous System	Sputum, Urine	Alimentary Tract
Roseolæ, typical in location, crops, appearance, containing typhoid bacilli. Sweating and miliaria less often and later. Icterus rare. Herpes rare.	Breathing somewhat increased from toxæmia (cerebral breathing), hypostasis, or other complications. Rales below and behind. Tympanitic note never heard in typhoid.	Early headache, replaced later by delirium, typhoid state, actual meningitis very rare; intoxication may closely simulate meningitis. Very rarely disk changes.	Typhoid bacilli in urine. Diazo test in nearly all cases.	Mouse-like breath, semi-characteristic tongue, ochre-yellow stools, tympany, diarrhœa, bowel hemorrhage, perforation, bacilli in stools. Retracted abdomen or early vomiting rare.
Roseolæ absent or extremely rare, and then not in crops. Drenching sweats common. Herpes more than in typhoid. Skin anæmic and very often cyanotic.	Breathing increased even 40 to 60; cyanosis and dyspnœa without adequate physical findings. Fine and localized rales heard above and in front. Tympanitic percussion note from lung relaxation. Evidences of pleural adhesions, apical changes, fresh pleural rub.	Meningitis tuberculosa very frequent; optic neuritis; choroidal tubercles.	Tubercle bacilli sometimes found in sputum, urine and fæces, but are indicative of old tuberculous lesions, not of miliary tuberculosis. Diazo test frequent.	Diarrhœa not common, but may rarely occur, as indeed may hemorrhages; both may come from tuberculous ulceration of bowels.
In severe, eyes subicteric or markedly icteric. Herpes very frequent and urticaria the next most frequent exanthem.	If lung congestion (severe forms), it is variable, recurrent, shifts from lobe to lobe.	Headache at onset is rare; later is often pulsating or neuralgic. Delirium from beginning in æstivo-autumnal forms is frequent. Restlessness, anxiousness. In pernicious forms may have convulsions, coma, etc.	Hæmoglobinuria in pernicious malaria. Diazo test fairly frequent, though much less than in typhoid.	Nauseating breath. Sometimes abdominal pain even in mild types; may rarely have diarrhœa, even hemorrhage in alimentary pernicious types. Liver congestion frequent, also icterus.
Frequent sweating, miliaria. Septic (polymorphous) rashes. Subicterus, frequently purpuric skin eruptions, herpes not uncommon.	Bronchitis frequent. Pulmonary infarcts and pleurisy.	Dependent on cause. Septic meningitis (with arthritis, pleurisy, etc.), cerebral embolism; retinal hemorrhages or embolism.	Acute embolic nephritis. Peptonuria.	Septic diarrhœa.
Herpes in epidemic cerebrospinal form. Tache cérébrale.	Variable breathing; Biot's breathing.	Early triad, headache, retracted neck and hyperæsthesia. Headache violent throughout the disease, convulsions, paralysis, Kernig's sign, etc. Optic neuritis, retinal hemorrhage, tubercles in choroid.		Early cerebral vomiting. Retracted scaphoid abdomen.

dyspnœa, are suggestive, and the detection of the anthrax bacillus in the blood, urine, stools, or sputum, and the results of inoculation, establish the diagnosis.

Febrile gastritis may rarely simulate typhoid, but the fever is usually low or irregular, and is more often absent than present. Marked early gastric symptoms are not common in typhoid. *Febrile enteritis* is usually characterized by much mucus in the stools. Though typhoid may occur with an old enterocolitis, the typhoid stool seldom contains much mucus. Splenic tumor is most uncommon in enteritis. The fever in enteritis is lower or more irregular than in typhoid; and the bronchitis, the slow pulse, and the other cardinal signs of typhoid, are absent. Cases of *typhoid developing suddenly* may be, at first, confused with grippe, diphtheria, or other diseases. *Febrile syphilis* seldom causes diagnostic trouble but must be considered in dubious cases.

Paratyphoid is due to a bacillus differing from the bacillus of typhoid and of dysentery, and from the Bacillus coli, and resembling the Bacillus enteritidis, Bacillus psittacosis, etc. Two types of organisms are recognized—one producing a permanent and the other a transient reaction in milk. Infection may occur from milk, water, and tainted meat. The bacilli are found in the stools, blood, roseolæ, and urine, and agglutinate in higher dilutions (1 to 1,000 or 2,000) than the typhoid bacilli. The first cases were reported by Schottmüller, in 1900. In paratyphoid the temperature is more brusque in onset and more irregular than in typhoid, shows greater remissions, and may end by crisis. The pulse is slow and dicrotic. Roseolæ are often present (80 per cent.), the spleen is enlarged less often and later, and no leukocytosis is observed. Prodromal catarrhs, diarrhœa, cervical adenitis, œdema of the lids, vomiting, rigors, epigastric pain, herpes, myositis, arthritis, pleurisy, endocarditis, and meningitis, are more common than in typhoid and enable the practitioner to differentiate paratyphoid, clinically. The diazo reaction is positive in 30 to 60 per cent. The clinical course covers two weeks. Relapses are common. The infection is usually mild, sometimes severe, and rarely fatal (3.6 per cent.). Ulceration of the intestinal lymph-structures is absent in the fatal cases, though other necroses occur, thus explaining the occasional hemorrhage (5 per cent.). Pneumonia and perforation are unknown. Nervous symptoms are not prominent. The disease is comparatively rare. The blood does not respond to the Widal test nor are typhoid bacilli found. Grunberg and Rolly hold that the Widal reaction is less a specific than a group reaction, as they found agglutination in the paratyphoid group by typhoid serum. This does not imply, however, that failure to respond to the Widal test excludes typhoid (Johnstone). A mixed infection with typhoid may occur.

Trichinosis is differentiated from typhoid by the history, by muscular pain and œdema in the striped muscles, by trichinæ in the stools and muscles, and by eosinophilia. In typhoid these symptoms are ab-

sent, while the Widal reaction, rose spots, and enlargement of spleen, are present. Three cases of double infections have been reported (McCrae).

Prognosis.—The death-rate was formerly 20 per cent., based on 50,000 cases collected by Jaccoud. At present it is lower (5 to 12 per cent.) because of (*a*) more accurate diagnosis, thus including lighter cases in our figures; (*b*) the virulence of typhoid becoming less; (*c*) better hygiene; and (*d*) better treatment, Brand method, etc.

The *prognostic factors* are:

1. Age: The least mortality is between the second and the tenth years; the highest after the twenty-fifth. The very highest mortality is in babes.

2. Constitution: Meagre and muscular patients are more likely to recover than the plethoric, or adipose.

3. Sex is immaterial, aside from pregnancy.

4. Acute diseases modify the prognosis,—e. g. scarlatina, measles, variola, diphtheria, malaria, erysipelas, etc.

5. Chronic diseases, such as gout, diabetes (low temperature, the sugar often disappearing), cardiac, pulmonary, and renal disease; alcoholism (34 per cent. mortality from weak heart or hemorrhagic diathesis); convulsive affections, such as epilepsy or chorea, may cease.

The prognosis is better in relapses than in the original case. Recrudescences are dangerous. In war times the prognosis is usually less favorable; the Spanish-American war was an exception, the death-rate being but 7 per cent., while in the Boer war the mortality was 21 per cent.

Death usually occurs between the eighteenth and twenty-eighth days, and is caused by—

1. *Toxæmia*, the most frequent cause of death (30 to 50 per cent.), including: (*a*) Hyperpyrexia; (*b*) hemorrhagic types; (*c*) heart weakness, vasomotor paralysis; and (*d*) brain paralysis.

2. *Local typhoid lesions,* as meteorism, diarrhœa, peritonitis (in 16 per cent. of fatal cases), or hemorrhage (in 20 per cent.).

3. *Complications,* as lung inflammations (in 15 per cent.), nephritis (4.5 per cent.), etc. Septicæmia has been little studied except by French writers (“*forme septicémique généralisée*”), the symptoms of which are high fever, chills, sweats, abscesses, enlarged glands and spleen. Pyæmia may result from acute osteomyelitis, suppurative parotitis, adenitis, phlebitis, or splenic infarction.

4. *Collapse. Sudden death* has been discussed under changes in the heart muscle. Dieulafoy estimates that sudden death occurs in 2 per cent. of typhoid cases. The pulse is always more important than the fever, and early rapidity is unfavorable. Bradycardia and marked agglutination are favorable prognostics.

5. We have already considered the ominous significance of pseudo-collapse, the typhoid state, early delirium, diarrhœa, and meteorism; of the various severe complications, such as noma, gangrene, laryngeal perichondritis, and pneumonia; and of the more severe types, such

as the hyperpyretic and hemorrhagic; and of the ambulatory varieties. Typhoid beginning suddenly entails a high mortality.

Treatment.—Dujardin-Beaumetz said: "The best treatment for typhoid is a good physician." The treatment concerns (*A*) prophylaxis; (*B*) specific treatment; (*C*) hygiene and rest; (*D*) the question of intestinal antisepsis; (*E*) antipyretic measures (hydrotherapy); (*F*) diet; (*G*) symptomatic or expectant treatment; (*H*) treatment during convalescence.

(*A*) Prophylaxis.—Prophylaxis has as wide a scope in medicine as asepsis and antisepsis in surgery. In Chicago, during the years 1889 to 1892, inclusive, there were 4,747 deaths from typhoid. In the four years after the drainage-canal was opened there were 2,235 cases; that is, per population, the mortality was reduced 75 per cent. In 1905, typhoid cases were rare. General prevention is difficult, because of the ubiquity of the germ and the carelessness of individuals. Systematic coöperation regarding *sewage* regulation and *water supply* could efface typhoid. Koch has particularly emphasized this point lately and succeeded in wiping out certain typhoid foci. One difficulty lies in the infrequent recognition of light cases, which are particularly dangerous, another in the selfish carelessness of many persons, and a third in the fact that some individuals "carry bacilli" for years. Such persons should be quarantined. It is easy to boil water for home use, and yet it is done in but few families, even in those of physicians. The traveler should avoid unboiled water and milk. In the treatment of a case, everything with which the germ may come in contact must be sterilized. Next to the fæces, the urine is the most important conveyer of the bacillus. They are the usual means of dissemination, and their proper disinfection in every case would surely obliterate typhoid. The bacilli may persist for years in the fæces and urine. Hexamethylenamina (urotropin) is recommended for such cases. Chloride of lime should be placed in the bottom of the urinal, or bed-pan, before and after use. Acidulated sublimate (1 to 500) or 10 per cent. crude carbolic solution may also be used; but neither at once disinfects, for which reason the dejections should stand an hour or two in about a quart of the disinfectant. Both the pans and urinals must then be sterilized. The same urgent disinfection is indicated in the case of thermometers used in the rectum, and rectal syringes or tubes. The perineum, after each movement, should be sponged with a 1-to-2,000 bichloride solution, and also the back and thighs, when there are involuntary evacuations. The rubber draw-sheets and linen should be sterilized regularly with carbolic acid or bichloride of mercury, sheets and linen should be sterilized regularly with carbolic acid or bichloride of mercury, and the blankets, pillows, and mattresses, steamed when taken off the bed. The bath-tub should be carefully cleaned with carbolic solution, as the patient's soiled skin and the urine often voided during the bath are obvious sources of

danger. It seems superfluous to urge cleansing the hands of the nurse or physician after examination or tubbing, but the not infrequent direct transmission of the disease to nurses, orderlies, and doctors, is often forgotten. Rubber gloves are the safest protection. Direct infection is growing in clinical importance. In the Spanish-American war 20 per cent. of our soldiers were directly infected by flies, and in the Boer war more died of typhoid than were killed by bullets, infection being carried by flies and sand. The few cases among the Japanese is one of the many lessons they taught us. Flies should be carefully excluded from the house by screens.

In convalescence the room should be fumigated with formaldehyde, not by the useless sulphur method (see SMALLPOX).

(*B*). SPECIFIC TREATMENT.—Specific treatment awaits the further development of serotherapy initiated by Chantemesse, Widal, Brieger, Wassermann, Wright, and others. Preventive inoculation in India and South Africa reduced the percentages of typhoid morbidity from 14 to 2 per cent., and the mortality from 3 to ½ per cent.

(*C*). HYGIENIC TREATMENT.—Absolute physical and psychical rest is imperative, and the best results are obtained in cases which have been put to bed early. Cases of the ambulatory type never fare as well. The invariable use of the bed-pan and urinal should be insisted upon, as every patient can learn to use them. In all infections, the furniture, carpets and hangings, should be removed from the room, and it should have free ventilation,—fever patients do not catch cold. The nurse should have written instructions. The stools, baths, etc., should be watched or directed by the physician, especially at the onset. The single bed should be low, should have a firm mattress, rubber draw sheet, and perfectly smooth cotton or linen sheets. A second bed is desirable for change and convenience.

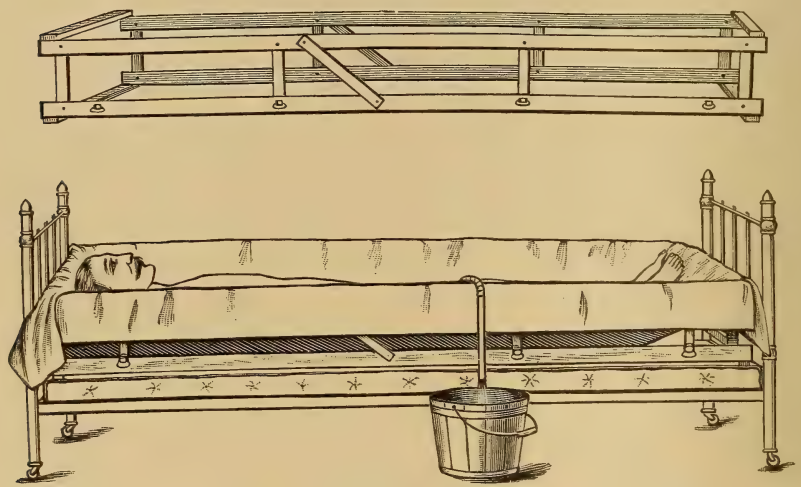
(*D*). ANTISEPTIC THERAPY.—Antiseptic therapy is largely of historical interest, as typhoid is not a local enteric disease but a septicæmia or bacillæmia. Calomel was given early in typhoid by von Ziemssen and Liebermeister, who thought that it reduced the virulence of the attack and lessened bowel symptoms and complications; it is unnecessary, and at the acme is actually harmful. Salol (phenylis salicylas) is the least objectionable, since it is mild; it is given in doses of grs. x., t. i. d. The urine should be closely watched for signs of renal irritation. Dilute hydrochloric acid, $\overline{3ss}$ after each feeding, prevents diarrhœa and tympany. No one of this group of agents essentially influences the clinical course. They are efficacious solely against the common organisms of fermentation.

(*E*). ANTIPYRESIS.—(1). *Drug Antipyretics.*—These are seldom indicated, for they merely reduce fever and do not eliminate its cause, the toxins. Quinine is recommended by Liebermeister and Curschmann, in one or two doses of fifteen to twenty grains, given before the fever reaches its highest point. No cardiac depression results, and its effects last from eight to twelve hours; the fever, however, is but moderately influenced by quinine. Coal-tar products,

in full repeated doses, depress the brain, circulation, respiration, and metabolism; cause vomiting and eruptions; increase the danger of collapse; and are not indicated in typhoid as they are in the fugitive fevers. In practically all cases, drug antipyresis is far inferior to—

(2). *Hydrotherapy.*—Hydrotherapy, which is the only logical method of reducing temperature, since it alone eliminates the fever-exciting toxins. Nature's method of reducing temperature by sweating should not be ignored. Evaporation from the moist skin should be helped by removal of the bed-clothing. (*a*) The *full cold bath* of Currie (1787) and Brand is given with water at 64° to 68°; it lasts fifteen to twenty minutes and is repeated every three hours

FIG. 6.



Burr's bed.

for temperature of 102° or over. Chilling is averted by vigorous friction, which must not abrade the skin; by cold affusions to the head; or by administration of alcoholic stimulants. The apparent brutality of the Brand method has excited considerable popular and professional prejudice, but it is more valuable than any other measure in typhoid. As Jürgensen remarks, every deviation from the simple principles above outlined gives less satisfactory results. Early resort to hydrotherapy gives the best results. When the portable bath-tub is not available, the method of A. H. Burr is equally good. He uses a wooden frame, shaped like a large box; the base fits over any bed; its upper part sustains a heavy rubber sheet, which passes under the patient and constitutes the sides and bottom of the tub (Fig. 6). Its use obviates the necessity of lifting the patient, and saves excitement. Water is poured over the patient from pails and is removed with a siphon. The *advantages* of Brand's method are: (i) The mortality is lowered 6 to 8 per cent.; (ii) toxæmia is less-

ened, and therefore nervous and digestive symptoms, heart weakness, and pulmonary complications, are relieved; fever is successfully reduced, and there is less visceral degeneration; (iii) the renal secretion is stimulated, thereby washing the toxins out of the blood; (iv) the skin becomes more active and bed-sores less frequent, although furunculosis is seemingly increased; (v) the clinical course is shorter, and, on the whole, fewer complications occur, although the percentage of hemorrhage and perforation is not lowered, and relapses seem slightly more frequent. *Contra-indications* to the use of Brand's method are hemorrhage, perforation, great prostration, alcoholism, advanced arteriosclerosis or cardiac disease, and marked phlebitis. Pregnancy, nephritis, and pneumonia, are not contra-indications. Various *substitutes,* for Brand's method, though less effectual, may be indicated for special reasons:—(*b*) The *gradually cooled bath* is identical with the Brand method, except that the temperature of the water is gradually reduced from 90° to 75° or 65°. It is adapted to cases coming under treatment late and to those with great dyspnœa, or tendency to collapse. (*c*) The *protracted tepid bath.* (*d*) *Cold sponging,* first with warm water to dilate the skin-vessels and then with cold water to abstract heat. Equal parts of alcohol and water should be mixed, to aid evaporation. One part after another of the body is exposed and sponged. It is a refreshing and quieting placebo, but enormously inferior as an antipyretic. (*e*) The *cold pack* is also much less successful. In sthenic cases, ice applications to the head, axilla, spine, etc., may be used, or the body may even be ironed with ice. Ice-water enemas are never used in typhoid, though sometimes employed in other fevers.

(*F*). Diet.—Fifty ounces of water should be given daily at definite intervals. It flushes the kidneys, lowers temperature by increasing the sweat production, and lessens parenchymatous degeneration of the viscera. Pure water is superior to barley- or albumin-water, lemonade, etc., which dull the appetite and often derange digestion. Care of the mouth may be considered here. The mouth and pharynx should be carefully cleansed of sordes and food particles lest infection travel to the ears, salivary or lymphatic glands. A boric-acid solution, with tincture of myrrh or tincture of hydrastis, meets every requirement. The liquor antisepticus, U. S. P., contains 2 per cent. boric acid and antiseptic oils. The cleansing must be gentle lest fresh abrasions be inflicted. The diet must be fluid, since there are intestinal lesions. It must also be easily digestible, since fever lessens all the digestive juices. In the diet no reference is made to individual symptoms, as there is no correspondence between the clinical symptoms and the anatomical ulceration of the intestines. Graves was the first to feed fever patients, who up to that time had been starved, but there is, at the present time, almost equal danger of overfeeding. Milk is the standard diet. Six to eight ounces should be sipped at intervals of three hours, day and night, as the patient, after being wakened, readily falls to sleep again. It

is best given diluted with half an ounce of lime-water or with a little carbonated water, which should not be cold, since the gas expands at body heat. Though milk alone is said to contain insufficient albumin to meet the tissue-waste, there is risk of overfeeding; the physician himself should inspect the stools for curds, for milk, curdling in the stomach, practically becomes solid food. As in infant feeding, curds indicate skim-milk. Very few patients cannot take milk. In other words, it can be forced upon unwilling patients. Recalcitrant patients may be given some variation, such as koumyss, matzoon, buttermilk, whey, or certain baby-foods. If the digestion is peculiar or weak, peptonized milk, given with very thin, strained gruels, or egg, may be tolerated. Carbohydrates are theoretically indicated to lessen nitrogenous waste, and well-boiled cornstarch and tapioca are allowed by some clinicians. They are permissible if no tympany results, and the author agrees with Hare that taka-diastase aids their assimilation. Meat-juice is obtained from freshly slaughtered beef, by a meat-press. If its taste is disagreeable to the patient, a few drops of peppermint oil may be given first. It may also be given frozen (meat juice, ʒ iij.; sugar, ʒ ij.; lemon-juice, ʒ j.; yolk of one egg; cognac, ʒ j.). The following articles afford some variety: Nutrose, somatose, eukasin, yolk of egg in soup or wine, and white of egg with twice its weight of water. Foods containing gelatin conserve metabolism (Senator) but may irritate the bowels. Beef-tea should be avoided, as it contains only stimulating substances, less than 1 per cent. albumin, and no fat or carbohydrates and may irritate the kidneys and intestines. In stupid patients with anæsthetic throats, food may enter the larynx and cause aspiration pneumonia. The nasal catheter is then indicated. To its outer end a funnel is attached, through which food, drink, and medicines, are given. A safety-pin across its outer end prevents its escape downward. The catheter should be long enough to pass into the œsophagus beyond the larynx, and care should be exercised that it does not enter the latter. Rectal feeding is sometimes indicated.

(*G*) Symptomatic or Expectant Treatment.—1. *Digestive System.* (*a*) *Vomiting.*—It must be ascertained that the patient has been given no solid food by solicitous friends and that perforation has not taken place. Rectal feeding, the use of warm carbonated water, cocaine gr. ¼, or tincture iodine ♏ ½, for several doses, sinapisms or an ice-bag to be placed on the epigastrium, are indicated. Bartholow's formula is very serviceable:

℞ Phenolis ..... gr. iv.
Bismuthi subnitratis ..... ʒiss.
Cerii oxalatis ..... ʒj.
Sodii bromidi ..... ʒss.
Sacchari albi }
Acaciæ } ..... āā q.s.
Aquæ menthæ piperitæ ..... q.s. ad ʒij.
M. et ft. emulsum.
S.—One teaspoonful, undiluted, every 15 to 30 minutes.

(*b*) *Constipation* should be treated with enemas of water or sweet oil every other day. Typhoid cases with constipation do better than those with diarrhœa. Cathartics are never used, as they cause meteorism. (*c*) *Diarrhœa* is most frequently due to toxæmia and is, therefore, most efficaciously treated with the cold bath; curds (prevented by diluting or skimming the milk), beef-tea, impure milk, or solid food, may be its cause. Treatment is indicated when there are more than four movements daily, and when there is colic, tenesmus, unrest or weakness. Cold tea with whiskey and pulv. aromaticus gr. xv., or paregoric and bismuth each ʒj., after each movement, extr. opii. gr. i., or the starch-and-laudanum enema usually give relief at once. Pepper considered silver nitrate almost specific (gr. $\frac{1}{4}$ to $\frac{1}{2}$).

℞ Bismuthi subnitratis ..... ʒj.
Morphinæ sulphatis ..... gr. j.
M. et div. in pulv. xij.
S.—One to four a day. (Alonzo Clark.)

℞ Acidi tannici ..... gr. x.
Tincturæ opii ..... ♏vj.
Olei terebinthinæ ..... ♏v.
Mucilaginis ..... ʒij.
Aquæ menthæ piperitæ .....ad. ʒj.
M. f. haust.
S. To be taken every two hours. (Murchison.)

℞ Plumbi acetatis ..... gr. iss ad iij.
Opii pulveris ..... gr. i ad ijss.
Sacchari albi ..... gr. lx.
M. et divide in pulv. vj.
S.—A powder every three hours. (Yeo.)

(*d*) *Tympany.*—This is sometimes an indication that the patient has been given solid food. In severe cases albumin-water must be substituted for milk. The cold bath often gives relief, since most cases of meteorism are due to toxæmia. Hydrochloric acid after feeding may lessen the tympany. The turpentine stupe of Jenner should be applied to the abdomen, flannel cloths being dipped in a mixture of warm water and ʒss ol. terebinthinæ and pressed hard together to spread the oil evenly and thus avoid blistering. The ice-coil is also beneficial. The old turpentine emulsion of Wood (emulsum olei terebinthinæ, ʒj.) is excellent, though the urine must be watched carefully for signs of renal irritation:

℞ Ol. terebinthinæ ..... ʒiss.
Ol. caryophylli ..... gtt. vj.
Glycerini } .....āā ʒss.
Mucilaginis acaciæ }
Syrupi et aquæ .....āā ʒiss.
M. et ft. emulsum.
S.—A dessert spoonful every 3 or 4 hours.

Whiskey and spices are effective. Emulsum asafœtidæ, Oj.; spiritus chloroformi, ʒij.; and ol. terebinthinæ, ʒss; as a warm enema, often give relief. (*e*) *Hemorrhage* necessitates absolute quiet, men-

tal, physical, and alimentary. The patient should not be moved for any reason whatever. In severe cases he should lie in the blood which has been passed, with cotton tucked around the hips and between the legs to catch the urine and fæces, which should pass into it without his being lifted on the bed-pan. No food, no water, no enema, no bath, should be given. Morphine is primarily indicated. It quiets the excitement attending hemorrhage, lessens peristalsis and enforces bodily quiet. It should be given in sufficiently large amounts, gr. ¼ being injected every half-hour for three doses, then a quantity sufficiently large to produce its full physiological effects, for the dose of a drug is not measured in grains but in its effect; it should be repeated every two, four, or six hours in order to sustain the primary indications above named. The author has given even four to six grains in the first day, when the greatest danger occurs, but in such cases the patient must be very closely watched. The author considers that the sole valid objection to morphine is that it may mask coincident perforation, which occurs once in every five cases of hemorrhage; he believes, however, that close circumspection invalidates this objection. Opium or styptics by the mouth are dangerous and uncertain. The lead- or iron-salts probably never reach the bowel. The ice-coil placed on the abdomen may be beneficial. Gelatin may be injected as in aneurysm (*q. v.*). The question of stimulation is a difficult one. On the one hand, there is the risk of forcing blood through the leak by stimulation; on the other hand, the volume of blood must not sink too low; but first the heart must be allowed to run down,—nature's own and most certain method of hemostasis. At a later time only, should strychnine or digitalis be administered hypodermatically, or saline transfusions be given; delay in their use is better than haste. (*f*) *Perforation* necessitates an early diagnosis and immediate operation. Most reported recoveries were operated on in the first twenty-four hours. Recovery occurred in 30 per cent. of 358 operations (Cazin). In twenty cases operated on at Johns Hopkins Hospital, 35 per cent. recovered.

2. *Circulatory System.*—Heart weakness is best prevented by absolute rest, and hydrotherapy. An ice-bag placed over the heart tends to regulate and calm its action. *Strychnine,* gr. $\frac{1}{30}$, is the best heart tonic, and may be given two or three times a day as a vasomotor tonic in cases which do not show signs of heart-failure, but it is best to await definite indications for stimulation. Digitalis is rarely beneficial in fever cases, and may produce urgent signs of cumulative action if the fever should fall. The author has seen one case in which, with rather sudden defervescence, the pulse dropped from 140 to 40. Camphor, gr. ij., disagrees when given by the mouth and should be given under the skin in 1 to 10 parts of sterilized olive oil. Other cardiants are considered under valvular disease and pneumonia. Sudden dilatation should be treated by venesection and stimulation. *Alcohol* may be considered under this head. In ty-

phoid it may be indicated (*a*) as a food, with milk and eggs, thus aiding digestion: ℥j. to iv. of whiskey may be given every two to four hours in hot milk. Stokes' mixture contained cognac, ℥ij.; vitelli ovi., one; syr. aurantii, ℥v.; water q. s. ad ℥v. Eggnog is valuable as a food which may be readily absorbed. (*b*) As a cardiant (though pharmacologists hold that pure alcohol is not a cardiant); Meltzer states that it equalizes the circulation. It is indicated especially in young alcoholics, and in most cases over forty years of age. Cognac may be given by the mouth, in coffee; or by rectum, in peptonized milk with two drachms of tincture of valerian. (*c*) As a nervine in debility or old age, as whiskey, champagne (especially for vomiting), sherry, or port. (*d*) To a less degree as an antipyretic, since it increases the sweat production. Its use must be suspended when the pulse becomes bounding, the respiration hurried, the skin or tongue dry, and when there is restlessness, or the odor of alcohol appears on the breath. Like other good things alcohol has been abused, and of late the reaction against its use is in part sound and in part excessive. With care and discretion alcohol is invaluable in severe cases.

Thrombophlebitis is treated by quiet, elevation, warmth, and bandaging from the toes upward.

3. *Respiratory System.*—Epistaxis usually ceases spontaneously. If the blood runs into the throat a tampon should be introduced. Laryngeal complications may necessitate early tracheotomy. Only 2 per cent. of severe laryngeal complications recover without operation. Bronchitis is treated most efficaciously by hydrotherapy, or rarely, by codeine. Expectorants are always avoided since they produce gastric disorder. Hypostasis is best treated by hydrotherapy, cardiants, and change of posture every two hours, for which explicit written orders should be given.

4. *Nervous System.*—The initial headache may be relieved by hydrotherapy, the ice-cap, ergotin and acetphenetidinum (phenacetin) each gr. v., for several doses. Depression is relieved by valerian and whiskey. For delirium, and insomnia, hydrotherapy is the most useful measure; veronal, gr. v.–vij. in warm water, produces a very natural sleep; alcohol; morphia, gr. $\frac{1}{4}$; codeine, gr. $\frac{1}{2}$; sodium bromide, ℥ss to j.; and hyoscinæ hydrobromidum, gr. $\frac{1}{120}$ to $\frac{1}{80}$, may be indicated.

℞ Morphinæ hydrochloridi ..... gr. ¼.
Sodii bromidi ..... gr. xxxxv.
Syrupi aurantii ..... ℥iij.
Aquæ chloroformi .....ad ℥ij.
M. f. mist.
S.—Half to be taken at bed-time, and the remainder in three hours, if necessary. (Whitla.)

Typhoid patients should never be left alone, lest they leave the bed or leap from the window. Restlessness may suggest a full bladder.

5. *The Skin.*—Massage with alcohol or olive oil is useful. The

skin functions are helped by the full bath. In the formation of bedsores, local heat, pressure, moisture, maceration, or filth are factors. These are generally avoided by frequent changes of posture, thick pads or rubber rings and cleanliness. In some cases great debility may frustrate prophylaxis. The least erythema, papule, abrasion, or pustule, on dependent parts is a matter of solicitude; without great care, skin infections may cause ward-epidemics (Edsall). If bed-sores have already developed, the simplest treatment is the best, since antiseptics and powders may aggravate. The balsam of Peru is excellent. Collodion and adhesive straps are always to be avoided as far as possible.

6. *Bacilluria.*—Bacilluria is met by phenylis salicylas and hexamethylenamina, each gr. x., t. i. d. Special care is necessary in the use of catheters.

(*H*). Treatment in Convalescence.—The convalescence must be actual, and the possibility of relapses must be excluded before the patient can have substantial diet or be allowed to sit up. It is best to err on the safe side, as embolism and collapse are not unknown in convalescence. Eggs, toast, gruels, rice, etc., may be given after one week of normal temperature. The patient should not leave his bed for two weeks. Irregular fever lingers in some cases until solid food is given and the patient sits up. In other cases, nitrogenous food will cause a higher temperature (*febris carnis*). Vegetables and fruits should be postponed for a few weeks. An excellent tonic in convalescence is the following:

℞ Strychninæ sulphatis ..... gr. j.
Acidi nitrohydrochlorici ..... ʒj.
Tr. gentianæ co ..... ʒv.
M. et S.—One teaspoonful after meals in half a glass of water.

TYPHUS FEVER.

Synonyms.—Typhus fever; exanthematous typhus; war, prison, ship or famine fever.

Definition.—An acute, highly contagious, specific infection of unknown causation, mostly epidemic; with a sudden onset and a high temperature, falling by crisis, and with a cyclic curve, a characteristic eruption, and severe nervous symptoms. Typhus, first described by Corradi (1083), was clearly distinguished by Fracostorius, in Verona (1505).

Etiology.—All stages of the disease are highly contagious. The unknown virus exists in the exhalations from the skin and lungs (Murchison), and survives for a long time in clothing and furniture. Infection is usually direct from person to person. The danger to attending nurses and physicians is therefore great, especially in severe epidemics. The more intimate the contact the greater is the danger of direct infection. It is also communicable indirectly by a third person, by means of fomites and fleas may convey infection.

The atrium is probably the lungs. Susceptibility to the disease is general. Immunity is usually conferred by one attack.

Geography.—Ireland, Russia, Galatia, Hungary, Italy, and Mexico, are its most frequent homes, and there are always cases in China, Asia Minor, and Hungary. It is endemic in Berlin, occurring among the lower classes, among vagabonds, and in the unclean, poorly-fed, closely-packed and unhygienic population. Typhus was one of the greatest scourges of the middle ages, with its wars and famines, but it has decreased in the past century, the epidemics in Ireland (1817 and 1846) and in the Turko-Russian war (1877–78) being the last general outbreaks. Its most recent occurrence in this country was in New York (1881–82 and 1893), in Philadelphia (1883), and in Baltimore in 1901 (Osler).

Age.—Most cases occur between the fifteenth and twenty-fifth years. It is rare in the young or aged. It may occur with other epidemics. The bacteriology of typhus is undetermined. A streptococcus was found by Hlava (1891) and a diplococcus by Dubieff and Bruhl (1893).

General Clinical Picture.—1. Incubation.—The incubation lasts twelve days or less in children, and is usually unaccompanied by symptoms.

2. The Stage of Invasion.—The invasion is *sudden,* exhibiting one or more chills and a high temperature on the first day. Thirst follows, with anorexia, coated tongue, vomiting, epigastric oppression, suffusion of the face, conjunctivæ, nose, and pharynx, apathy, delirium, epistaxis, backache, severe pains in the extremities, rapid pulse, and early splenic intumescence.

3. The Exanthematous Stage.—On the third or fifth day a roseolous eruption develops, at first pale-red and purely hyperæmic. It is seen first on the abdomen, and chest, extending rapidly to the back and extremities. The eruption then develops a petechial character, with some serous infiltration in the centre or edge of the flecks, which become dirty and copper-colored. It lasts seven to ten days. In this stage there is no pain, but often apathy, prostration, delirium or mania. The tongue and mouth are dry and dark. Catarrhal inflammations of the pharynx, larynx and bronchi develop, with hoarseness and cough. The fever is continuous. Coma-vigil, rapid respiration and pulse, and hypostatic pneumonia may end the course.

4. Defervescence.—A crisis usually occurs on the tenth to the fourteenth day. The pulse becomes slower; the skin often desquamates in dusky scales; profuse sweats are common; the tongue clears, and convalescence is established in two weeks. Long defervescence suggests complications.

Special Symptomatology.—1. Fever.—After a rigor, the temperature rapidly rises to 103°, 105°, or even 107°. No other acute exanthematous disease produces an equally high early temperature. The maximum temperature is observed on the fifth day. On the

tenth day the temperature often remits, except in most severe cases. The crisis occurs from the tenth to the fourteenth day. Curschmann considers that lysis is as frequent as crisis. Death occurs with a preagonal rise or with collapse-temperature.

2. The Skin.—The eruption consists of two elements: Roseolæ changing to petechiæ, and a dusky-red mottling under the skin. The *roseolæ* are seen on the third or the fifth day; they increase rapidly though never in recurrent crops. They are located chiefly on the lower abdomen, chest, back or shoulders, are least abundant on the thighs, and do not appear on the face except in women and children whose skin is delicate. The palms of the hands and the soles of the feet are generally exempt. The roseolæ are pale-red, washed in outline, round or oval, the size of a pin-head or a lentil. They are hyperæmic, and therefore disappear on pressure and after death. They are easily overlooked at night. They last one, two, or at the most three days, later becoming livid, copper-colored *petechiæ* persisting on pressure and after death, became hemorrhagic. The petechiæ occur mostly on the back, or in the folds of the groin, and may lie between the roseolæ. The hemorrhage may extend more widely into the skin or subcutaneous tissue. Typhus cases presenting no rash are rare. A great multiplicity of petechiæ is an ominous feature, especially in cachectic and alcoholic subjects. A morbilliform eruption on the arms and legs is occasionally observed before the usual typhus rash, especially in thin-skinned individuals. The skin is usually dry, but miliaria may appear (in 10 per cent. of cases), notably in the young. Icterus without decolorization of the stools is frequent and is an unfavorable sign. Desquamation occurs and is sometimes as profuse as in scarlatina; herpes occurs in 5 to 20 per cent., and bed-sores in 3 per cent. of the cases. The skin has a peculiarly pungent offensive odor.

3. The Nervous System.—Toxæmia is profound. Headache is constant until the exanthem appears; it is usually frontal and temporal and radiates into the neck or shoulders. It is associated with vertigo, backache, and hyperæsthesia of the fingers and toes. The sensorium first shows depression, characterized by apathy, insomnia, incoherent speech, or disturbing dreams, which increase as the pains disappear. In the second week coma-vigil, low delirium, mania and hallucinations often develop. Suicidal tendencies may be noted. Involuntary evacuations and noisy cerebral respiration also occur. The earlier the nervous symptoms, the more ominous is their significance. Neuralgia is occasional in defervescence. It is transient, and occurs mostly in the lower extremities. Tremor of the forearms and hands is almost constant. Convulsions are rare, but ominous. They are observed chiefly in alcoholic or very irritable subjects and in children. They are most often epileptiform. Paralysis occurs from brain hemorrhage, embolism, etc. Cases of paraplegia and aphasia are recorded. Meningitis is most rare.

Special Senses.—There is conjunctival catarrh and the " pin-head

pupil" of Graves. The ears are involved in 32 per cent. of cases.

4. Circulation.—The heart and vasomotor system suffer greatly (Stokes). The pulse, which is of greatest prognostic value, is small and reaches 100 to 120, or more in women and children. It is slow only in light cases. It is labile and often slow in convalescence. Acute myocarditis may occur with the highly dangerous acute dilatation. Its symptoms are those described under Typhoid. In fatal cases the heart muscle is granular and deep-red. The blood is dark and fluid; the red cells and hæmoglobin are decreased, and the white cells are increased.

5. Respiration.—Epistaxis is occasional. Hoarseness, erosions, ulcerations, œdema, and perichondritis, may develop in the larynx and may lead to secondary abscess of the lung or empyema. Bronchitis, and tracheitis are part of the disease and are present at the onset, increase with the fever, and are evidenced by cough, and slight expectoration. Catarrh with weak heart results in atelectasis, hypostasis, and lobular pneumonia.

Pneumonia occurs in 15 per cent. of typhus cases, usually in the second week, and is frequently atypical.

6. Gastro-Intestinal Tract.—The tongue, at first moist and white, becomes dry and dark—the "parrot tongue." Thirst is common. Nausea and vomiting are fairly common, but meteorism is infrequent. The epigastrium only is tender. The liver is swollen, soft, and grayish. There is usually constipation (90 per cent. of the cases), although diarrhœa may appear at the crisis. Gastro-intestinal hemorrhage, a hemorrhagic diathesis, diphtheritic ulcerations, parotitis, ulceration of the œsophagus and pharynx, phlegmons, and peritonitis, are infrequent.

7. Genito-Urinary Tract.—The urine is febrile. The urea is decreased. Albumin frequently appears in the second week. Nephritis (in 6 per cent.) is dangerous. Uræmia and hæmaturia are rare. The urine will frequently show the diazo reaction.

8. Spleen and Glands.—An early splenic tumor is palpable in 33 to 66 per cent. and disappears in the second week. The splenic pulp is fluid and degenerated. Rupture has been described. The lymphatic glands are not usually enlarged, though adenitis was noted during the Crimean and Turko-Russian epidemics.

Recurrence.—This happened only once in 5,000 cases (Buchanan).

Anomalous Courses.—Light forms run a course of six to ten days, with less fever, fewer nervous symptoms, and less rapid pulse than in the more severe types. They include the abortive type without eruption, and the ambulatory type. Very severe forms are the hyperpyretic (fatal in two to three days), or the adynamic (in which the patient dies with low temperature, hemorrhages, etc.).

Differentiation.—This is not difficult if the case is seen from beginning to end, or during an epidemic. There are no absolute pathological or bacteriological criteria. Mistakes in the diagnosis of sporadic cases are often inevitable. In the initial stage only a probable diagnosis can be made.

In *smallpox* the chills, fever, prostration, severe aches, splenic tumor, and backache, are not final, since they are less marked in mild forms of variola. The initial scarlatiniform eruption in the angles of the arms and groins is characteristic of smallpox. Morbilliform eruptions occur in both. Smallpox invades the face more than does typhus, and the relation of the eruption to the temperature remission is not observed in typhus. Even at postmortem examinations, differentiation between the hemorrhagic form of typhus and *purpura variolosa* is frequently impossible.

Typhus.	Typhoid.
Invasion; sudden; chill, coryza.	Slower and more gradual; no coryza.
Roseola; more numerous and earlier, second day to fifth day.	Usually less points of eruption (are exceptions,—*e. g.*, measles-like); on extremities, as in Franco-German war.
Never in crops.	In successive crops.
Distribution on body and extremities.	On body; rarely on extremities.
Sparingly on face.	Almost never on face.
Flat, pale, washed, hyperæmic, not disappearing upon pressure when they become petechial.	Papular, pink, sharply outlined, always hyperæmic, disappearing upon pressure.
Course; more rapid.	Slower.
Face and eyes injected.	Paler; conjunctivitis rare.
Fever; abrupt rise, higher, less remission, if any; shorter; crisis, or short lysis, on fourteenth day.	Slow rise and regular stages.
Soon bedridden.	Oftener ambulatory.
Earlier and severer nervous symptoms.	Second week or later, and less severe.
Pulse; rapid and not dicrotic.	Dicrotism of less importance than slowness.
Spleen; early swelling, but less frequent.	Later, but lasts longer; two or three times more frequent.
Less meteorism or none.	Meteorism.
Stools; not characteristic.	Ochre-stools.
Blood; leukocytosis.	Leukopenia, Widal reaction; bacteria in blood, urine, roseolæ.
Diazo reaction in both.	

In *recurrent fever,* the fever-curve is important, but the diagnosis may be difficult during the first attack. The history, the lack of involvement of the sensorium, the generally favorable condition of the patient, the absence of pain, the characteristic anæmic yellow-gray skin, the spirilla and successive attacks, indicate recurrent fever.

In irregular epidemics, meningitis, with chills, fever, nervous

symptoms, coma, and headache (without rigid neck and with a roseolous eruption), at first presents difficulties, though in the later course, it may be distinguished after lumbar puncture and examination of the retinæ. The eruption is at first roseolous but later is petechial. If the petechiæ occur first, the disease is not typhus.

In *measles* the face is the first seat of eruption; the rash is rarely hemorrhagic; catarrhal symptoms precede the eruption, when the fever again rises. Typhus is rare under five years of age.

Prognosis.—The prognosis depends upon the intensity of the toxæmia, the fever, the nervous symptoms, and on the complications—especially pneumonia. Death from toxæmia usually occurs in the second week (twelfth day); from pneumonia in the third week. The mortality varies between 6 and 20 per cent., but reaches 50 per cent. in virulent epidemics and in patients over fifty years of age. Children rarely die of typhus.

Treatment.—The treatment is that of typhoid, with the added indication of strict isolation. Open-air treatment in tents is indicated, with thorough disinfection of everything coming in contact with the patient (see SMALL POX). Cardiac stimulants are often required.

RECURRENT FEVER.

Synonyms.—Relapsing fever; febris recurrens; typhus recurrens; seven-day fever; famine or prison fever; bilious typhoid.

Definition.—A specific infectious disease caused by Obermeier's spirillum; acquired more often by direct personal contact than by indirect infection; endemic in Ireland, Russian Poland, and India, though sometimes epidemic; clinically characterized by a cyclic course of febrile attacks lasting six or seven days, followed by an equally long apyretic interval, which cycle may be repeated several times.

Etiology.—Recurrent fever was first described by Rutty, of Dublin, in 1739. It appeared in America in 1844, and was last seen in 1869. Obermeier in 1873 described a specific spirillum or spirochæta (*Spirillum Obermeieri*). Recurrent fever is transmitted not by the spirillum itself, but by its spores, since the disease may recur after the spirillum has been killed by quinine. The spores are possibly the glistening bodies sometimes seen in the blood (*q. v.*). The breath and exhalations are infectious. The secretions are apparently not infectious. The disease has been inoculated in monkeys and in man, from postmortem cuts. Bed-bugs may also convey it. Infection is carried chiefly by vagabonds and emigrants. Males are especially affected (90 per cent.). Most cases occur between the twentieth and the fortieth years.

Symptoms.—INCUBATION.—After an incubation of from five to seven days, the invasion occurs, usually, without prodromes.

THE INVASION.—The invasion begins with a chill, a high temperature (reaching possibly 104° on the first day), a throbbing headache,

and, in the young, convulsions and vomiting. The vertigo and cerebral confusion may resemble acute alcoholism. Because of the severe neuralgic pain in the back, legs, and calves, the patient lies motionless. There is great depression, conjunctival suffusion, slightly icteric scleræ, a pale (even cachectic) facial color, epigastric oppression, with or without vomiting, and tenderness over the liver and spleen.

Specific Findings.—The specific findings are in (1) the blood; (2) the spleen; and (3) the bone-marrow.

1. *The blood* is dark and reveals the pathognomonic, motile, corkscrew-shaped spirillum, which usually appears with the fever. It usually disappears before the fever falls, and appears again with the next attack. The spirillum is found in the blood only. The number is variable and not proportionate to the severity of the disease; as, for instance one spirillum in ten to seventy fields at first, and twelve to thirty spirilla in a field at the acme. Without the immersion-lens, they may be located by the lashing about of the red cells. They always lie between and never in them, and appear as fine threads, sixteen to forty microns in length, with five to ten waves in each, and move by undulation along their long axes. They stain best with fuchsin, after the extraction of the hæmoglobin by acetic acid. Their life in the blood is usually thirty-seven days. The white blood-cells are increased. Large, fatty, granular protoplasma-cells, probably from the spleen, may sometimes be seen.

2. *The spleen* is nearly always enlarged, painful, and palpable, and varies in size directly with the temperature. According to Leube, in no other infectious disease is the spleen so large, being increased five- to sixfold and weighing even twenty pounds. The capsule is tense, sometimes with perisplenic adhesions, and the pulp is a fluid-red. The Malpighian bodies are increased, and necrotic areas and infarcts are common. Suppuration and rupture may occur. Microscopically there are hyperæmia, hyperplasia, spirilla, and fatty desquamated endothelium from the veins, thus explaining the spindle-cells found in the blood-stream.

3. The *bone-marrow* resembles the splenic follicles and contains many granular cells, and, occasionally, aggregations of white blood-cells.

Other Symptoms.—1. Nervous System.—Headache is frequent. The sensorium is usually free, delirium and insomnia being rare.

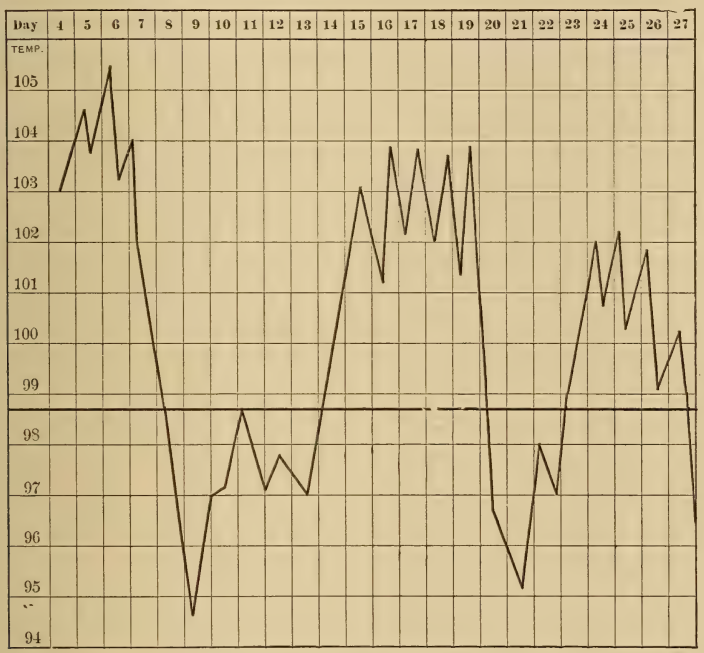
2. Temperature.—The temperature rises abruptly to 103° or 104°, and remains continuously high (105.8° to 107.6°), from five to seven days, when the crisis occurs. In no other disease is the defervescence so precipitate (Leube). The fever falls 5° to 13° (even 16°). In from five to seven days the patient is well, when in 99 per cent. of cases the cycle is repeated. In the second attack there may be no chill. There are usually but two or three relapses, though as many as seven have been recorded, each successive one being shorter by one to one and one-half-days. The temperature-

curve is diagnostic. Lysis is rare (1 to 2 per cent.). The crisis is followed by rapid convalescence, sweats, shrinkage of the spleen, and disappearance of the spirillum; sometimes post-critical diarrhœa and collapse develop.

3. Circulatory System.—The pulse is rapid (120 to 140). Compared with the temperature, it is full and hard, seldom dicrotic, sometimes irregular, and often slow after the crisis.

4. Respiratory Tract.—Bronchitis (30 per cent.) and increased respirations (thirty or more) have been observed. In fatal cases atelectasis, hypostasis, pneumonia (40 per cent.), glottis œdema (31 per cent.), and pleurisy (2 per cent.), have been found.

Fig. 7.



Fever chart in recurrent fever.

5. Gastro-Intestinal Tract.—The tongue is white and later, yellow-brown. Hæmatemesis, a tender and swollen liver, icterus (8 to 25 per cent.), gall-bladder distention, bloody effusions in the alimentary mucosa, and hyperplasia of the lymphatics, may be observed.

6. Genito-Urinary Tract.—The urinary findings resemble those of typhoid. Nephritis is infrequent (2 per cent.), and abortion occurs in 66 per cent.

7. Skin.—The skin is usually subicteric, yet the stools remain colored. Herpes, erythema, petechiæ, desquamation, and even roseolæ, have been noted. The skin becomes drenched at the crisis.

Diagnosis.—The specific points are: A prevailing epidemic; onset with chill and fever; swelling and tenderness of the liver and spleen;

muscular pains; abrupt crisis; apyretic interval with recurrence of the cycle, and spirilla found by blood examination.

Löventhal has found that the serum acts as a bactericide on the spirochætæ. In the bilious type (bilious typhoid or septic recurrent fever), there is severe icterus, destruction of the red corpuscles, and pathological liver-findings which are suggestive of acute yellow atrophy. Hemorrhages, involvement of the sensorium, and death from collapse in the first attack are common. The colored stools, rapid pulse, severe bronchitis, and enlarged spleen, are significant. Malaria, typhoid, typhus, yellow fever, acute yellow atrophy of the liver, and Weil's disease, are excluded by one examination of the blood.

Convalescence.—Convalescence is rapid, after four or five weeks. Death may occur from toxæmia, heart collapse, or from complications. The prognosis is good, the mortality averaging 2 to 10 per cent., although reaching 60 per cent. in the bilious type. One attack is not certain to confer immunity.

Treatment.—The treatment is that of typhoid, and no drug has any effect on the parasites. Pain often necessitates the use of opiates. The heart weakness is treated as it is in typhoid.

SMALLPOX, VARIOLA. VACCINATION.

Definition.—Smallpox is an acute, highly infectious, specific disease. It begins suddenly with a chill, headache, vomiting, and intense epigastric and lumbar pain, and is characterized by a typical fever-curve and by an eruption on the skin and contiguous mucous surfaces consisting of papules, vesicles, pustules, and crusts in successive stages.

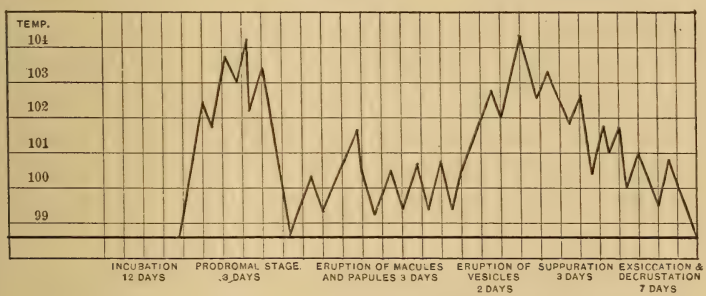
History.—Smallpox is said to have prevailed in China many centuries and in India 1,000 years before the Christian era. Galen's *pesta magna* of the second century is thought to have been variola. Epidemics occurred in the sixth century, and during the crusades. Rhazes (Arabia, ninth century) gave us the first accurate clinical description of the disease. Smallpox was introduced into Mexico in 1520, by the Spaniards, and three and a half million persons contracted the disease. Sydenham's classic of the seventeenth century is most accurate and reliable. In 1718, Lady Montague introduced into England preventative inoculation, which had been practiced for centuries in Asia, and though she reduced the mortality by this means to 1 per cent. or less, she only disseminated the disease in a mitigated form. Jenner's discovery of vaccination, in 1796, has greatly lessened the terrors of variola, which in Europe alone carried off nearly half a million persons yearly, and in Iceland cost the lives of 36 per cent. of the population.

Etiology.—The virus is still unknown. Weigert and others have found pyogenic organisms, although suppuration is due to a later mixed infection. Protozoa were first mentioned by Van der Loeff, in 1886.

The *cytorrhyctes variolæ* of Guarnieri (1892) is an amœba found in the blood in the incipient stage, in that of vaccinated children and of calves, and in the exanthem. Pfeiffer, Roger, Weil, Funck, Copeman, Councilman, Mallory, Calkins, Howard, and Perkins, have found similar structures. Small bodies are found in the lower epithelial layers. These bodies are structureless, measure one to four microns, and lie in vacuoles in the cells. They enlarge and become granular and segmented.

That the blood is infectious at an early stage was demonstrated by the development of the disease in an individual on whom skin had been transplanted from a person in the early stages of smallpox. Variola without eruption is contagious, and it therefore seems that the secretions, excretions, and exhalations, convey the virus. The virus causes constitutional infection before the eruption appears. The greatest infectiousness is noted during the eruption. Its chief sources are the dried pustules in which the virus localizes. Infection may be direct from person to person, or indirect by a third party or by fomites. Cadavers of variola victims are dangerous. In short, every new case comes directly or indirectly from a patient with variola. The virus is very penetrative, and infection may exist beyond the room, the building, or the area of the isolated case. The disease persists in infected communities for years, becoming quiescent and afterwards breaking out anew. The chief factor in dissemination is human intercourse.

FIG. 8.



Temperature chart and stages of smallpox. (Pfeiffer.)

Susceptibility to smallpox is almost universal; only 1 per cent. of people are immune. Individual immunity was noted by Morgagni, Boerhaave, and Diemerbroeck. The author knows two persons in whom vaccination has never been successful. Temporary immunity is more frequent than congenital. Ordinarily and practically, immunity is only acquired by vaccination or a previous attack. In prehistoric times, inoculation was practiced to afford immunity. Second attacks are infrequent. Either sex and all ages may be affected. Mothers with the disease may deliver babes with the florid eruption or with scars. The virulence of the virus is great in aboriginal and colored races (Indians and Mexicans), and in preg-

nant or menstruating women. Severe cases may follow mild infection, as shown in Osler's description of the Montreal epidemic, in which 3,164 persons, infected by a mild case in a pullman-car conductor, died among the French Canadians, who oppose vaccination. It is frequently conveyed by vagrants.

Point of Entrance.—Smallpox infection unquestionably occurs through the respiratory tract, and perhaps also through the digestive tract. Variola occurs sporadically, epidemically, or pandemically.

Symptoms.—Forms:

I. Variola Vera	—1. Discreta, discrete.
	—2. Confluens, confluent.
II. Variola	—3. Purpura variolosa (black smallpox).
Hemorrhagica	—4. Variola hemorrhagica pustulosa.
III. Varioloid	—5. Smallpox modified by vaccination.

1. Variola Vera Discreta.—(*A*) *The Incubation.*—The incubation averages twelve days (five to twenty). It usually presents no symptoms, though there may be headache, anorexia, and pharyngitis.

(*B*) *The Prodromal Stage.*—The prodromal stage (initial stage, or stage of invasion), dating from the first symptom to the cutaneous eruption, is the same in all types, and its duration averages three days. It is shorter in children and in the confluent type of the disease; the longer it is, the more severe is the infection (Trousseau). Its intensity bears no invariable relation to prognosis, for varioloid (mild smallpox), may present a severe onset; however, if the onset is mild, the disease will not be confluent or hemorrhagic. Prodromal symptoms are absent in rare instances.

The *invasion begins acutely* with chill, fever, rapid pulse and respiration, nervous toxæmic symptoms, intense headache, backache and vomiting. (*a*) The *chill* is usually severe, and frequently is repeated. It may follow one or two days of fever (Jaccoud). In children it is often absent. (*b*) The *initial fever* is almost invariably high, rising suddenly to 103° or 104°, and reaching its maximum on the second or third day. In rare instances there is no fever. The *pulse* is rapid (120), full, and not often dicrotic except in severe cases. The more frequent respiration (30 to 36) is somewhat characteristic, constituting the so-called toxæmic or "cerebral respiration." The *skin* is red, excessively hot (*calor mordax*), and usually dry, but there may be sweating in the discrete variety (Sydenham) and in favorable cases (Trousseau). The face is flushed but the eyes are clear. (*c*) *Nervous toxæmia* is evidenced by the benumbed sensorium, depression, restlessness, insomnia, delirium (especially in alcoholics), or convulsions and meningeal symptoms in children. *Headache* is almost constant, usually appearing with or just before the chill. It is frequently frontal or temporal, but may be generalized, occipital, or neuralgic. When severe or agonizing in character, it may be suggestive of meningitis, especially when accompanied by delirium, hard vomiting, and neckache. (*d*) *Backache* appears with the chill, and lasts one or two days. It occurs in nearly all cases though

rather less frequent than headache and vomiting. It is severe like lumbago or actually agonizing. The "veritable paraplegia" described by Trousseau is usually only immobility due to pain caused by toxins or by intense congestion of the kidneys, spinal muscles, or meninges (Jaccoud). It is rare in other fevers likely to be confused with variola. Pain is sometimes observed in the pharynx, larynx, or heart, and produces a sensation of constriction or actual dyspnœa; it may occur in the intercostal nerves, sciatics, muscles, and joints. (*e*) *Vomiting* and early epigastric pain are especially constant in children. The spleen is not often enlarged in the initial period. It is often tender, as are the liver and epigastrium. (*f*) The *initial* or *prodromal eruptions,* studied especially by Sydenham, Simon, Osler, Knecht, and Scheby-Busch, are present in 10 to 16 per cent. and have considerable diagnostic value. They are more commonly limited to the lower abdomen, inside of the thighs, sides of the thorax, axillæ, and sometimes to the extensor surfaces of the knees and elbows (Simon). There are two varieties:

1. The *morbilliform variety* (measles-like, roseola variolosa), which appears during the second day on the face and body as small maculæ. These disappear upon pressure, are sometimes crescentic, and usually last one day. They are due to hyperæmia but may be purpuric. They are seen most often in varioloid, and therefore have a favorable prognostic import.

2. The *scarlatinal form* (erythema variolosum) is more rare and occurs earlier, either on the first day, or before any other symptoms. It is less punctate, extensive, and bright, than it is in scarlet fever. If it is purple in color and purpuric, it is almost always variolous. It occurs on the lower abdomen, and on the thighs (forming a triangle with base upwards when the legs are together), but may even reach the knees. Sometimes it is found in the axilla or over the shoulders, and is seen in women more frequently than in men. These areas are rarely invaded by the pustular eruption. Some writers speak of an erysipelas-like prodromal rash, and others have observed prodromal urticaria.

(*C*). *The Eruptive Stage.*—The eruptive stage consists of several serial sub-stages:

1. The stage of *macules and papules* (*stadium maculosum et papulosum*) occurs on the fourth day, and advances for three days. The eruption begins on the forehead near the hair and on the cheek, with itching and burning macules, resembling flea-bites, which, at first purely hyperæmic, soon become papules. The papules are reddish, discrete, circular, elevated, and hard or "shotty." A day after their eruption on the face and head, they appear on the body, and on the next day, on the extensor aspects of the extremities; *i. e.,* the papular eruption advances for three days, and is descending. The papules are thickest on the face and trunk. Sometimes they are very thick on the neck. The eye-lids show collateral œdema. According to Stokes, the amount of the eruption depends on the vascularity of the

part. Trousseau and Sydenham maintained that if the eruption appeared on the second day of the disease, the confluent type might be expected; if on the third day of the disease, the confluent type might be anticipated. (If the eruption ceases at this point, the disease is called variola cornea, or verrucosa.)

Weigert holds that the first skin changes are not inflammatory but necrobiotic or diphtheroid, and that inflammation begins later with exudation into and about the necrotic areas in the rete Malpighii. Other authorities consider inflammation the primary, and necrosis the secondary process.

Unlike measles and scarlatina, *the fever remits when the eruption appears,* and the backache and headache improve. In two or three days the fever reaches normal by irregular steps. The lower the fever falls in this remission, the lighter is the type of smallpox.

2. In the stage of *vesiculation* (*stadium vesiculosum*), lymph gathers in many but not in all of the papules. This occurs on the seventh day of the disease and lasts about two days. The vesicles are multilocular, since they occur in the cells of the rete Malpighii. Umbilication occurs in the centres of many vesicles, caused by elevation of their edges by infiltration and by the follicles in their centres. It corresponds to the area of primary necrosis and is suggestive but not pathognomonic of smallpox (Jaccoud, Eichhorst). On the soles of the feet and the palms of the hands the fluid is situated deep in the resistant tissues. (If the changes stop short of pustulation, the disease is called variola crystallina.) The mucous membranes may be the seat of simple inflammation, specific eruption, or pseudomembrane.

3. The stage of *suppuration* (*stadium pustulosum*) or maturation begins on the ninth day with clouding of the vesicles and with inflammatory congestion (areola) about them. It advances for usually three and rarely more than five days. Increased mucous membrane catarrh develops simultaneously with the cutaneous eruption. The pustules become opaque, then yellow, and the thick pus obliterates the umbilication and renders the eruption globular. The areola, or halo, becomes more vivid and inflammatory œdema may result from fusion of the areolæ. This causes increased tension, deformity (especially in the face and parts where the skin is most loose), closure of the eyes, occlusion of the nose (of particular moment in children), and great tenderness and pain. Pustulation follows the descending order of the initial eruption and the pustules are less thick on the trunk than on the extremities and head. On the volar and plantar surfaces they are less prominent and there is less œdema. The pustules evacuate spontaneously, especially on the face, but may dry without rupture. Various stages of the eruption may be observed in different parts of the body at the same time; not all vesicles become pustules. The skin exhales a peculiar, penetrating, offensive odor, and bed-sores may develop.

(Anomalies are variola *abortiva,* in which the pustular stage is not

reached; variola *siliquosa*,—air in the vesicles or pustules; and very large eruptions three quarters of an inch in diameter.)

Pathologically, the pus originates from the vessels of the papillæ which may undergo much pressure-atrophy, or even necrosis.

The *mucous membranes* are involved in various ways, but the pock eruption occurs mostly in the upper mucosæ (mouth and naso-pharynx), and therein resembles its cutaneous distribution; the successive stages of papulation, vesiculation, pustulation, and crustation, may develop, but they are less typical than in the cutaneous eruption; erosions, diffuse catarrhal stomatitis, glossitis, phlegmonous angina, etc., may result. The eruption has been found in very rare instances in the external ear, Eustachian tube, eye, œsophagus, air passages, alimentary and genito-urinary tracts.

With pustulation there is in most cases a gradual rise in temperature, *secondary fever*, the fever of suppuration. It is due to the smallpox virus and mixed pyogenic infection. The duration of the rise is proportionate to the degree of pustulation and of dermatitis. In variola discreta the temperature does not remain high more than twenty-four to thirty-six hours, with morning remissions. A marked *leukocytosis* attends the secondary fever. It parallels the degree of infection and may even attain 30,000 to 34,000 cells per cubic millimeter. It ceases with involution unless complications intervene. The lymphocytes are increased (to 50 or 66 per cent.), the polymorphonuclears decreased (to 50 or 33 per cent.), and the myelocytes may reach 10 to 12 per cent.

The red cells occur in clumps rather than in rouleaux, and some deglobulization, even as low as 2,000,000, is observed. The fever may show a pre-agonal increase or collapse. Hyperpyrexia may occur. Repeated rises in temperature are very dangerous. Cerebral symptoms occur, such as great excitement, or delirium with suicidal tendencies. Albuminuria (33 per cent.), acute exhaustion, and heart paralysis, may develop.

(*D*). *The Stage of Involution.*—The stage of involution (*exsiccation, decrustation*), begins on the twelfth day. It occurs in the same descending order as the florition and suppuration, first on the head, then on the body, etc., with decrease in the œdema, in the redness, and in the pain. As the pain decreases, there is an intolerable itching and some pigmentation. Crusts form, and remain longest on the soles of the feet and palms of the hands where they may form complete casts of the part. Mucous erosions heal more readily than crusts. The hair falls out, and by the end of the second week the fever reaches normal. There is no desiccation fever, as claimed by some writers, and fever at this stage indicates some complication. Scars result from the granulations, and the higher the granulating surface, the deeper is the cicatrix. Scars occur when the true skin has been involved, and are seen chiefly on the face, scalp, palms of the hands, and soles of the feet. Sydenham's statement, that discrete smallpox rarely leaves its mark, holds good in most cases, according to Osler. Others

hold that variola vera implies suppuration with fever and cicatrices as necessary stigmata. Scar formation lasts three or four weeks. Complete convalescence follows the disappearance of the last crust.

2. Variola Vera Confluens.—This is a malignant type, which was more frequently observed in prevaccination days, and is now seen largely in unvaccinated persons or in those vaccinated but once. The initial stage is violent. The headache and backache are agonizing. The status typhosus and gastric symptoms are marked, the fever remission is slight or absent, and there is little or no improvement in the symptoms if a fall in temperature occurs. The exanthem is precipitate both in its development (on the second or third day), and in its extension. The earlier the exanthem in variola, the greater is the probability of confluent smallpox. The patient usually dies if the eruption does not appear. The eruption becomes confluent especially on the face and head, and sometimes also upon the hands and feet; whereas on the body and limbs it is largely discrete. With fusion of the eruption, great inflammatory œdema appears, the symptoms increase, swelling and erosion of the mucous membranes, closure of the eyes, keratitis, and obstruction of the nose, develop. The general aspect is desperate. High fever, high pulse, rapid, noisy, even irregular or stertorous breathing, delirium, albuminuria, persistent nausea and vomiting, great thirst, salivation in adults and diarrhœa in children (Sydenham), husky voice, enlarged cervical glands and often parotitis, are present. This type is severe from the onset and the prognosis depends upon the number of pustules upon the face. Sydenham says that "if upon the face they are as thick as sand, it is no advantage to have them few and far between upon the body." Death occurs from acute toxæmia (*variola typhosa seu adynamica*), with hyperpyrexia and cardiac asthenia, or, if the patient lives longer, from septicopyæmia, phlegmon, erysipelas, gangrene, pneumonia, pleurisy, and nephritis. Recovery from variola confluens is infrequent; in convalescent cases permanent deformity, such as ectropion, is probable. These cases run a course of three or more weeks.

3. Purpura Variolosa.—This is the black smallpox—*smallpox with primary hemorrhage* in the initial stages. It is the worst type, and results almost uniformly in early death. It was frequent in the great pandemic of 1870–72. It is also important because of the difficulty in its diagnosis. It is seen most frequently in the unvaccinated, but may occur in the vaccinated, in women during the puerperium, in the young, and in robust males. The incubation is short (six to eight days), the invasion is stormy, with agonizing lumbar pain, great prostration, a rapid, small, soft, and sometimes dicrotic or irregular pulse, and disproportionately increased respiration. The initial epigastric pain and vomiting may last until death.

On the second day (or even on the first), there appears a dark scarlet or plum-colored eruption, with brick-red, purple, or inky ecchymoses, especially about the eyes. The aspect of the discolored and

swollen face, and ecchymotic, sunken and intelligent eyes, is desperate. Hæmaturia is the most common of internal or visceral hemorrhages. Other forms are: Hæmatemesis, enterorrhagia, epistaxis, metrorrhagia, and hemorrhage from the gums, ears, and bronchi, sometimes accompanied by gangrene of the pharynx. Tympanites and albuminuria may be noted. Miscarriage is frequent. The spleen is not enlarged. The disease does not usually reach the real eruption of variola, or, at the most, only the papules are observed, because, as Rhazes observed, the course is almost invariably fatal in four or five days. The mind often remains lucid to the end. In the majority of cases the fever is not high, but in the most fulminant type it reaches 105° or 106°, with delirium, collapse, and coma. Death results in a few hours, even before cutaneous hemorrhage is seen, although internal hemorrhages are found at autopsy. According to Unna, the "blood dissolution" of the older writers is a mycotic coagulation thrombosis.

4. Variola Pustulosa Hemorrhagica.—This is more common than Purpura Variolosa (primary hemorrhage). In this type of smallpox hemorrhage occurs after the eruption appears (*secondary hemorrhage*). This form occurs in weakly or alcoholic subjects. The initial stage is severe, and secondary hemorrhages occur into the vesicles or pustules, or into the lower parts of the body if the delirious patient has left his bed, or there may be "blood dissolution," epistaxis, hæmaturia, and metrorrhagia. Adynamic manifestations are usual, and the outcome is almost always fatal, though in one type, hemorrhage at the vesicular stage may be followed by rapid abortion of the rash and speedy recovery (Osler).

5. Varioloid.—Varioloid (*variola modificata s. mitigata, variolois*) is *variola mitigated by vaccination.* The more marked the initial eruption, which is often morbilliform, the less the number of pustules. Varioloid begins suddenly. The fever is atypical. It may reach 103° and usually falls with the specific eruption, reaching normal on the fourth day. Secondary suppuration fever is rare and euphoria is common. The eruption is superficial, and, according to French clinicians, there is no suppuration. The eruption varies in amount, being present on the trunk only, or being typical chiefly on the hands and feet. It may be absent entirely. It may not be umbilicated and may desiccate rapidly without rupture. The exanthem is usually small and scars are rare. The mucous surfaces are little involved, or are entirely unaffected. The entire course is shorter, more irregular, and rudimentary, and more benign than in variola vera.

Complications and Sequelæ.—The complications and sequelæ of variola are many, occurring chiefly in the more severe forms and during the stage of suppuration. They embrace severe early toxæmia and later, secondary infections.

1. The Nervous System.—Nervous complications are very frequent. *Delirium* is caused by brain hyperæmia, which results from swelling of the face and scalp, simple toxæmia, alcoholism, or hyper-

pyrexia. Convulsions are frequent in children. *Psychoses* are usually asthenic and are not frequent. Meningitis suppurativa, encephalitis, hemiplegia, embolism, cerebral softening, phlebitis, and abscess, are occasional complications. Paraplegia and monoplegia, both caused by myelitis, Landry's paralysis and neuritis in the trunk, extremities, or cranial nerves, are exceptional sequels.

2. The Vascular System.—Vascular changes are more rare. The heart muscle may degenerate, becoming soft and relaxed, and microscopically revealing segmentation, and cloudy and fatty conditions. Dilatation may occur. French writers speak of a coronary endarteritis and myocarditis. A systolic bruit may be heard at the apex, with weak first tone, and tachycardia, as is the case in typhoid. Aortitis, pericarditis, ulcerative endocarditis and marantic thrombosis are infrequent.

3. The Respiratory Tract.—Œdema of the larynx and perichondritis are usually fatal, for laryngeal anæsthesia promotes inhalation pneumonia. Obstruction of the larynx by submucous hemorrhage is uncommon. *Bronchitis* is invariable in severe cases. *Pneumonia,* usually lobular, occurs in 60 per cent. of fatal cases; lobar pneumonia is rare. Lung abscess, embolism, and gangrene, are uncommon. Pleurisy is present in some epidemics, occurs chiefly in the stage of suppuration, and is especially and rapidly fatal in children (E. Smith).

4. The Digestive System.—Glossitis is rare, retropharyngeal abscess, parotitis, noma, phlegmonous œsophagitis, and hemorrhage, or membrane formation in the throat, are ominous though uncommon complications. The early vomiting rarely persists. Diarrhœa is not common, but a simple form is seen in children (Trousseau, Sydenham), and a dysenteric form is occasionally seen in adults. Peritonitis, retroperitoneal cellulitis, mediastinitis and perineal abscess are infrequent. The alimentary tract, with the exception of the rectum, is usually considered exempt from the eruption. The liver may suffer cloudy or fatty change, focal necrosis, or diffuse hepatitis. The spleen is usually swollen and soft.

5. The Genito-urinary Tract.—This is not often involved. Albuminuria occurs early in 25 per cent. of cases, during the eruption, during suppuration, or in convalescence. Nephritis is not frequent, but may develop during the fever or in convalescence; it is often glomerular. Focal necrosis may be noted. Pyelitis from cystitis or myelitis, ovaritis, and vulvar gangrene, have been observed. Menorrhagia is common in the early stages of onset or in hemorrhagic forms, and abortion from hemorrhagic endometritis is the rule in pregnant women. Orchitis variolosa is very frequent, the inflammatory foci being multiple (Trousseau, Chiari, Velpeau). They lie mostly in the connective tissue, but are rarely suppurative, or associated with focal necrosis.

6. The Special Senses.—The eye is involved in 1 per cent. (Hebra). Pox in the conjunctivæ are rare, as are conjunctivitis,

keratitis, diphtheria of the conjunctiva, iridochoroiditis, neuroretinitis, metastatic panophthalmitis, and retinal hemorrhage. Otitis, mastoid caries, and suppurative thrombophlebitis, may occur.

7. The Locomotor System.—Muscular abscess or hemorrhage and arthritis, especially in the knee and shoulder, may occur during desiccation. Arthritis is more often suppurative than serous, and is a feature of the secondary sepsis. In the bones there are almost invariably hemorrhage into the marrow, hyperplasia of the marrow cells, and multiple foci of necrosis or nonsuppurative osteomyelitis (in 72 per cent., Chiari).

8. The Skin.—Decubitus, erysipelas, abscesses (local or pyæmic), post-variolous eruptions, erythema, pustules, bullæ, great desquamation, or acute gangrene may develop in severe cases. Hemorrhages into existing exudates, and less often into the viscera, cellular tissues or brain also occur.

Diagnosis.—The diagnosis of smallpox is usually easy when chill, fever, headache, backache, and vomiting, occur in the presence of an epidemic or after exposure to infection. Vaccination and revaccination are factors and their scars must be searched for. It is often impossible to avoid mistakes in the diagnosis of the first cases of an epidemic. In abortive or purpuric cases a diagnosis is also difficult or impossible. Hemorrhagic scarlatina or measles sometimes cause difficulty. In the former the mucous membrane hemorrhages are less frequent than in smallpox. Differentiation between smallpox and virulent hemorrhagic or fulminating purpura may be difficult. The initial suggestive scarlatiniform eruption is important but has been unduly emphasized; the prodromal eruptions *plus* purpura are highly suggestive. Exposure to infection, an acute onset and high temperature are significant, although acute onset and high fever may occur in typhus (chill, macular and petechial rash, especially on the body, and persistently high fever), in pneumonia (chill, vomiting, herpes, pain in the chest, and rusty sputum), in scarlatina and influenza, with an initial rash, and in meningitis (joint involvement, headache, or vomiting). Lumbar and sacral pain is more common in smallpox than in typhus, pneumonia, or scarlatina. Intense frontal headache, severe backache, vomiting, chill, and fever, are sufficient grounds for a tentative diagnosis, and isolation. Nevertheless, the diagnosis is uncertain until the eruption is seen. In the *variola sine exanthemate* (the *febris variolosa* of Rhazes and Sydenham, the *variola sine variolois* of de Haen, the *varioles frustes* of Costes) the diagnosis must be made from the history of exposure, the presence of an epidemic, fever, pain, delirium, possibly the initial rash, and the absence of secondary fever because there is no suppuration. An eruption which appears on the third day of the affection, accompanied by a fall of the fever and euphoria, is most important.

VARICELLA———*vs.*———VARIOLA.

Etiology different. Vaccination and smallpox do not protect.	Smallpox may closely resemble chickenpox (epidemic of Lee); very mild cases are especially misleading.
Age is not distinctively differential; varicella usually occurs before puberty; it may occur in adults.	Usually after puberty, with numerous exceptions.
An initial stage is practically absent.	It is severe, even in mild cases.
Temperature; no remission with outbreak of rash.	Typical remission with secondary fever (except in varioloid).
Prodromal rash; very exceptional.	Frequent (10 to 16 per cent.).
Vesicles and roseolæ (maculæ) occur in crops and are simultaneous; very rarely shotty.	Never in crops; first macules, then hard, shotty papules, followed by vesicles, etc.
Rash evolution; is much more rapid; vesicles on the first or second day.	Much slower; vesiculation on the seventh day (see Pfeiffer's table).
Eruption is universal, successive crops; most abundant and characteristic on back; begins on body; less on face, hairy scalp, mucosæ, hands, and feet.	Note place of first development; development progresses downward—face first, then hands and feet; less on trunk.
The vesicle is superficial, and the transparent fluid shines through. (Wyss has observed confluent eruption and umbilication).	The fluid is pearl-colored, not transparent, and has a thicker covering.
Areola (halo) is usually absent.	Marked.
Involution is rapid.	Tardy.

Mistakes may occur among smallpox experts, but attention to the history, somatic findings, and course, rather than to the eruption, prevents many disastrous results.

MEASLES———*vs.*———VARIOLA.

Both present the same prodromal duration; in each the eruption begins on the face; according to some observers, simultaneously on face and trunk.

Age; eminently a childhood affection.	Chiefly in adults.
Catarrhal stage characteristic.	Catarrh present, if at all, in rudimentary form.
Fever; present in catarrhal stage, but low before exanthem, and rises with its appearance.	High before eruption, and falls with florition of the exanthem even in severe cases.
Exanthem; Koplik's spots on buccal and labial mucosa; bluish-white surrounded by red.	Absent. Early papular eruption on palate, etc.
Eruption later with rise of fever.	Initial morbilliform eruption with the primary fever.
Papules "grouped," crescentic.	Never so.
Papules remain relatively flat, smooth, velvety.	Change into vesicles, pustules, crusts, scars; hard and shotty.
Grisolle's sign absent and the eruption disappears under pressure.	Grisolle's sign present; *i. e.*, persistence in smallpox of the papule on stretching the skin, which distinguishes its eruption from scarlatina, varicella, etc.
Areola (halo) absent.	"Halo" present.

Scarlatina	*vs.*	Variola.
Rash on the second day, angina, strawberry tongue, desquamation, glands, nephritis, etc. (See cardinal symptoms of scarlet fever.)		Rash later; slow evolution of different phases. No angina (or late, at least), no glands, no strawberry tongue; the initial scarlatinal eruption is not punctate, less extensive and bright, and does not desquamate.
No double elevation of fever-curve.		Double summit to curve.

In *drug rashes* (copaiba, etc.), the pustules occur largely on the face, head, arms, and forearms, and much less frequently upon the trunk or legs. In *syphilis* the initial lesion, the glands, the polymorphism of the rash, and the absence of fever, or its presence in moderate form, are easily distinctive.

The lumbago form of *acute nephritis* (Robin), acute myelitis or rheumatism, erythema, menstrual rashes, septicæmic rashes, acne (cachectic and vulgar), and lichen, only superficially resemble variola.

Glanders may cause more difficulty in diagnosis. Its features are pyrexia, pains, papules, pustules, nasal discharge, considerable exudation, and the proper bacteriological findings. Its general symptoms are more severe than the degree of skin eruption would indicate.

Prognosis.—The prognosis depends on (*a*) previous vaccination, the disease being very fatal (50 per cent. mortality) in those unprotected by vaccination and revaccination. Smallpox is rarely fatal in persons who have been vaccinated within five or six years; only 8 per cent. of those vaccinated once, die and only 4 per cent. of those vaccinated twice. (*b*) On the *genius epidemicus:* In our last epidemic the death rate was but 3 per cent. (*c*) On the type, being (i) practically 100 per cent. in purpura variolosa; (ii) nearly 100 per cent. in variola pustulosa hemorrhagica; (iii) 60 per cent. in variola confluens—especially fatal in the first decade of life; (iv) 25 per cent. in variola discreta,—particularly dangerous in those over fifty or under ten years, in pregnancy, in the puerperium, in weakly and in alcoholic subjects; (v) 1 per cent. in varioloid; (vi) recovery usual in *variola sine exanthemate.*

d. The prognosis also depends upon *individual symptoms:* 1. Incubation is short in unfavorable forms, especially in purpura variolosa.

2. Initial symptoms are mild, chiefly in varioloid, but may be severe in both the benignant and malignant forms.

3. The initial rash is usually a partial erythema in mild forms of smallpox; in general, morbilliform eruptions occur in varioloid. Scarlatiniform eruptions occur in rather severe forms, and a universal scarlatinal hemorrhagic eruption, especially in the groins, occurs particularly in purpuric types.

4. The danger is proportionate to the amount of the eruption on the face and hands (Sydenham). A slow typical exanthem indicates variola discreta, and a precipitate atypical eruption presages variola confluens.

5. A prompt fall in temperature after the eruption is favorable. A slow, complete fall occurs chiefly in the severe forms. Persistent apyrexia, after florition and early desiccation indicate a mild type. Accession of fever after pustulation is ominous (Rhazes). There are fewer complications with an early fall of temperature. Hyperpyrexia or temperature collapse during suppuration is a sign of imminent death.

6. In the stage of suppuration the danger is parallel to the height of the fever and the amount of cutaneous and mucous efflorescence. Enlargement of the spleen is ominous.

Treatment.—1. Prophylactic Vaccination.—*Vaccinia.*—Protection from smallpox by cowpox vaccination was discovered by Jenner, who published his results in 1796, although he was certain of his evidence twenty years earlier. The idea had been long prevalent among farmers that cowpox afforded immunity to smallpox. Professor Waterhouse, of Harvard, in 1800, introduced vaccination into this country, and, among others, President Jefferson was instrumental in disseminating its practice.

The nature of cowpox, or vaccinia, is still bitterly disputed. The most probable view is that cowpox and horsepox are human smallpox modified by transmission through animals. Copeman suggests that vaccinia and variola have descended from a common stock. Variola inoculated in the cow produces typical vaccinia. The other view, held largely by the French, is based upon Chauveau's experiments, which conclude that the two affections are distinct. The bacteriology of vaccinia is also uncertain; micrococci, bacilli, and amœbæ (Guarnieri's *cytorrhyctes vacciniæ*) have been found.

Usual Symptoms. The incubation period is three days, during which there may be slight traumatic local reaction. The eruption occurs on the third day as a hard papule surrounded by a delicate halo. The papule enlarges, and on the fifth (or sixth) day shows Jenner's vesicle filled with clear serum, and umbilicated in the centre. It merges peripherally into the already indurated and painful skin, in which, by the eighth day, the magnifying lens shows many minute vesicles. By the tenth day, the serum of the vesicle has become purulent and the surrounding tissues are very hard and painful. By the eleventh day, a small central crust appears, which, by the end of the second week, covers the entire vesicle and is accompanied by a recession of the adjacent cellulitis. In one or two weeks, the ulceration, decrustation, and granulation, are usually complete, and a scar or pit remains, which pales with time. Constitutional reaction is greatest in children. The fever in most cases appears on the fifth day, and sometimes lasts four or five days, with leukocytosis, restlessness, headache, or backache. A swelling, depending on the site of inoculation, occurs in the axillary or inguinal glands. School physicians should not accept certificates of recent vaccination but should watch the evolution of the vaccination.

Unusual Symptoms.—The vesicles may develop early and run a

course which may be shorter by half than the usual one. Crusts may form in a week. In other cases the eruption may be tardy and atypical. In both instances revaccination is indicated.

Complications.—These are rarely fatal. In Germany, in 1895, two deaths (from erysipelas) occurred in two and a half million vaccinations. To offset this theoretical risk there is the almost universal susceptibility to smallpox in all unvaccinated individuals. (1) *Erythema,* urticaria, roseolæ, or erysipelas (third to eighth day). (2) Accessory vaccinia near the original inoculation, or generalized vaccinia, which develops about the eighth day on the trunk, extremities, and wrists; bullous or miliary vesicles sometimes continue to develop for over a month, and in weakly subjects are very exceptionally fatal. (3) *Secondary infection* in the first week or two, either local (deep ulceration, gangrene, lymphangitis, or lymphadenitis), or systemic (sepsis, pericarditis, meningitis, and parotitis). Very few cases of *tetanus* have occurred; McFarland (1903) collected 68 certain cases, the majority of which occurred in Philadelphia. Albuminuria is infrequent. (4) *Later infections,* such as tuberculosis or syphilis. Tuberculosis is but seldom inoculated, because it is rare in calves. Its inoculation can easily be prevented by the Belgian method of not using the lymph until the animal has been killed and found healthy. In some cases a latent tuberculosis in the patient may be awakened by vaccination, which seems to act like tuberculin. The danger of inoculating syphilis is far less now than when human virus was used. Fifteen days are necessary for the development of a chancre, and after about eight weeks more the secondaries appear. Syphilis may be inoculated accidentally and also intentionally, as in the oft-cited case of Dr. Cory. In exceptional cases syphilis already in the system may be aroused by vaccination. There are no clear cases of inoculation of leprosy.

Age.—Children should be vaccinated in the third or fourth month, and it should *never* be delayed beyond the first year.

Technique.—The arm (or leg, in the case of girls), should be washed, but no antiseptics should be employed, lest they neutralize the virus. A fold of skin should be raised and either rubbed with a piece of sterile gauze or scraped with a knife held almost parallel to the skin, until the epidermis is removed and slight oozing of lymph appears. The virus must reach the lymph stream, and therefore blood should not be drawn, as coagulation somewhat inhibits "taking." The vaccine should be slowly rubbed over and into the abraded skin. The spot should be covered with a sterile dressing or cloth held in place by adhesive plaster, only after the virus has thoroughly dried. Boric, carbolic, or bichloride gauze must not be used.

Lymph.—Vaccinia virus is harmless if it is pure and has been collected in the vesicular and not in the pustular stage. It can best be preserved in the fluid form, in sealed sterile tubes with three or four parts of glycerin. This obviates pyogenic infection, preserves the virus longer (four months), and is more economical than any other

method; twenty to twenty-five times as many vaccinations are possible with the same amount of virus when preserved by this method. When human virus must be used it should be taken from a perfectly sound child which has been carefully examined for tuberculosis and syphilis. It should be taken from the clear and unbroken vaccinia vesicle on the seventh or eighth day. No blood should be withdrawn, and the vesicle must be free from pus.

Results.—The successful vaccination, accompanied by typical papulation, vesiculation, pustulation, crustation, cicatrization, and by local and often general reaction, confers immunity in almost all cases, though there are isolated instances when variola has closely followed successful vaccination. Although immunity is not absolutely infallible, vaccination is the greatest triumph of prophylaxis yet achieved. Immunity is not as a rule life-long, as Jenner thought. In some few cases, the individual is permanently protected by one vaccination, and revaccination is unsuccessful. Protection lasts on an average ten years, after which 60 (or 90) per cent. can be successfully revaccinated once or twice. Conversely, the child should be revaccinated in his tenth to twelfth year, and if the vaccination does not "take," it should be repeated. The reaction, fever, induration, and size of the vesicle and scar, are less in revaccination. It is also held that immunity is more certain and enduring when more than one inoculation has been made; the Germans vaccinate two areas or even four or six. The nature of the immunity conferred is still disputed, but, unlike the immunity of diphtheria, it is probably due to actual tissue change. Pfeiffer advances the idea that in smallpox the germ endures in the tissues, and in this way maintains permanent immunity. Vaccination immediately after exposure is also thought to prevent or modify an impending attack. Partial immunity is evidenced by mild varioloid (smallpox mitigated by vaccination). The general beneficence of vaccination needs no multiplied statistics. In general, epidemics prevail in communities which are least protected, and conversely, they disappear with systematic compulsory vaccination. The lesson regarding the alleged immunity is that repeated, careful vaccination nearly always produces vaccinia.

2. Other Methods of Prophylaxis.—Isolation hospitals and stations are indispensable, because smallpox patients are infectious in all stages of the disease. Isolation is necessary from the incipiency until the disappearance of the last crust. The dead body is dangerous and the funeral must not be public. The clothes used by the patient must be steamed, and other articles must be washed with bichloride of mercury and fumigated with formaldehyde vapor; Schoch's method has the advantages of not requiring any special form of apparatus, of cheapness, and freedom from danger of fire. The following are required: Good quicklime in lumps, commercial sulphuric acid, and ordinary 40-per-cent. formaldehyde solution. For every 1,000 cubic feet of space, the size of the average room, one pound of commercial formaldehyde (40 per cent.), one-half pound of sulphuric acid, and

three pounds of quicklime are required. The acid and the formaldehyde are first mixed in an earthenware vessel by pouring the acid into the formaldehyde. Then the lime is placed in a shallow vessel in the centre of the room. All openings to the room are carefully closed, the mixture is poured on the quicklime, and the operator leaves the room. The rooms should remain closed for from five to eight hours. Disinfection of the face, hands, beard and hair of attendants by bichloride solution is imperative. The family should be isolated for sixteen to twenty days.

3. Actual Treatment.—As Jaccoud early announced, there is no specific treatment for smallpox. Its *hygiene* and *dietary* are the same as in typhoid. For *headache,* the ice-bag, opium, and hydrotherapy are employed. *Pain* is relieved by opiates.

The initial *vomiting* is difficult to overcome, but its duration is fortunately short. Ice-pills, lime-water, iodine, chloroform, morphia, carbolic acid, and bismuth, may be used as they are in typhoid (*q. v.*); mustard plasters may necrose the skin.

Early hydrotherapy is important in *fever.* It lessens the dermatitis, though later, when the rash has developed, baths are contraindicated.

Delirium may be relieved by hydrotherapy, by chloral, bromides, hyoscine, and morphia. The latter, in half-grain doses, is invaluable as a routine treatment, since it lessens *itching* and pain and conserves metabolism. There is great danger that the patient, if delirious, may escape, injure himself, or commit suicide. Dr. McNamara, a classmate of the author, killed himself by jumping through a window which had been carelessly left open near him. Patients should never be confined by straps, for this may cause serious skin necrosis. It is better to dress them, or even walk them up and down the room.

The *eruption* may be pricked and cauterized with silver nitrate (Arabian method), treated with bichloride or boric compresses (Hebra), covered with mercurial plaster, or with 1-per-cent. carbolic vaseline. The latter is the best, because it counteracts the particularly offensive odor. Carbolic salve should be used on the face only, since general use over the body may cause carbolic-acid poisoning. These measures are not specific nor do they prevent cicatrix formation, but they relieve dermatitis and itching. Protecting the face from the light with wet compresses seems to lessen the pitting. The crusts should be kept soft with olive oil. The hair should be cut short, and the eyes should be closely watched, and douched frequently with boric-acid solution.

The mouth, the larynx, the circulation, and diarrhœa, should be treated as they are in typhoid fever. For bed-sores, a water-bag or -ring should be used. Sydenham allowed his patients to leave bed as soon as the fever declined.

For sepsis, quinine (*v.* sepsis) and alcohol (*v.* typhoid) are given by the rectum, since dysphagia is always present. Eggnog properly prepared will not injure the throat. During convalescence the crusts

should be carefully washed, and the patient may be considered safe only when the skin is clear of the last scab.

CHICKENPOX (VARICELLA).

Definition.—An acute specific febrile contagious disease, chiefly one of infancy and childhood, characterized by an exanthem of vesicles which usually appear in crops.

History.—Varicella was recognized by Ingrassias in 1553, Heberden in 1767, and by Trousseau, as a disease distinct from smallpox.

Etiology.—It is transferable by direct inoculation, by personal contact, by the air, or by a third person. It is infective while crusts remain. Epidemics of varicella may coincide with, precede, or follow, smallpox. Smallpox does not prevent an attack of varicella nor is the converse true. The disease occurs chiefly in epidemics. Season has no influence upon the appearance of the disease. It occurs chiefly in the first year of life, or, according to others, 66 per cent. of the cases occur between the years of two and six. A second attack is rare.

The actual cause is not determined. Bareggi described a peculiar coccus; Guttmann, a staphylococcus; and Pfeiffer and Guinon, a protozoön.

Symptomatology.—1. Incubation.—The stage of incubation, like that of measles, lasts thirteen or fourteen days.

2. Stage of Prodromes.—Prodromal symptoms are rare. If there are any prodromal symptoms, they last but twenty-four hours (Trousseau). Severe intoxication is rare, but the following have been observed: Delirium, convulsions, violent vomiting, hyperthermia, dyspnœa, angina, conjunctivitis, dysphagia, muscle pains, lassitude, intestinal irregularity, bloody vomiting and stools, and an initial erythema (usually scarlatiniform, rarely morbilliform, in the fifteen cases recorded).

3. Stage of Eruption.—The exanthem sometimes appears suddenly after a slight chill, usually in the first twenty-four hours, as red *macules* or *papules*. They are comparable to the typhoid roseolæ, are slightly elevated, disappear on pressure, and are lenticular. Some regress, but most become *vesicular* in from six to twelve hours. The vesicles mature within a day. They are very superficial, and do not occupy the whole roseola, but leave a slight areola about them, which is hyperæmic and not inflammatory as in smallpox. In some cases the areola may be absent, the eruption appearing like drops of water sprinkled on the skin. Though usually discrete, Wyss and Immermann describe confluent eruptions. The vesicles may be few or numerous; Thomas counted 800 in one case. The eruption is mostly elliptical or circular. Rare forms are reported with jagged, sinuous edges, when the vesicle overlaps the skin. The vesicles may be very large, and, in exceptional cases, resemble pemphigus.

The eruption begins on the trunk, back, and chest (Osler, Guer-

sant, Blache); on the face, temples, forehead (Eichhorst, Immermann), and, according to Thomas, rarely fails to appear on the scalp. Trousseau held that there is no point of predilection for the eruption. Henoch finds it often on the part on which the patient lies or where irritation exists. No part can be considered exempt, though most vesicles appear on the trunk, fewer on the face, arms and legs, and the least number upon the hands and feet. The eruption is sometimes grouped, as it is in herpes zoster.

The contents of the vesicle is clear at first, becomes turbid in thirty-six to forty-eight hours and is neutral or alkaline. Pustulation and hemorrhage into the vesicle rarely occur. The vesicle may contain air after rupture. Umbilication, though rare, may occur (Wyss); Eichhorst denies the statement made by Goruall that the umbilication of variola exists from the beginning of the eruption, while that of varicella occurs late. Crusts may simulate umbilication. The vesicles of varicella are not always unilocular.

Occurrence in crops is frequently observed; the skin then shows simultaneously macules, vesicles, and crusts. Yellowish-brown crusts form on the third and fourth days and gradually disappear, leaving a somewhat flattened epidermis, which may desquamate slightly. Distinct scars may result from scratching or infection, and a few cicatrices are usual in every case of varicella, but they are smooth, white, and some of them disappear with time.

Enanthems are not frequent; vesicles seen on the palate may easily be overlooked; breaking down, they resemble aphthæ. On rare occasions they are seen on the tongue, cheek, nose, larynx, external ear, prepuce, or labia.

Fever.—The fever is slight, if indeed there be any. It is atypical and is not parallel to the eruption. Fever as high as 106.8° is recorded by Thomas. It is very rarely present in the prodromal stage, but may occur with profuse eruptions, or with successive crops; its persistence suggests complications.

General Symptoms.—These sometimes include dysphagia, cervical and submaxillary swellings, tracheitis, bronchitis, or albuminuria.

The entire course of an uncomplicated case lasts one to two weeks.

Complications and Sequels.—These are usually dismissed with a few words. *Nephritis,* first described by Henoch, is generally mild, tubular, and occurs from three to fourteen days after the rash declines. Though usually mild, fatal cases have been reported, as well as some eventuating in chronic nephritis, cardiac hypertrophy, and uræmia. *Gangrene* of the skin, seen in delicate or tuberculous children, and first described by J. Hutchinson, may occur in the extremities, scrotum, or eye-lids. *Pneumonia* is rare. Infrequent complications are abscess of the lung, pleurisy, acute miliary tuberculosis, erysipelas, sores in the skin, glandular swelling, pemphigus, infantile hemiplegia, osteitis, polyarthritis, peritonitis, otitis media, neuritis, etc.

Diagnosis.—The diagnosis is very easy if the case is seen early. Differentiation from smallpox has been considered. There is usually no fever, the eruption is more vesicular, without halo, larger, but less in number than in smallpox. In *pemphigus* the vesicles are larger and run a slower course of weeks or months. *Miliaria rubra* are preceded by sweats, occur chiefly on the covered parts, have acid contents, are smaller than the varicellous eruption and disappear more rapidly. Sometimes consideration must be given to molluscum contagiosum (soft, umbilicated tumors with white granular contents); prurigo varicelliformis (with crop-like eruptions at longer intervals); eczema vesiculosum, which always occurs with great itching; herpes, always appearing in groups and often along nerve-trunks; varicellæ syphiliticæ, and medicinal rashes from cantharides, bromide, arsenic, and iodide.

Treatment.—Active treatment is rarely indicated.

SCARLET FEVER (SCARLATINA).

Definition.—A specific infective disease of unknown bacteriology, characterized by sudden onset, fever, diffuse exanthem, and angina.

History.—It was recognized by Ingrassias and Coyttar in the sixteenth century, but was first fully described by Sydenham (1660); before this, it was known as measles. Scarlatina was introduced into America in 1735.

Etiology.—The etiology is unknown. Babes, Babinsky, Pearce, Wright, and others, have found a streptococcus in 58 per cent. of fatal cases, and, while their results in no way prove that it is the cause of scarlatina, it is probable that the *streptococcus* is the most important factor in the production of complications and in their mortality. Hektoen, Weaver, and Ruediger, in 100 cases, found it in the blood in 12 per cent. Combining several statistics, the streptococcus was found in about 70 per cent. of the fatalities; *i. e.*, it is the cause of the malignancy of the disease, but not of the disease itself. The virus of scarlet fever produces severe necrosis, but no suppuration. Mallory describes a protozoön, the *cyclaster scarlatinalis.*

Susceptibility is not universal and is inversely proportional to the age; only 38 per cent. of children and 5 per cent. of adults exposed to infection acquire the disease. Ninety per cent. of cases occur under ten years of age (Murchison). It is rare in the first year of life. Susceptibility varies, also from time to time in the same family.

So-called *"surgical and puerperal scarlatina,"* noted in 1864 by Paget, are, in the vast majority of cases, sepsis, erythema congestivum, erythema toxicum, or drug rashes. Alice Hamilton (1905) collected 174 cases from the literature, and concluded that these forms are mostly septic, or that scarlatina is a mere accident in a surgical case.

The *virus* circulates in the blood, whence the possibility of fœtal

infection. The mother may contract the disease and the child may escape. It is usually held that the virus is especially active during desquamation; Aaser holds, and justly, that the throat secretions are also dangerous. The streptococci may be spread by coughing (Hamilton). Infection is often spread even two months or more after desquamation is complete. In no other disease is the virus so *tenacious;* it may persist as long as ten years in clothes, furniture, and on the walls. Light forms are as contagious as severe ones. Inoculations from the living subject have occurred and Leube acquired the disease by a cut during an autopsy upon a scarlatina case. The atrium is not known, but it is probably through the throat.

As to the degree of *infectiousness,* smallpox ranks first, measles second and scarlatina third. Infection may be spread by servants, by physicians, by any third person, and by means of toys, books, clothes, and milk, and in schools; but the mode of infection is often unknown. One attack usually confers *immunity,* but second, and even third, attacks are known. In some individuals there appears to be a temporary or permanent immunity. Scarlet fever occurs at all seasons, but probably more often in the autumn and winter. Scarlatina is observed more in the cities, and measles prevails more diffusely through the country. Scarlatina is more permanent than measles. When it becomes epidemic it is likely to spread widely and usually with increased mortality.

Scarlatina sometimes occurs with other infections, such as diphtheria or measles, and less often with variola, varicella, pertussis, erysipelas, or typhoid.

General Symptomatology.—1. The Stadium Incubationis.—The incubation, with no noticeable symptoms except perhaps anorexia or slight depression, lasts from two to four days; McCollom places the average at ten to fourteen days.

2. The Stadium Invasionis.—The invasion lasts one day. Symptoms begin suddenly, with perhaps a chill. This is followed by (*a*) *vomiting,* early, and characteristic, which occurs in scarlatina more often (75 per cent.) than in other diseases of childhood except pneumonia; (*b*) headache, dulness, *convulsions,* and delirium; (*c*) *sudden elevation of temperature,* even to 105°, with disproportionate *pulse-rate,* 120 to 150, with dry, burning skin, and febrile urine; (*d*) dryness, redness, burning, *pain in the throat,* dysphagia, and *intumescence of the cervical glands.*

3. The Stadium Exanthematicum.—The exanthem lasts four to six days. It appears on the second day, first below the clavicles, on the upper trunk, and on the neck, then on the extremities, and least on the face and scalp. The skin about the mouth is pale. The exanthem pales on pressure. It consists first of small red spots (swollen hair follicles), the size of a pin-head, which fuse as the skin swells, and result in an intense diffuse lobster-colored erythema. The tongue swells, becomes covered with a grayish-yellow coating, is red on the tip and edges, and shows swollen red papillæ

—" cat's tongue," or " strawberry tongue." Later the whole tongue becomes red and dry. On the tonsils and palate a grayish-yellow coating and confluent spots appear. The nostrils become slightly swollen, and redness of the conjunctivæ develops with secretion. Some rales over the larger bronchi, a slight cough, swelling of the cervical and other glands, and sometimes albuminuria, may be noted.

4. The Stadium Desquamationis.—The desquamation lasts several weeks, though the rash itself lasts but three to six days. Desquamation begins first on the face, from the sixth to ninth day. The glandular swelling disappears, the fever falls by lysis, and convalescence is established unless complications intervene.

Special Symptoms, Complications and Sequels.—These are given in detail because of the extreme variability in the clinical symptoms:

1. The Fever.—The fever is less typical than in smallpox and measles. In the invasion there is a rapid rise to 104° or 105°. The fever is usually somewhat higher on the second day, with slight morning remissions and gradual subsidence when the rash is mature.

In the most severe types there may be low fever or no fever at all, or it may rise to 107°. We usually note a close parallelism between the abundance of the exanthem and the height of the fever. The temperature should be taken during one month after the rash disappears, lest such complications as sloughing of the throat, pleuritis, endocarditis, pericarditis, synovitis, or adenitis be overlooked. A long course of fever may exceptionally occur without complications, but it is rather ominous.

2. The Eruption.—The eruption is often atypical. Typically, it begins in the form of light-red, pin-head-like points which become darker as the skin swells and the eruption diffuses. It then appears as an even red, and on close inspection is found beset with small points. These are the first part of the exanthem to develop, and the last to go. The eruption, then, first consists of red spots, especially in the axilla and groin; second, of red spots in a diffuse erythema; and lastly, of red spots again. This minute papulation is a very important aid to diagnosis in the second week. There may be minimal white lines in the eruption, but rarely ever spaces as there are in measles. The skin is sometimes vesiculated. Though it spreads less regularly than in measles, the eruption appears first on the neck, below the mastoid processes, and below the clavicles, then upon the trunk, arms, and hands, and lastly upon the legs and feet. It is most intense over the extensor surfaces of the joints and on the trunk, and is least developed on the face and scalp. The palms of the hands and soles of the feet are usually free. The pallor around the mouth contrasts vividly with the scarlet cheeks and minutely papulated forehead. This pale area is circular or triangular, bounded by the nasolabial folds above and the chin below. Though not pathognomonic, it is always valuable in differentiation, and is due possibly to vascular spasm. Herpes near the angles of the mouth is very common. The eruption is occasionally roseolous about the

ankles and wrists. The skin is always swollen, and sometimes itches, or is wet with sweat and vesiculated. The eruption is hyperæmic, and therefore pales on pressure and disappears after death, unless there are punctate hemorrhages, which are not uncommon in severe types of the disease. After twenty-four hours of the eruption, the tension disappears, and the bright-red color becomes a yellowish-red. Pathological changes in the skin, according to Unna, consist of hyperæmia, especially in the papillæ, but rarely œdema or inflammation. After complete florition the skin becomes dry, and desquamation begins. This is usually bran-like as it is in measles, on the head, face, neck, and trunk, and it is more scale-like and lamellar on the hands and feet, complete casts of which are sometimes shed. The scales are often large in proportion to the intensity of the eruption. In severe cases even the hair and nails are shed, and the nails become furrowed. Desquamation results from a slight exudation of serum between the layers of the skin. It begins on the cheek with the "rouge-and-powder" appearance, and is usually first observed when the fever and angina decline. The skin may present a "pin-hole" or "worm eaten" aspect before desquamation begins. Its duration is most variable, usually lasting from one-half to three weeks. *Anomalies of eruption: Scarlatina lævigata* is the typical rash described above. Other forms are *scarlatina lævis* (rudimentary or localized eruption); *scarlatina sine exanthemate* (the rash being absent); *scarlatina papulosa* (marked papulation); *scarlatina miliaris* (from sweating, and exudation between the rete Malpighii and the epidermis); *scarlatina variegata* (like measles, though not equally elevated); *scarlatina hemorrhagica* (usually ominous, and at times part of a general hemorrhagic diathesis). In general, irregular forms are dangerous.

3. The Throat.—The throat changes are the most constant lesions in scarlet fever, and constitute the internal eruption (enanthem). The disease begins in the throat with dryness, uniform redness, and invariably with early swelling. The throat very soon becomes speckled with fine red points like the skin papulation. Though always present, it varies in intensity from simple angina to diphtheritic or gangrenous inflammation: (*a*) In mild cases, a simple catarrh with little swelling, a thick mucous coating, and more or less reddening of the palate and tonsils, develop. (*b*) In moderate cases there is, in addition to the above, more swelling of the palate, and tonsillitis. (*c*) In severe cases, membrane, small abscesses, gangrene, small hemorrhages, sloughing, and, perhaps, sudden hemorrhage, œdema, adenitis, periadenitis, or cellulitis, develop. Severe inflammation in the throat occurs in 65 per cent. of cases (von Ranke).

Clinical and anatomical diphtheria are often confused. "Necrotic inflammation" in scarlatina is that caused by the virus of scarlatina alone. Genuine diphtheria may accompany or follow scarlatina; of these, post-scarlatinal diphtheria is the more dangerous (Trousseau).

Diphtheritic Membrane	*vs.*	Scarlatinal Membrane.
Firm, adherent.		Softer, loosened in fragments.
Yellow-gray.		Brownish tint.
Deep ulceration rarer, in more severe cases only, and later.		More frequent and earlier (even in twenty-four hours).
Phlegmon rare.		Not infrequent.
Laryngeal membrane (croup) frequent and deeper.		Rare, membrane thinner; lung complications rare.
Bacteriologically, the Klebs-Loeffler bacillus.		Streptococcus (Klebs-Loeffler bacillus only as a complication).
Paralysis frequent.		Rare, except local paralysis due to local swelling of pharynx.
Recurrence frequent.		Infrequent.

Since the bacteriological cause of scarlatina is undetermined, it is doubtful whether the pyogenic cocci found in the throat are integral or complicating.

4. The Respiratory Tract.—Though often unaffected, the respiratory tract may become secondarily involved by a descending process. The nose is involved in direct ratio to the severity of the throat developments, such as necrosis, diphtheria, and gangrene. Acute œdema of the glottis is infrequent. The bronchial mucosa is more often involved than is clinically recognized. Bronchopneumonia, acute lobar pneumonia (usually in the upper lobes and co-existing with nephritis), gangrene, infarcts, inflammatory œdema and hypostasis, are not common. Pleurisy, described especially by R. P. Howard, occurs in 5 per cent. of the cases. It is often overlooked, is likely to be severe, is very frequently purulent, and often occurs during convalescence.

5. The Circulation.—The *heart* is greatly damaged by the scarlatinal virus in 35 per cent. of cases, not including the cases of sepsis and nephritis (Schmatz). Endocarditis is frequently mural, whence its frequent latency, but it may result in chronic valvular disease, especially of the mitral valves. "After rheumatism, scarlatina is the most frequent cause of endocarditis" (Leube). With increased arterial tension due to kidney disease, the heart is slowed, and hypertrophy may result, unless pancarditis (endo-, myo-, and pericarditis), exist. Pericarditis is usually septic. Disquieting cardiac dilatation may be seen in severe cases, accompanied by the usual evidences of acute cardiac insufficiency; *myocarditis* may occur during the fever (Rhomberg), or in convalescence (Schmaltz). Tachycardia may often be observed from toxæmia or acute infective myocarditis. Dilatation and hypertrophy are much more frequent in children than in adults, and often complicate renal lesions. The pulse is irregular in 90 per cent. of cases. *Blood.* Leukocytosis develops early and may become extreme in fatal cases. The polymorphonuclears are

absolutely and relatively increased two or three days after the eruption. The eosinophiles are also increased. The hæmoglobin and red cells suffer an early and increasing reduction. The red cells average 4,000,000. Hemorrhagic diathesis seldom occurs except as a result of sepsis.

6. The Digestive System.—The tongue is swollen, indented, broad, and thick. Its papillæ become larger and separated from each other. Its coating is first grayish, and only the edges and tip of the tongue are reddish, but later the coating disappears, leaving the tongue very red, dry, and glistening, on which the papillæ appear granular and warty—the "strawberry," "raspberry," or "cat's tongue." This appears from the third to the fifth day and lasts a week. It is quite pathognomonic, according to Leube, and is only second in importance to the angina. McCollom found it the only constant sign in 1,000 cases of scarlatina. The breath may be sweet and heavy. Diphtheritic ulcers and secondary necrosis in the stomach, œsophagus, and intestines, have been found. Vomiting may result from nephritis, brain toxæmia, or coughing. The liver and spleen may be slightly swollen. There may be abdominal pain. Constipation is usual. Diarrhœa, however, may result from (*a*) simple transitory catarrhal enteritis; (*b*) dysentery, with tenesmus and blood; (*c*) the so-called "scarlet-typhoid" of Thomas, which is accompanied by tympany, hemorrhage, very rapid pulse, and lethal outcome. Its resemblance to typhoid is heightened by the autopsy findings of splenic intumescence, and hyperplasia and ulceration of Peyer's patches.

7. The Kidneys.—The urine is febrile. It often responds to Gerhardt's reaction and to the diazo test. *Albuminuria* occurs in 75 per cent. of all cases. The "initial catarrhal nephritis" is accompanied anatomically by hyperæmia, parenchymatous changes, some fluid in the glomeruli, a few cylinders in the tubules, and little interstitial involvement and runs a short course.

Nephritis usually develops in the third week. Its frequency varies with the epidemic, between 5 and 70, or even 90 per cent. No apparent relation exists between its frequency and the severity of the infection. No single anatomical form is invariable, but the glomerulonephritis of Klebs is most frequent. It is characterized *anatomically* by hyperæmia, increased consistence, and such glomerular changes as gray color, diffuse fatty granular degeneration, increase of nuclei, and increase in the size of the tufts, which become sausage-like solid masses. We also note exudation of leukocytes about the afferent vessels, multiplication of capsular epithelium with occlusion of the lumen, hyaline alteration of the vessels, and small and early interstitial changes, and later connective-tissue growth. Friedländer observed septic interstitial nephritis and the large variegated kidney, in cases accompanied by diphtheria, or phlegmonous complications.

Clinically, hydrops in the vast majority of cases is renal and shows

the characters of renal dropsy. It appears first under the skin, as the cutaneous blood- and lymph-vessels are altered by the virus. It shifts its location, varies without cause, and may also produce hydrothorax, ascites, œdema of the larynx, lungs, or genitalia. The older writers claimed that hydrops occurred without nephritis, and that kidney inflammataion might exist without appropriate urinary alterations. Such views can be proved by autopsy only. The temperature is inconstant. There may be lumbar pain, vesical tenesmus, and *uræmic symptoms,* such as headache, twitchings, convulsions, wide pupils, amaurosis, disturbance in motility, sensation, and cerebration, coma, involuntary evacuations, vomiting, diarrhœa, cardialgia; rapid pulse and breathing, and pale skin tinged with cyanosis. The retinæ are usually normal. Many *convulsions* usually imply a lethal termination in coma. The prognosis is more favorable if the pupils are not immobile. The *urine is decreased or suppressed,* is heavy with sediment and *albumin* (averaging 0.5 per cent.), is poor in urea, turbid (specific gravity 1030), and is *full of casts of all types*—especially, long, flat, band-like, twisted, and frayed casts. Much oxalic and uric acid is found and, in many cases, blood. Uræmia, hydrops, and intercurrent inflammation, are always dangerous. Convalescence is the rule after four to six weeks (eight to eighteen months), and the nephritis rarely becomes chronic.

8. The Special Senses.—Decreased secretion in the eye is common. Cerebral complications promote inflammation in that the eyes close less frequently. Catarrhal conjunctivitis, diphtheritic inflammation, involvement of the lachrymal sacs, herpes or necrosis of the cornea; panophthalmitis, muscular paralysis, and disturbance in accommodation, are possible complications. Uræmic amaurosis, neuroretinitis septica seu nephritica, and retinal hemorrhage, are rare. *Middle-ear* inflammation (11 to 33 per cent.) usually begins before desquamation, but may be observed later. It may easily be overlooked, and is accompanied by fever alone or with pain, glandular swelling, difficult hearing, tinnitus, a reddened, glistening, sunken membrane, and, in children, by some brain manifestations. It is quite frequent in early life and rare after the fifteenth year; 10 per cent. (even 42.6 per cent.!) of cases of acquired deafness are said to result from scarlatina.

9. The Lymphatic System.—The lymphatic system is generally involved; *e. g.,* glands, spleen, follicles, and in severe cases, all the lymphatic structures, are affected by the sepsis. McCollom found the cervical glands enlarged in 50 per cent.; they suppurated in 8 per cent. The "collar of brawn" results from cervical adenitis and periadenitis. Gangrene, retrosternal abscess, and jugular phlebitis with sepsis, occur in desperate cases. After recovery the neck may remain swollen for a long time.

10. The Bones and Joints.—Scarlatinal arthritis ("rheumatism") is found in 6 per cent. of the cases, usually in the second or third week, with desquamation and subsidence of the fever, and usu-

ally lasts three or four days. It attacks the smaller rather than the larger joints, the wrists rather than the elbows or shoulders, and the ankles rather than the knees. It develops with pain, and is not so migratory as ordinary rheumatism. It may leave no autopsy findings, or it may become purulent. Swelling of the joints does not always occur. It occurs in old rather than in young subjects. The prognosis is usually good. Bone involvement is rare.

Scarlatinal sequels are anæmia, hemorrhagic diathesis, retarded development, and decreased physiological resistance. Tuberculosis rarely follows scarlatina. Latent sepsis with brain foci, valvular disease, chronic nephritis, chronic adenitis, otitis, mastoiditis, meningitis, hemiplegia, cerebral venous thrombosis, facial paralysis, ascending spinal paralysis, chorea, and exhaustion psychoses, are fortunately rare complications and sequels.

Diagnosis.—Typical cases are easily determined when an epidemic prevails, and when the eruption is not considered of chief diagnostic value. The criteria are as follows:

1. Sudden onset and rapid development, with fever and vomiting.
2. Angina, pain, redness, punctate spots in vault of the mouth, swelling and dysphagia, which are practically always present. Severe symptoms in the throat, especially during an epidemic, are suspicious.
3. Strawberry tongue, which is constant.
4. Exanthem, first about the clavicles, with characteristic progression, the face less involved, and the mouth free; it is intense on body; papules then erythema develop; there are no free spaces; it results in desquamation.
5. Lymphadenitis. In this, the cervical are less important than the inguinal or other glands.
6. Fever, hydrops, albuminuria, and nephritis in atypical cases.
7. Scaling, tender joints, and albuminuria, are strong indications of scarlatina if the initial symptoms were not observed or were not clear; they help in making a retrospective diagnosis.

Diagnosis of Atypical Scarlatina.—1. Severe scarlatina. (*a*) The anginose form resembles septic diphtheria, with marked toxæmia, necrosis, and adenitis. (*b*) Atactic form. Violent early intoxication; high fever and death even on the eleventh hour of the disease (Trousseau), before the eruption appears; blood dissolution, vomiting, diarrhœa, convulsions, coma, and an excessively rapid, weak heart, are the chief symptoms. (*c*) Hemorrhagic scarlatina. This is characterized by severe fever and brain symptoms at the onset; incomplete exanthem, necrosing angina, marked glandular and splenic swelling; subcutaneous, serous and mucous membrane hemorrhages with exulceration. It is usually lethal after ten to twenty hours from hyperpyrexia, exhausting vomiting, diarrhœa, stupor, delirium, convulsions and dyspnœa. It is encountered especially in reduced subjects under two years of age.

2. Very light cases may present (*a*) all the usual signs, though

but mildly developed; or (*b*) some symptoms may be absent (*scarlatina fragmentaria*), as in cases with no temperature (*scarlatina sine febre*), no rash (*scarlatina sine exanthemate*), or no angina (*scarlatina sine enanthemate*). In some atypical cases a definite diagnosis is impossible, as in acute nephritis with uræmia, fever, sore throat, and gastro-enteritis, without the characteristic rash. Recurrent forms are not frequent. They occur in the middle of the third week, and result from incomplete elimination of the virus during the first attack. Many of these cases are septic complications rather than actual recurrences. According to Körner, only forty relapses are on record.

Differentiation.—The differentiation is as follows:

Scarlet Fever	*vs.*	Sepsis.
Bright-red erythema, with small red papules.		A very deep purple-red rash, sometimes spreading over entire body.
The eruption is much the same in both diseases, the same places being exempt.		
Miliaria are rare.		Frequent.
Rather typical desquamation.		Desquamation also observed, but less frequently.
Criteria (*q. v.*); angina, tongue, abrupt onset, glands, etc.		Etiology, chills, sweats, fever irregularity, polymorphous exanthems, etc.

Diphtheria is often differentiated with difficulty. A simple erythema is sometimes observed in diphtheria, which is darker, more on the trunk, and more transitory than in scarlatina (see Throat Complications).

Drug rashes, from belladonna, iodoform, quinine, iodide, chloral, or copaiba, may be easily differentiated, if the cardinal signs of scarlatina rather than its rash alone, are considered.

Measles and *German measles* (*q. v.*). *Acute exfoliating dermatitis* also begins with fever, with a spreading erythema, and very early desquamation, but the other cardinal criteria of scarlatina are lacking. *Erythema* is more transitory; the points are absent; it occurs with less fever and without angina or adenitis.

Erysipelas presents a sharp border, œdematous swelling, marked pain, and a slow advance.

Prognosis.—This should be guarded for at least one month. The outcome often depends on the *genius epidemicus,* since the mortality ranges between 2 and 40 per cent. The death-rate averages about 10 per cent. The prognosis is least favorable in hospital cases and in children. Most deaths occur under six years; in children under five years, the mortality is 20 to 30 per cent. (Holt). In the first year of life the disease is very fatal.

Uræmia accounts for 50 per cent., and respiratory complications for 20 per cent. of scarlatinal fatalities. The social status, severity of the infection, hemorrhagic forms, severe toxæmia, complicating sepsis,

necrosis in the throat, otitis, and heart complications (high pulse-rate being always unfavorable), are the other factors in prognosis.

Treatment.—There is no specific therapy and the disease runs its self-limited course.

I. Prophylaxis.—Prophylaxis is equally imperative in severe and light cases. Indeed, the light or ambulatory forms more often convey infection. Isolation is important, because susceptibility is not universal and the virus is less penetrative than in measles, and it is therefore possible to circumscribe its diffusion. Care should be taken to prevent transmission through schools and by means of fomites and by third persons. The infected room should be kept empty for one month afterwards, and should be exposed to the wind and sun for several weeks. No susceptible person should use the room for several months. The physician should wear an operating gown and carefully wash his face, beard, and hands, after leaving the sickroom. Quarantine should be maintained from six to eight weeks. Even this is probably too short a period. A sheet saturated with carbolic acid or formaldehyde, and hung before the door, is of no special value. Formaldehyde fumigation has been discussed under smallpox. Care in surgical and puerperal cases is important, but the danger has been overestimated. All discharges should be disinfected. Bichloride wrappings should be placed about the dead body, and the funeral should be private.

Schools should be closed during epidemics. In hospitals the cases of scarlatina with streptococcus infection should be separated from cases of simple scarlatina. Sound children should be sent away from the house and isolated lest they spread the disease. If they remain at home, about one-quarter of them will contract scarlatina (Chapin).

II. General Treatment. 1. *Position.*—The prone position in bed is imperative in all cases, both during the disease and in the first two weeks of apyrexia.

2. *Room.*—The room should be light, well ventilated, dry and warm, especially in convalescence. A grate fire is advisable and the windows should be open at the top. During the fever the skin should be covered with linen, and in convalescence with wool.

3. *Skin.*—The eruption should be brought out, when necessary, by mustard baths. During the fever, the patient should be washed daily with warm water and soap, though unnecessary exposure should be avoided. The tension of the skin is thus lessened, the fever decreased, and toxæmic symptoms, such as insomnia and restlessness, are allayed. During desquamation, oil-rubs were once employed to decrease the risk of infection by dissemination of dry scales but are now considered deleterious, as they decrease the functions of the skin, which are of great importance when the kidneys are involved. Besides, infection is more probably carried by means of throat secretions, than by desquamation. Soap and warm water serve equally well. The author is opposed to antiseptic soaps because of the chance of absorption which may result in renal injury.

4. *Fever.*—The following is a harmless fever mixture:

℞ Liquoris ammonii acetatis ..... ʒiss.
Tincturæ aconiti ..... ♏xxiv.
Aquæ .....q.s. ad ʒvj.
Misce et fac misturam.
S.—One to three teaspoonfuls every four or five hours, according to age.

This mixture tranquilizes, promotes the rash, and allays fever. Hydrotherapy is useful, such as the warm sponge or full warm bath. Special care is indicated (*a*) in adynamic cases, because of danger of heart-failure; therefore the full cold bath should always be avoided and the moderately warm bath should be used; (*b*) in pericarditis; (*c*) in stenosis of the air-passages, or dyspnœa; (*d*) in hemorrhagic forms; (*e*) in arthritis; and (*f*) in nephritis.

5. *Diet.*—Water should be given very freely. A *fluid diet* is indicated by the fever, the percentage of renal complications, and the dysphagia. This may consist of concentrated foods, milk, soups, and jellies. In defervescence, milk-toast, custard, puddings, and bread and butter, may be given. Eggs and meats may cause albuminuria, and should therefore be avoided. Fruit and vegetables may be given freely at all stages, if this can be done without irritating the throat. In severe throat complications, rectal feeding is indicated. (See Gastric Ulcer.)

6. *Scarlet Arthritis.*—The A.B.C. liniment (aconite, belladonna, and chloroform), warm wool and flannel bandages, are helpful.

℞ Tr. opii ..... ʒiij.
Phenolis ..... ʒj.
Tr. belladonnæ ..... ʒiij.
Ol. olivæ ..... ʒiv.
M. et S.—Applied on muslin covered by cotton.

Salicylates are most beneficial, but unless given carefully, may injure the kidneys. If the arthritis is obstinate or purulent, aspiration with all surgical care and even drainage may be indicated.

7. *Angina.*—Ice in the mouth, ice-cravats, hot fomentations especially for the enlarged glands, and Dobell's solution as a nasal douche should be given. Care should be exercised in the use of antiseptics lest children swallow them. A 10-per-cent. peroxide of hydrogen, 2-per-cent. chlorate of potash, or 2-per-cent. carbolic gargle, may be used. Formalin gargles are too irritating. Caustics are contra-indicated.

For the nose the following formula is excellent:

℞ Phenolis ..... ʒss.
Acidi borici ..... ʒss.
Glycerini ..... ʒj.
Tr. myrrhæ ..... ʒj.
Aquæ .....q.s. ad ʒv.
M. et S.—Locally, in such dilution that no irritation is felt.

Heubner injects 3-per-cent. carbolic solution into the tonsils (ʒss b.i.d. on each side). A 10-per-cent. nitrate-of-silver solution is used

with great advantage in the early stages of the angina. Coincident diphtheria is treated by antitoxin (described under diphtheria), and the antistreptococcus serum may be exhibited in streptococcic angina.

8. *Heart.*—Cardiac stimulation may be regulated according to Rotch's dosage table:

Age	Tr. Digitalis	Strychnine	1% Sol. Nitroglycerine	Atropine
3 months	$\frac{1}{10}$–$\frac{1}{2}$ minim	$\frac{1}{2000}$–$\frac{1}{1000}$ grain	$\frac{1}{75}$–$\frac{1}{30}$ minim	$\frac{1}{3000}$–$\frac{1}{1500}$ grain
6 "	$\frac{1}{10}$–$\frac{3}{4}$ "	$\frac{1}{1500}$–$\frac{1}{500}$ "	$\frac{1}{25}$–$\frac{1}{15}$ "	$\frac{1}{2500}$–$\frac{1}{1000}$ "
9 "	$\frac{1}{4}$–1 "	$\frac{1}{500}$–$\frac{1}{300}$ "	$\frac{1}{25}$–$\frac{1}{10}$ "	$\frac{1}{1500}$–$\frac{1}{750}$ "
12 "	$\frac{1}{4}$–$1\frac{1}{2}$ "	$\frac{1}{500}$–$\frac{1}{250}$ "	$\frac{1}{25}$–$\frac{1}{5}$ "	$\frac{1}{1000}$–$\frac{1}{500}$ "
2 years	$\frac{1}{2}$–2 minims	$\frac{1}{500}$–$\frac{1}{150}$ "	$\frac{1}{15}$–$\frac{1}{3}$ "	$\frac{1}{750}$–$\frac{1}{250}$ "
3 "	$\frac{1}{2}$–3 "	$\frac{1}{300}$–$\frac{1}{100}$ "	$\frac{1}{10}$–$\frac{1}{2}$ "	$\frac{1}{500}$–$\frac{1}{200}$ "
4–10 "	1–5 "	$\frac{1}{200}$–$\frac{1}{60}$ "	$\frac{1}{2}$–$\frac{3}{4}$ "	$\frac{1}{250}$–$\frac{1}{150}$ "
10–12 "	3–8 "	$\frac{1}{100}$–$\frac{1}{40}$ "	$\frac{1}{2}$–1 "	$\frac{1}{200}$–$\frac{1}{100}$ "

9. *Lymph Glands.*—Secondary infection may occur, against which prophylactic care of the mouth and throat is important. Ice should be applied in the beginning only, and later hot boric-acid dressings when the blood- and lymph-streams become stagnant. Iodine and ichthyol salves and surgical intervention are indicated in the order named.

10. *Otitis.*—Otitis is treated by copious boric irrigation, given quite hot and at a slight elevation, and followed by equal parts of glycerine and laudanum, 5-per-cent. carbolic acid and 1-per-cent. cocaine in olive oil. Leeches over the mastoid often relieve the pain. Examination of the drum is rather impracticable in children with a small sensitive meatus. Early puncture under cocaine is much safer than awaiting spontaneous rupture. After rupture, a small amount of cotton with iodoform or boric powder should be applied.

11. *Nephritis.*—Nephritis is treated (*a*) according to the old prophylactic rule, by which all cases, severe or light, are confined to bed for at least four weeks. The urine must be watched. According to Jaccoud, the milk diet is valuable in the prevention of nephritis. Irritants, like cantharides, are always to be avoided. Tepid baths lasting a quarter of an hour should be given daily, especially in convalescence. Watch the temperature and the urine in convalescence. Wiodowitz in 102 cases used urotropin successfully as a prophylactic. The number of his cases is too small for generalization, but his results are at least suggestive. (*b*) Treatment of established nephritis. Hot baths increase the sweat and the flow of urine. They should be given twice daily, and may last for even an hour, but the patient must be kept afterward between blankets. Alkaline diuretics,

such as potas. acet., pot. bicarb., p. citrat., and infus. tritici repentis, may be given; pilocarpine for a child two years old, $\frac{1}{40}$ to $\frac{1}{0}$ gr., will often relax the skin. (See TREATMENT OF NEPHRITIS.) Calomel as a laxative and diuretic must be used with great care. Digitalis in moderate doses aids diuresis.

℞ Infusi digitalis ..... ℥v.
Potassii acetatis ..... ʒss.
Syrupi simplicis ..... ʒij.
Aquæ destillatæ .....q.s. ad ℥ij.
M. et S.—One teaspoonful every two hours.

High arterial tension should be treated with nitroglycerine. For convulsions, chloroform, chloral, and bromides are valuable. For vomiting, iodine is efficacious (see VOMITING, TYPHOID). Venesection and the administration of digitalis are necessary in severe pulmonary œdema. For marked uræmia, two drops of croton oil in milk or placed undiluted on the tongue, should produce free catharsis in two hours. If not successful by that time it should be repeated. Pulvis jalapæ compositus (gr. xv. to ʒj.) will unload the bowels in less urgent cases. Early evacuation is indicated in pleurisy, if chain cocci are found by puncture.

12. *Tonics.*—Such tonics as iron, arsenic, and cod-liver oil, are indicated in convalescence. The heart must be watched for weeks, because the myocardium is often degenerated.

13. *Serum.*—Marmoreck's and other streptococcic sera have given no definite results. Everything depends on whether streptococci are the primary cause or only secondary symbiotic organisms. Again, immunization against one variety of streptococcus does not produce immunity against other varieties. In Vienna, Moser's serum has apparently been efficacious. In the St. Anna Children's Hospital, the mortality was 8 per cent., and in other institutions it amounted to 14.5 per cent. without serotherapy.

MEASLES (MORBILLI, RUBEOLA).

Definition.—An acute, highly infectious disease, characterized by an initial coryza and inflammation of the air-passages, by a rapidly spreading eruption, a fever-curve with a double summit, and by toxæmia.

History.—Its first clear description dates from Rhazes (980–1037 A. D.). It is a very old disease and was widely epidemic in the eighteenth century.

Etiology.—(*a*) *Susceptibility* is almost universal; few individuals escape (14 per cent. only, Madsen), and one attack generally confers immunity. Maiselis collected 106 cases with a second attack; third and fourth attacks are known. (*b*) *Age*—It is rare during the first five months of life, and occurs most frequently after the first year. Twenty instances of placental transmission to the child are reported. It is more common in adults than is scarlet fever. In Schwalbe's

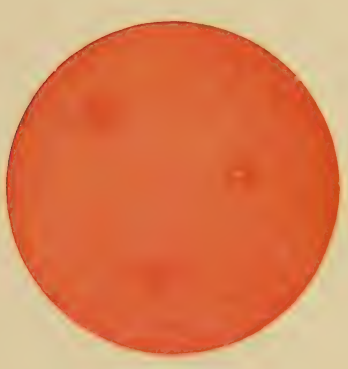
PLATE II

FIG. 1

FIG. 2

FIG. 3

FIG. 4



The Pathognomonic Sign of Measles (Koplik's Spots).

FIG. 1.—The discrete measles spots on the buccal or labial mucous membrane, showing the isolated rose-red spot, with the minute bluish-white centre, on the normally colored mucous membrane.

FIG. 2.—Shows the partially diffuse eruption on the mucous membrane of the cheeks and lips; patches of pale pink interspersed among rose-red patches, the latter showing numerous pale bluish-white spots.

FIG. 3.—The appearance of the buccal or labial mucous membrane when the measles spots completely coalesce and give a diffuse redness, with the myriads of bluish-white specks. The exanthem on the skin is at this time generally fully developed.

FIG. 4.—Aphthous stomatitis apt to be mistaken for measles spots. Mucous membrane normal in hue. Minute *yellow points* are surrounded by a red area. Always discrete.

collection of 16,981 cases, 50.4 per cent. occurred in the first five years; 42 per cent. between five and ten years; 5.2 per cent. between ten and twenty years, and 2.4 per cent. after twenty years of age. (*c*) *Season*—Measles occurs mostly in the winter and in the cooler spring months. (*d*) The actual *virus* is unknown. The longevity of the virus without the body is not great. It exists in the blood, tears, skin, and in the bronchial and nasal secretions. Its activity is greatest during the catarrhal stage, but it is also dangerous during the eruption, and possibly even during incubation and desquamation. Hektoen twice succeeded in inoculating the disease from the blood of measles patients.

Propagation usually occurs by personal contact, and rarely through a third person. Aerial transmission is frequent, whence in the home the disease usually runs through the family. The most frequent means of dissemination is the school-room. It is more often epidemic than is scarlatina, especially through the country. After smallpox in the unvaccinated, measles is the most infectious of diseases.

General Clinical Course.—The stages are: (1) Incubation of nine or ten days; (2) initial, catarrhal, or prodromal stage of three or four days, with fever, catarrhal symptoms in the eyes, nose, etc., and an enanthem; (3) the eruptive stage, lasting four or five days, with renewed fever, intoxication, eruption, and inflamed mucosæ; (4) the desquamative period of variable duration.

1. Incubation.—The incubation lasts nine or ten to fourteen days.

2. Catarrhal Symptoms.—Then the catarrhal symptoms appear. Chilly sensations, malaise and frontal headache attend redness of the eyes, lachrymation, photophobia, coryza, and sneezing; dryness, redness, and pain in the nose, throat, and bronchi, and early cough, which develops after one day. Vomiting may rarely occur. The respiratory symptoms are due to a mucous-membrane enanthem (Rehn). Examination of the throat reveals *Koplik's spots* (Plate II) in 86 to 97 per cent. of cases, described by him in 1886, and by Filatow in 1885. They are practically constant on the buccal mucosa near the molars, and are found on the first day. They are round, of a bluish-white color, and are surrounded by a reddish areola. Early catarrhal symptoms are more common in measles than in any other acute infection of childhood. The initial fever reaches 102° or 103°, and declines gradually during the third and fourth days. Occasionally there is a diffuse prodromal erythema which is mostly hyperæmic.

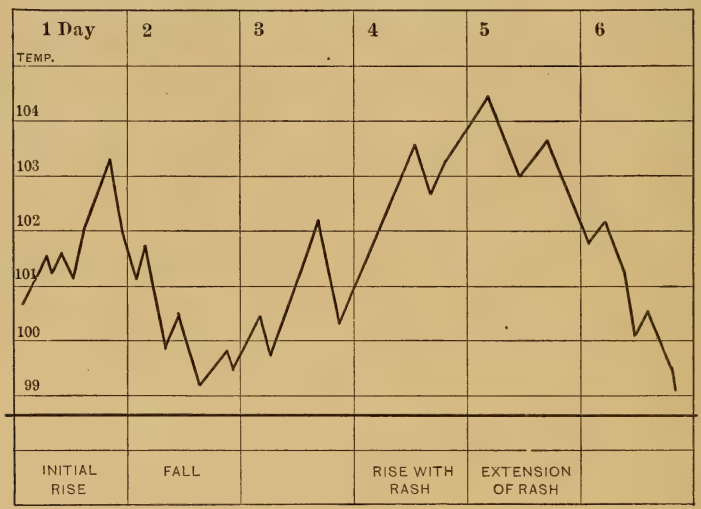
3. The Eruption.—The eruption begins on the fourth day with papules on the temples, cheeks, forehead, then on the back, wrists, forearms, and on the anterior surface of the body and legs, in order of progression as named; with the appearance of the eruption, the fever again rises. The eruption disappears on pressure or on lateral stretching of the skin, and is palpable and superficial. There are always islets of normal skin, even in the thickest eruption. The glands are usually enlarged and there is more or less marked sweating.

The general and local symptoms usually abate on the sixth day, and the fever falls by crisis. In severe cases, epistaxis, diarrhœa, cerebral intoxication, bronchiolitis, pneumonia, hypostasis, cyanosis, and asphyxia, may be observed, and death may result from lung involvement, mixed infection, or decreased physiological resistance.

4. Desquamation.—In the desquamative stage the skin pales, becomes slightly pigmented, and scales form. Convalescence is established in eight or ten days.

Individual Symptoms in Detail.—1. The Eruption.—It is essentially the same on the skin and on the mucosæ. In *the mouth* the mucosa is red on the first day of fever, and *Koplik's spots* are found; on the second day redness without swelling is especially noted on the tonsils and palate. The enanthem may reach the larynx, intestines, and genitalia. Though the enanthem tends to spread, the *skin erup-*

Fig. 9.



Fever curve in measles.

tion (*exanthem*) is circumscribed. The rash is due to hyperæmia rather than to actual exudation. It fades on pressure; in some cases the roseolæ do not fade on pressure, but leave a brown-colored area, especially in the nodular form of measles. The *roseolæ* usually are discrete, are slightly *elevated*, especially in the centre, from spastic contraction of the bloodvessels and œdema, are light to dark red in color, are *round or crescentic* in shape, and average 2 to 6 millimeters in diameter. They *always leave islets of normal skin* between the independent spots of eruption, even in the most diffuse types. The eruption follows a descending order and reaches the lower parts of the body last. When the eruption develops on the body first, the course is usually anomalous. Itching may be noted, and sweating may be marked. Desquamation follows the same order as the erup-

tion, and is usually fine. Scars never result. The eruption is rudimentary in those who are debilitated by chronic diseases, and is exuberant in the healthy.

The varieties are: (*a*) *Morbilli læves,* the usual form, with small elevations. (*b*) *M. vesiculosi seu miliares.* (*c*) *M. papulosi,* nodular measles; the eruption may feel slightly "shotty," but such induration is always superficial. (*d*) *M. confluentes seu conferti.* (*e*) *M. hemorrhagicæ,* benign or malignant (black measles; typhoid, septic, adynamic types); early hemorrhage into the eruption is almost always fatal. (*f*) *M. sine exanthemate; sine enanthemate.* "Recession" of the eruption (fading or "striking in") is usually a sign of heart-failure.

2. The Fever.—The curve is not absolutely typical. It begins suddenly without a chill, and falls on the second day, perhaps even to normal, usually rises again with the eruption, and reaches its maximum in thirty-six hours, thus giving *a curve with a double summit.* It usually falls by crisis before the eruption disappears. Fever, after the eruption begins to fade, indicates complications. In rare cases fever is absent. The temperature is normal, even subnormal during desquamation.

3. The Respiratory Tract.—*Nasal catarrh* is of special moment in sucklings. Catarrhal laryngitis may occur, accompanied by hoarseness and false croup; 50 per cent. of the cases suffer from erosions or ulcerations of the larynx (Rillet and Barthez), 16 per cent. from pseudomembranous laryngitis, and 15 per cent. from simple inflammation. Laryngeal complications are not necessarily febrile. *Bronchial catarrh* cannot be excluded by the absence of rales. It may reach the smaller tubules and usually, but not always, occurs late in the disease. It produces a clear, stringy, viscid sputum which later may become purulent and nummular. The bronchial glands are always swollen. Simple atelectasis may occur, and may occasion indeterminate breathing and dulness, which decrease with change of posture or deep breathing. *Bronchopneumonia* occurs in 6 to 12 per cent. of cases and *is the most frequent fatal complication;* Henoch and others affirm that patches of bronchopneumonia occur in every fatal case. It differs from other lobular pneumonias in its rapid development, and its greater tendency to pus formation, which result from the fact that the measles-catarrh favors pneumococcic and pyogenic infection. The temperature is often no higher than in bronchitis. Bronchial breathing and other physical signs are not always detected clinically. Toxæmia and carbon-dioxide narcosis accentuate the patient's distress. Bronchopneumonia is rapid in development, but slow or sometimes incomplete in resolution. It may result in interstitial pneumonia, gangrene, or *tuberculosis.* The latter is a dreaded sequel of measles and occurs (*a*) as a miliary invasion, and in the brain more frequently than in the lungs. It is caused by the bacilli, from old caseated bronchial glands, which are released by solvent substances reaching them from the lungs by way

of the lymph-vessels. It also occurs (*b*) as a fresh tuberculosis developing upon a bronchopneumonia. This variety may heal or apparently recover, to break out anew at a later time. Croupous pneumonia is as infrequent as pleurisy. Pleurisy is in most cases secondary to lobular pneumonic foci and may cause effusion.

4. Cardiac Symptoms.—These are rare. Accidental bruits may be heard. Toxæmia may, in exceptional cases, cause myocardial degeneration.

5. The Digestive Tract.—The tongue is coated. There is thirst and aphthæ and stomatitis may be observed. As a result of an intestinal enanthem, diarrhœa, mucus in the stools, and, anatomically, swelling of Peyer's patches, have been described. The liver is seldom swollen.

6. The Nervous System.—Nervous symptoms, such as delirium, stupor, coma, or convulsions, may, in rare cases, result from severe toxæmia, even though the delicate nervous system of children is seldom involved in measles. Nervous symptoms may occur in protracted pneumonia, and doubt may exist as to whether they are caused by the measles, miliary tuberculosis, tuberculous meningitis, or carbon-dioxide narcosis. Paralyses, myelitis, muscular atrophy, polyneuritis, and polymyositis, are most infrequent.

7. Genito-Urinary Tract.—Acetonuria and albuminuria are frequent. Ehrlich's diazo reaction occurs in 80 to 100 per cent. of cases. Nephritis is very uncommon. It is most often due to complicating diphtheria, or tuberculosis. Gangrene of the genitalia has been observed.

8. Other Symptoms.—The spleen and lymph-nodes are but slightly swollen; bone and joint complications are rare; keratitis is infrequent; otitis media occurs in a small percentage of cases. It is said that streptococci in the conjunctivæ are much more frequent in severe than in mild cases. The red cells are little altered, while the leukocytes show a relative lymphocytosis, with an absolute leukopenia.

Diagnosis.—The diagnosis depends upon the existence of an epidemic, and upon consideration of all the symptoms (coryza, cough, fever, enanthem) rather than of the eruption alone. Differentiation from *typhus* is made by the greater general disturbance in typhus, its more sudden onset, higher fever, swelling of the spleen, and by the roseolous eruption becoming petechial.

Sepsis with a morbilliform exanthem, the early stage of *influenza, syphilis* (in which other secondary symptoms appear with the eruption, and *variola* (*q. v.*), may sometimes delay the early diagnosis. In the *drug-eruptions* of turpentine, iodides, quinine, chloral, cubebs, copaiba, and antipyrin, the diagnosis may be most difficult. The fever, Koplik's spots, catarrh, and central elevations in the eruption, are indicative of measles. (See page 97.)

Prognosis.—The mortality is very great among aboriginal races. In private practice 3 per cent. is the average death-rate. In the Fiji Islands the death-rate was very high. In the Samoan Islands 4,000

DIFFERENTIATION FROM GERMAN MEASLES AND SCARLATINA.

GERMAN MEASLES	SCARLATINA	MEASLES
Incubation; 14 to 21 days.	2 (1 to 7) days.	9 to 10 days.
Prodromes; absent, short, or light.	Brief, a few hours to 1 or 1½ days.	3 to 4 days, generally markedly catarrhal.
Invasion; very mild.	Sudden, stormy, vomiting, convulsions, angina, and adenitis.	Catarrhal symptoms.
Exanthem; the rash is the first symptom, usually appearing on the first or second day on the face; as small red dots, rosy and brighter than in measles; small, little elevated, and thick about the mouth. They do not fuse. Less crescentic than in measles.	Appears on first or second day, reaching a maximum on second to fourth day, first below clavicles, involving face later; dusky-red, diffuse, burning, leaving mouth and eyes free.	Occurs on fourth day, greatest on fifth or sixth day on the face, forehead, and about the mouth; brick-red, elevated papules or crescentic islets of eruption, always with areas of healthy skin between.
Enanthem, etc.; may be diffuse redness of throat and conjunctivæ.	Angina proportionate to skin eruption; dusky-red; tonsillar plugs; conjunctivæ, bronchi, intestines usually intact.	Koplik's spots; patchy redness of throat; photophobia; conjunctivitis, marked bronchitis, very often bronchopneumonia; diarrhœa in some cases.
Glands; usually enlarged, hard, tender, especially posterior cervical, also axillary and inguinal. Spleen somewhat enlarged.	Proportionate to angina, hence largely cervical. Enlarged spleen.	Mostly at angle of jaw; rarely systematic enlargement. No enlargement of spleen.
Constitutional symptoms; few; no depression; no leukocytosis; little pulse increase and fever.	Much depression with rash, coated tongue with peeling on the fourth day (strawberry tongue); fever 105° to 106°; pulse increased more than fever; lysis on seventh day; leukocytosis.	Depression, coated tongue, anorexia; fever-curve with two elevations; crisis on seventh day; no leukocytosis.
Complications; practically none.	Nephritis, arthritis, endocarditis, sepsis, periadenitis.	Catarrhal pneumonia, pleurisy, tuberculosis.
Convalescence; rapid; desquamation rarely copious, and always fine.	Prolonged, tardy; desquamation copious and usually in shreds (hands and feet).	Often prolonged; desquamation rarely copious and always fine.

deaths followed the introduction of measles in 1893. The usual important factors are: (*a*) The *genius epidemicus:* Henoch records 30 per cent. and Heubner 37 per cent. in certain epidemics. The

mortality in a German epidemic of 2,881 cases was only 1 per cent. (Heissler). (*b*) *Individual resistance:* It is higher in institutional practice and in the army. It is 16 per cent. in the children's hospitals in Paris. Measles ranks third among the exanthemata in mortality. (*c*) *Age:* The death-rate is highest at the second year; 55 per cent. of fatal cases occur in the first two years of life (Henoch). (*d*) *Complications* are more important than the disease itself, *e. g.*, tuberculosis and bronchopneumonia, which is the worst complication, with a mortality of 33 per cent.

Treatment.—1. Prophylaxis.—Prevention is important, especially in children under two years, in severe epidemics with pneumonia, in the weakly, and in cases of pertussis. Though the mortality and complications are greater in adults, it is of no advantage for the young to contract measles, and the too prevalent practice of allowing the disease to run through the family must be condemned. In all cases, the secretions from the nose, throat, and bronchi, must be disinfected, and general sanitary care should be exercised as in smallpox (*q. v.*) and scarlatina.

2. Hygiene.—The patient should have fresh air, warm quarters, rest in bed until after desquamation, and a liquid diet. A mustard bath may be employed in order to bring out the rash.

3. Catarrhal Symptoms.—Photophobia does not necessitate a dark room. Some sunlight should be admitted. A boric-acid solution should be dropped frequently into the eyes and vaseline applied to the lids. Prophylactic care of the mouth is necessary. To relieve pain in the ears, belladonna, opium, and a 1-per-cent. cocaine solution may be used; paracentesis is indicated for gathering. The nose should be douched with Dobell's solution. Tr. belladonnæ, ♏i.–iii. may be given every two hours for coryza until flushing of the face, increase of pulse-rate, or a dry, red throat, develops.

4. Antipyresis.—Antipyretics are indicated in sudden high elevation with severe cerebral toxæmia, and in high fever with diffuse bronchitis or pneumonia. They are given most efficaciously in the form of baths, with cold affusions to the head and neck. Wine, leeches to the mastoids, mustard plasters, and laxatives, may also be indicated in toxæmia.

5. The Circulation.—The heart rarely requires treatment, except in septic types or in pulmonary complications, when camphor, aromatic spirits of ammonia, strychnia, wine, or coffee, is indicated. (See dosage table, page 91.)

6. Respiratory Symptoms.—Care of the mouth, a soft or liquid diet, inhalations of steam, hot fomentations over the larynx, warm baths, emetics (such as apomorphine or ipecac), and possibly intubation or tracheotomy, are indicated for laryngeal complications. Bronchitis should be treated with ipecac, codeine (morphine), emetics, inhalations of steam, massive hot packs to the chest, and cold affusions over the nape of the neck (see Acute Bronchitis); stimulation for pneumonia (*q. v.*) may be indicated.

7. Alimentary Tract.—Enemata relieve constipation. When diarrhœa is excessive, opium, hot wine, or cognac, demulcents, hot applications, and physiological salt-solution by rectum, are indicated.

8. The Skin.—The skin should be rubbed with olive oil; a 2-per-cent. menthol salve, or 1-per-cent. carbolic solution, relieves itching.

9. Complications.—The treatment of complications, such as tuberculosis, pertussis, etc., will be described under these topics.

RUBELLA.

Synonyms.—Rubeola notha, German measles, Rötheln. By the Germans it is called rubeola, though we employ rubeola as synonymous with measles.

History. Definition.—Rubella was first described by Bergen (1752) in Germany, and its existence as an independent infection was established by Maton (1815) in England. It is more often confused with atypical scarlatina than with atypical measles. One attack confers immunity.

Etiology.—Its etiology is not known. Most cases occur during the first half of the year. The disease most frequently attacks children.

Symptoms.—1. The Incubation.—The incubation lasts from ten to twelve (perhaps fourteen to twenty-one) days.

2. Prodromes.—Prodromes are usually lacking; for a period of two hours to a day, there may be cough, photophobia, sneezing, slight temperature, chilliness or headache.

3. The Eruption.—The eruption occurs on the first day, usually in the palate and throat. It resembles measles and consists of hyperæmic punctate red spots (Forchheimer's spots). The skin is red, and the eruption disappears at first under pressure; then the macules become darker and more circumscribed; later, the eruption does not entirely disappear under pressure. There are two forms, the circumscribed and the diffuse. The eruption becomes slightly elevated, does not fuse as it does in measles, and erythema is not present about the points of eruption as it is in scarlatina. Anatomically, it is a capillary hyperæmia of the papillary and upper layers of the corium, accompanied by slight inflammation and exudation between the corium and epidermis. It advances in "jumps," and fades in one place while it blossoms in another. It appears first on the scalp and face, and progresses downward to the neck, body, arms, and lastly to the legs and feet. Unlike scarlatina, spots are aggregated about the mouth. The eruption is greatest on the upper parts of the body, and is increased by constriction or by pressure of the clothing. Its average duration is from two to four days. The papillæ of the tongue show slightly through the fur. Desquamation is minimal and fine. Tracheitis, or, rarely, bronchitis, may be observed. Almost characteristic is the swelling of the posterior cervical and post-auricular glands. There may be some angina, dysphagia, or anorexia.

4. The Constitutional Symptoms.—The constitutional symptoms are few. The fever is usually slight. Its duration averages

three days, and it falls by crisis or lysis when the eruption has matured. Except in a few American epidemics, urgent nervous and other complications, such as icterus, albuminuria, nephritis, or colitis, have rarely been observed.

Diagnosis.—The diagnosis is often very difficult and the unity of the disease is sometimes questioned (see MEASLES). The *"fourth disease,"* described by Dukes, cannot be admitted to our nosology. *Erythema infectiosum,* emphasized particularly by Escherich, is a weakly contagious, maculo-papular erythema, prevalent particularly in the spring and fall, and occurring chiefly on the extensor surface of the extremities, and rarely on the trunk.

Prognosis.—The prognosis is good, and *therapy* is symptomatic. Isolation is theoretically advisable.

ERYSIPELAS.

Synonym.—St. Anthony's Fire.

Definition.—An acute, febrile, contagious disease caused by Fehleisen's *Streptococcus erysipelatos.* It is characterized by a peculiar circumscribed inflammation of the skin and adjacent mucosæ, which tends to rapid and broad extension. It usually runs a self-limited course, with slight, if any, visceral involvement.

History.—Erysipelas was known to Hippocrates and Galen. Trousseau (1848), in France, maintained that it was always a wound infection. Werner, in England, maintained that it was an infectious disease, and Koch (1880) found the streptococcus, which was more fully described by Fehleisen (1881) and bears his name.

Etiology.—It is wide-spread, and sporadic cases are seen in almost every centre of population. It occasionally breaks out epidemically. It prevails largely in the early spring months; 60 per cent. of cases occur between January and May (Anders). In the preantiseptic era it was the dread of hospital physicians, and even at this day it occasionally visits the most sanitary institutions. It is conveyed by a third person and by fomites; and, infrequently, by unclean instruments, and vaccine. Cases of placental transmission to the fœtus are known. The virus is not particularly penetrating but may adhere to clothing and furniture with great tenacity. Individual predisposition varies, but many persons have, apparently, a natural, or perhaps an hereditary, susceptibility to the disease. It is like rheumatism or acute angina, in that it often recurs repeatedly. It occurs mostly in the female sex (73 per cent. of cases). Alcoholism, debility, and chronic nephritis, are predisposing factors. Any distinction between "idiopathic" and *"wound" erysipelas* is artificial, for in practically every instance, some abrasion, though perhaps too small for the eye to detect, occurs in the skin or mucosæ.

Bacteriology.—The Streptococcus erysipelatos (seu pyogenes longus) was found in the lymph-vessels by Fehleisen, who first drew the distinction between erysipelas and phlegmon. Its absolute differ-

entiation from the Streptococcus pyogenes is impracticable. Inoculations from cases of erysipelas sometimes produce suppuration. Pus from abscesses has caused erysipelas, and, in certain cases, erysipelas and suppuration have developed simultaneously. This has given some weight to the theory of the bacteriological unity of erysipelas and suppuration.

Symptoms.—1. Incubation.—Incubation lasts from three to seven days (fifteen to seventy-two hours in direct inoculations).

2. The Eruptive Stage.—(*a*) Usually preceded by a few hours of general discomfort, this stage begins with *vomiting, fever, chill, and the eruption.* The fever rises rapidly to 103°. The chill occurs in 75 per cent. of cases and is chiefly absent in erysipelas secondary to other diseases. (*b*) The *eruption* first appears on the face (95 per cent. of cases), near the inner canthus of the eye, near the nose, or on the cheek. It consists of a hard, bright-red, slightly elevated, burning, and painful area of inflammation, which pits but little on pressure, though its color may disappear. Pathologically, erysipelas is simple inflammation; in and under the skin are found œdema, hyperæmia, white- and red-cell extravasation, and *streptococci* which advance *in the lymph-clefts,* and are most abundant in the spreading edge, but also in the lymph-vessels beyond it. *Its elevated red edge* is sharply marked, or *"wall-like."* As the area spreads, the *skin becomes much swollen, œdematous, smooth, tense, red, and hot,* causing a shapeless swelling of the eyes, nose, lips, and ears, and obliterating the facial lines and expression. The extent of this area is indicated by tenderness, and is therefore more accurately measured with the finger than with the eye. The *skin blisters* and under the lens, small vesicles are always found. The *prong-like advance of the eruption* is similar to that of spilt alcohol, or, as Volkmann puts it, like burning paper; *i. e.,* it disappears behind as it advances forward. Its extension is irregular, because the streptococci follow the lines of least resistance. If obstructed by dense tissue, deep attachments, or the so-called ligamenta cutis, its course becomes circuitous, asymmetrical, and tardy in one area though rapid in another. It is limited at the base of the skull, and rarely affects the chin, or passes over it to the opposite side. The neck resists its advance, often successfully, but erysipelas may course over the entire body. The sternum, condyles, ilium, and Poupart's ligament, check or divert its progress. When apparently obstructed, new islets of eruption appear near by, which are nearly always consecutive, as may be seen by the faint lymphatic involvement between the foci. The scalp is often unaffected, but if invaded it becomes swollen, tense, and pale or blue rather than red, and the hair falls out. The genitalia may swell enormously. Necrosis of these tissues, and also of the elbow and knee, may result from excessive tension. The first area pales, and a coarse desquamation results, while the eruption at more remote points advances. The lymph-glands are almost constantly swollen, tender, and palpable, unless hidden by œdema. They enlarge proportionately

to the dermatitis. The eruption presents few variations: *Erysipelas miliare* (*vesiculosum*); less frequently *erysipelas bullosum; erysipelas squamosum,* or, in old and cachectic subjects, *erysipelas hemorrhagicum.* (*c*) The *fever* rises suddenly, and is highest on the third or fourth day (103° to 104° or higher). It is continuous, then becomes remittent or irregular, and finally, after seven to nine days, resolves by crisis, or less often by lysis. Fever is almost always present. In attenuated erysipelas there is little fever and there are few general symptoms, though the rash may be well developed. In general the intensity and the progress of the infection are marked by the degree of fever.

Other General Symptoms.—The pulse is soft, and ranges between 100 and 120; albuminuria occurs in 38 to 67 per cent. of cases; moderate leukocytosis is the rule, and there may be some pain and swelling of the liver and spleen. Nervous toxæmia, with the exception of headache, is uncommon.

Course.—When limited to the face and head, eight to ten days is the usual duration, and a course longer than two weeks is uncommon. *Erysipelas migrans* may last weeks or months, travel from limb to limb, and recur again and again. In one of the author's cases it lasted six months.

Complications and Sequels.—1. Nervous.—Toxæmic symptoms are infrequent except in aged, debilitated, or alcoholic subjects. Delirium is a toxæmic unfavorable development and occurs especially when the scalp is invaded. A profound typhoid condition may ensue in debilitated, or alcoholic patients. Meningitis, formerly confused with toxæmia, infrequently follows orbital cellulitis or extension along the fifth nerve. Post-febrile psychoses, multiple neuritis, degeneration of the cord, and eye inflammation are exceptional.

2. Circulatory.—Endocarditis, pericarditis, myocarditis, acute dilatation, and arterial or venous thrombosis, are infrequent. Leukocytosis is usually moderate, and is parallel with the fever. High counts indicate concomitant suppuration. Streptococci in the blood are usually ominous.

3. Respiratory.—Erysipelatous coryza, otitis, and stomatitis, are infrequent and are difficult of recognition. Erysipelatous angina is characterized by much pain, sharply marked redness or lividity, a glazed varnished surface, marked œdema of the uvula and epiglottis, and the formation of vesicles or bullæ. Sometimes a wall-like margin is seen. Gangrene may develop in severe instances. The cervical glands may be swollen. For the recognition of erysipelatous angina, coincident facial erysipelas is necessary. Laryngeal erysipelas is most often secondary to pharyngeal erysipelas. It is rare, severe, is easily or necessarily confused with glottis œdema, and is marked by great turgescence, rapid spreading and swelling, and high, early fever. Without tracheotomy, fatal stenosis and extension to the lower air-passages may occur. The rare so-called "erysipelas pneumonia" is especially described by French writers. Though any infection

may travel downward from the throat, it is more probably a septic localization than "internal" erysipelas. In streptococcus pneumonia, the spleen is enlarged (sepsis) more frequently than it is in ordinary pneumonia. The advance is rapid, the extension is wide, the gray hepatization is great, and fibrin formation is scant or lacking. Nevertheless, in pneumococcus infection the same "wandering pneumonia" may be seen. Pleurisy is usually metastatic (septic).

4. Alimentary.—Vomiting is often cerebral. Exceptional complications are parotitis, œsophagitis, gastritis, enteritis, intestinal ulceration, hemorrhage, pain, icterus, and pylethrombosis.

5. Genito-Urinary.—Albuminuria (67 per cent.) is the most constant visceral finding. Nephritis (4 per cent.) is often of the acute glomerular type. Streptococci have been found in the urine and kidneys, especially in septic types. Typical erysipelas may begin in the external genitalia, especially in *puerperæ,* or may first infect the internal mucosa which has been abraded during labor. (See Septic Affections.)

6. Septic.—Subcutaneous small *abscesses* which resemble cold abscesses appear on the head or body in 6 per cent. of cases, especially in alcoholic, nephritic, diabetic, or debilitated patients. Phlegmons may develop, usually on the legs, and even without mixed infection. Gangrene is fortunately rare (1 of 1 per cent.). It may develop early in the eye-lids, genitalia, or over the joints, or it may develop later in the extremities. Herpes is not infrequent. Baldness may also result. The joints may be painful or swollen; suppuration is metastatic. Suppurative adenitis may occur with or without mixed infection. General fatal sepsis, aside from the puerperal form, is infrequent. It bears the same relation to local erysipelas as does miliary tuberculosis to a local tuberculous focus (Ponfick).

Diagnosis.—Diagnosis is easy, when the area is the size of a dollar, (*a*) because of its very location; (*b*) because of the early involvement of the lymphatics and of the lachrymal sac; (*c*) because in most cases the point of entrance can be found; it may have healed; it may be concealed by the swelling, or lie an inch or more away from the first eruption; (*d*) because of the redness, smoothness, swelling, and œdema; (*e*) because of the elevated, wall-like margin; and (*f*) because of the prong-like, irregular progression.

In *weeping eczema,* the edges are not raised, the skin is not smooth, there is less fever, the development is slower and itching is present. *Malignant pustule* may cause redness and swelling, but anthrax bacilli are found under the primary papule and crust. *Dermatitis venenata,* or *medicamentosa,* and pemphigus, are unlike erysipelas. Erythema nodosum is discrete, though diffuse, and does not spread. Diffuse simple erythema is a febrile affection, and of short duration. *Dermatitis exfoliativa* (Ritter) in the new-born is afebrile, develops first about the mouth and chin, and reaches the body and limbs later. Its diffuseness and redness are characteristic. In *lymphangitis* the swelling of the lymphatics advances toward the centre and

presents cord-like or net-like areas, which are not confluent, vesiculated, uniformly red, or evenly edged. In *phlegmon* and deep *cellulitis,* the process is deeper and harder, and the blisters and margin of erysipelas are lacking.

Erysipelatous angina may not be diagnosed without coincident facial erysipelas. The local findings are not pathognomonic; neither are the general findings, such as high fever, albuminuria, nephritis and splenic tumor.

Erysipeloid, a zoönotic affection described first by Rosenbach, occurs especially in persons who handle meat. It is recognized by the "butterfly" eruption which develops on both cheeks or on the finger-ends, and is unaccompanied by fever or general symptoms. The edge of the eruption is clear-cut, its advancement slow, its color a dark livid-red, and its course lasts one to three weeks with spontaneous recovery. Gilchrist (1904) reported 329 cases, of which 323 were caused by crab-bites.

Prognosis.—(*a*) In uncomplicated *facial erysipelas* the outlook is good. In general the mortality is 3 to 4 per cent. in private practice, and double that figure in hospitals. (*b*) In *erysipelas migrans* the outlook is less favorable. (*c*) In *erysipelas neonatorum* (Trousseau, Lorain) the outlook is almost invariably fatal on the fifth to seventh day. It begins on the navel, and less often on the vulva, and on circumcision-wounds. Vomiting, toxæmic diarrhœa, peritonitis, icterus, pneumonia, necrosis, gangrene, suppuration, and sepsis, are much more common than in adult forms. Fortunately, like puerperal fever, this form is now infrequent. (*d*) *Erysipelas secondary* to other infections is serious, especially in chronic maladies, such as alcoholism, diabetes, nephritis, and tuberculosis, and also in bed-ridden, or senile subjects, in whom the asthenic, hemorrhagic, or gangrenous types are chiefly observed. In acute diseases such as typhoid, diphtheria, and pneumonia, this complication is dreaded. (*e*) Puerperal forms or involvement of the mucosa, especially that of the larynx, are unfavorable (Hippocrates). (*f*) *Recurrence* is more frequent than in any other infection, which shows that no permanent immunity is conferred. Relapses (10 to 15 per cent.) are attacks recurring within a few days. Recurrences (20 to 50 per cent.), properly speaking, return after months or years; indeed, in some individuals, erysipelas may be called habitual. It is observed most frequently in the young and in women (75 per cent.), and mostly on the face. It may result in elephantiasis. Five to fifteen attacks are not uncommon, and Roger has seen forty in one person.

Treatment.—1. Prophylaxis.—In this bacteriological era, the disease has been decreased by means of asepsis and isolation, though in regard to the latter, many physicians are still remiss. Chronic rhinitis, rhagades, decayed teeth, and neglected ulcers, must be considered in cases of habitual erysipelas. (See Smallpox; disinfection.)

2. Mechanical Means.—Mechanical efforts to limit extension of

the disease are still used, but unsuccessfully. These consist of binding with elastic strips, elastic constriction, or heavy applications of collodion.

3. Local Applications.—Iodine, creolin, bichloride of mercury, carbolic acid, alcohol, iodoform, boric acid, and numerous other topical applications, may be recommended but the water they contain is the best ingredient, and was recommended by Hippocrates himself. Mechanical and local applications cannot reach the deeply-situated cocci. Cold compresses afford a sure relief for pain and tension. Ichthyol ointment, 10 to 20 per cent., is soothing but in no sense curative.

℞ Creolin ..... 1 part.
Iodoformi ..... 4 parts.
Adipis lanæ hydrosi (Lanolin) ..... 10 parts.
M. f. ung. (Koch.)

4. Drugs.—Erysipelas is a self-limited disease, therapeutically unapproachable. Tincture of the chloride of iron (Hamilton Bell, 1851) is well tolerated in large doses (♏ x–xxx) but neither shortens nor mitigates the disease.

℞ Tincturæ ferri chloridi ..... ʒiiss.
Quininæ sulphatis ..... gr. xxiv.
Spiritus chloroformi ..... ʒij.
Aquæ .....q.s. ad ʒvj.
M. f. mist.
S.—Two tablespoonfuls every three or four hours. (J. Burney Yeo.)

5. General Treatment.—The symptoms are managed expectantly,—*i. e.,* as they arise. Headache, delirium, pain, vomiting, and cardiac weakness, are treated as in typhoid. *Rest in bed* and as *generous nourishment* as the stomach will tolerate, are indicated.

6. Marmorek's Serum.—Chantemesse reduced the death-rate of his cases to 2½ per cent. by serum treatment, a figure often reached by more simple measures (Roger and Botognesi).

Erysipelas as a *curative agent* (*erysipelas salutare*): Permanent cure of lupus has followed a complicating erysipelas. Syphilis quickly subsides after this infection, and its beneficial effects have been observed in leukæmia, Hodgkin's disease, carcinoma, sarcoma, chronic arthritis, tuberculous adenitis, diphtheria, and tuberculosis.

MALARIA.

Definition.—An acute infection caused by the Plasmodium malariæ, inoculated in man by the mosquito and characterized clinically by (1) paroxysms of intermittent, quartan, tertian, or quotidian fever, or by (2) remittent or subcontinuous fever, and sometimes by (3) pernicious, or (4) chronic cachectic forms.

History.—Malaria, known to Hippocrates and Celsus, is the most completely investigated of all diseases, in that (*a*) its cause is exactly known; (*b*) its prophylaxis has become known since the discovery of

infection by the mosquito; (*c*) the disease can be watched in different stages in the blood; and (*d*) there is a truly specific treatment.

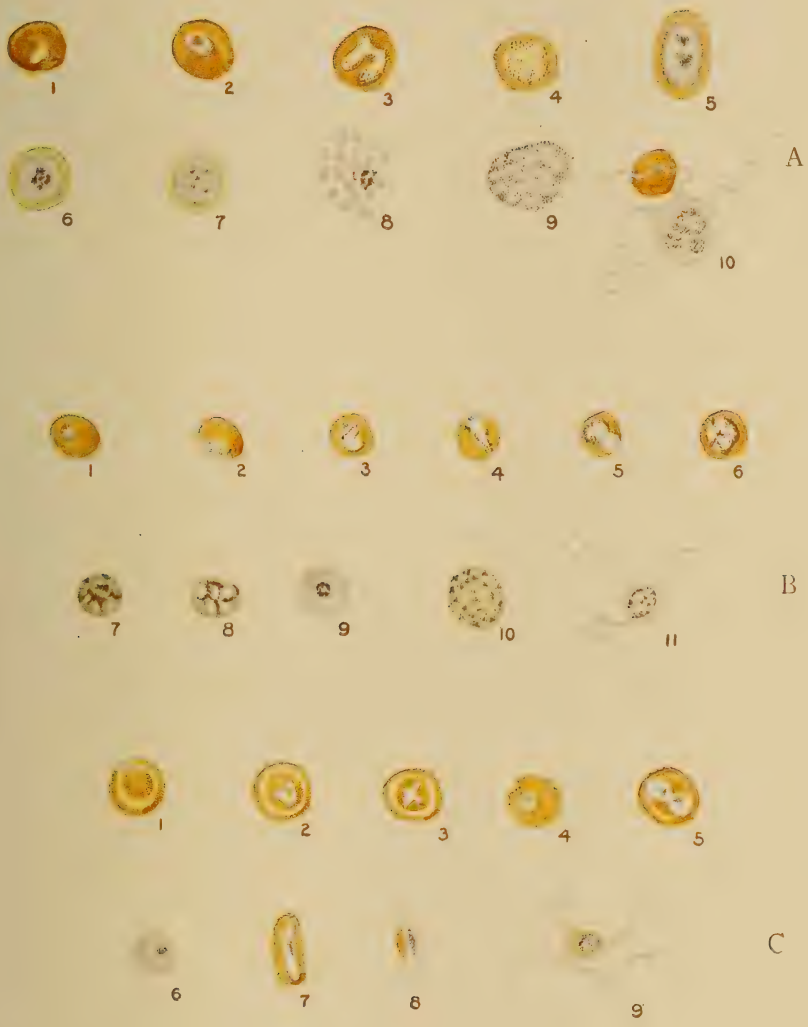
Etiology.—All ages and both sexes are affected equally. Men are more exposed to infection, and children are readily infected on account of their thin skin. Such occupations as sewer and railroad building, ditching, and night work, especially, are predisposing factors; 32 per cent. of the French soldiers in Madagascar in 1895 acquired malaria; reduced physiological resistance, exposure, excesses, and alcoholism, also predispose to the disease. Negroes have a relative immunity. Instances of familial or individual immunity have been noted. Epidemics may occur, as happened during the building of the Paris boulevards. Indirect causes, such as warmth of climate, the warm season, moisture, rain, swampy localities, telluric influences, bad air (mal-aria), and impure drinking-water, are now proved to be *causes* only in so far as they *relate to the breeding of the mosquito.* In Africa, severe forms of malaria are found everywhere except in its lower part and in the Sahara. In the far East its chief seats are India, Burma, Assam, Faral, and the East Asiatic coast; Asia Minor, Japan, and the East Indies. In Europe, lower Russia, Italy, Hungary, the Balkans, and Greece, are most affected. Malaria also abounds in the northern three-fourths of South America. In the West Indies, severe types prevail. In the United States, malaria is slowly moving southward, and cases seldom develop on the Pacific coast, in the Northwest, in the dry West and in the Lake region. Even in the South it is becoming less frequent.

Direct Cause.—The direct cause is the malarial parasite, described by Laveran, in 1880, and also by Marchiafava and Celli, in 1885. In this country Councilman, Abbott, Osler, James, Dock, Koplik, Thayer, Hewetson, and others have studied the organism. It is a red-cell parasite, a hæmocytozoön. The biology of the *Plasmodium malariæ* concerns (*A*) its life in the human blood, and (*B*) its life in the mosquito. The unity of all species was maintained by Laveran, but various species must be recognized because (1) their morphology varies; (2) their geography, seasonal prevalence, and malignancy vary; (3) on inoculation, the species and fever type are maintained; and (4) the type remains the same in the same individual.

(*A*). The Plasmodium in Man.—Special forms of the parasite:

1. The *quartan parasite* has a life-cycle of three days. In the first twelve hours the young form is without pigment. A small amorphous bright hyaline spot lies in the red cell, whose form flattens the parasite. There is some amœboid movement upon warming the slide. It gains little in size for one day, when some pigment or melanin appears in the form of coarse dark rods and granules with little movement. Melanin is the hæmoglobin digested by the parasite. It is usually dark or black and resembles the melanin of malignant growths. It is dissolved by alkalies and ammonium sulphide, but not by acids. Movement of the parasite ceases as pigment increases. The parasite occupies one-half to two-thirds of the red

PLATE III



Malarial Parasites.

A. Tertian parasite. 1, young form; 2-4, stages of growth and pigmentation; 5-7, segmentation; 8, rupture of spores from corpuscle; 9, extracellular form; 10, flagellate form.

B. Quartan parasite. 1-6, development and pigmentation; 7, 8, central grouping of pigment and spore formation; 9, rosette of spores with nuclei; 10, extracellular form; 11, flagellate form.

C. Æstivo-autumnal parasite. 1, ring-like body; 2, 3, amœboid forms; 4, parasite in brassy degenerated red cell; 5, pigmentation; 6, segmentation; 7, 8, half-moon bodies; 9, flagellate forms.

cell after forty-eight hours, and almost the entire cell after sixty hours. There is rarely more than one parasite in each cell. Flagella (*v.i.*) are not frequent. During growth the red cell on which the plasmodium feeds either undergoes no change in size or becomes smaller and darker peripherally. As sporulation occurs, the pigment gathers centrally and the spores, usually not numbering more than ten, develop into the "daisy" form. Vacuoles are seen in some cases. After sixty-nine hours, mature segments finally separate, each with a bright spot or nucleolus; *i. e.,* segmentation occurs in the peripheral blood three hours before the paroxysm, which corresponds with their rupture from the red corpuscle into the plasma. Sporulation means death of the adult parasite, or its "death in childbed," as Mannaberg well describes it. The spores rupture through the red cell, and escape into the plasma, whence they attack and enter other red cells, to repeat their life cycle. The pigment and fragments of red cells are picked up by the leukocytes and are carried to the liver and spleen. Some forms (gametocytes) do not mature nor sporulate. They are taken up by the mosquito, to complete in it another cycle. (Plate III.)

2. The *tertian organism* has a forty-eight-hour cycle. In its first day it is small (1μ to 2μ), and appears as a bright, unpigmented, disklike or annular spot in the red cell, thrusting out pseudopodia, with most active amœboid movement, and possessing a relatively large nucleus,—partly clear chromatin, and partly chromatin deeply stained by the Romanowsky method. It fills about one-third of the cell. In its second day, pigment rapidly accumulates as fine granules or rods, which are usually peripherally located, and have an actively swarming movement, compared by Laveran to that of boiling water. This is due to currents in the protoplasm of the parasite. The amœboid movement decreases in twenty-four to thirty-six hours, as the parasite grows and as the pigment increases, but some movement remains for a long time. Vacuoles are frequent. The parasite occupies two-thirds to three-fourths of the red cell, which becomes distended and chlorotic. The most common form of the sporulation is the concentric accumulation of pigment and the development of fifteen to twenty rough, refractile spores, which form in two concentric circles ("sunflower" arrangement), or are more frequently irregular, like a bunch of grapes. The spores are small and round. Sporulation occurs three hours before the chill and the rise of temperature, and is completed in forty-eight hours, when the toxins secreted produce the rigor and fever. Most spores are seen at the time of the chill, and even then few can be seen, because sporulation occurs to a greater extent in the internal organs than in the peripheral blood-stream. When spores are seen between paroxysms they represent the sporulation of another generation of parasites, too weak or too few to induce a paroxysm. The generations are never absolute as they are in the quartan type, and the parasites of the same generation vary six or eight hours in their cycle. This explains the

clinical variations in periodicity. Flagellated forms may be seen a few minutes after withdrawal of the blood. The nodulated and clubbed flagella lash about and indent the red cells. They may break off and swim about. They occur in numerous parasites which never sporulate in human blood, and remain as large bodies with vibrating pigment. Formerly regarded as degenerated parasites, they really represent gametocytes or the "sexual forms," which develop later in the mosquito (MacCallum, 1897).

3. The *æstivo-autumnal parasite* has a life-cycle of from twenty-four to forty-eight hours. It has a very small, unpigmented body, and possesses most active amœboid movement. It thus attracts early attention but is easily overlooked, as it possesses almost the same refraction as the red cell. It becomes quiet after drawing the blood from the body, when it appears characteristically ring-like, often with a central vacuole. The shape may be successively amœboid, diskoid, annular, cruciform, or often may have a slight protuberance at one side—the seal-ring form. Mature forms contain particles of hæmoglobin. The pigment is extremely fine, powder-like, dark, sometimes reddish, peripherally located, and has some movement. When the parasite reaches one-third the size of the corpuscle, it is usually motionless, and the pigment usually becomes concentrated centrally. Motion in the parasite has been seen in the cadaver by Ziemann. The spores number six to sixteen, are very small, round, and without movement or vacuoles. Sporulation occurs as in the other forms. Fever, pigment concentration, and sporulation, coincide (Golgi). If large parasites with much pigment are seen in the peripheral blood, they denote the onset of a paroxysm, and after the paroxysm, ring-forms without pigment may be observed. The young forms circulate in the general blood-stream, though sporulation usually occurs in the viscera. The *half-moon bodies,* or *crescents,* are delicate, highly refractile bodies, measuring eight to ten microns by two to three microns, are developed in the bone-marrow or spleen, and possess considerable pigment, which is usually mesially located and has little, if any, motion. They have no amœboid movement but may slowly change form. In shape they are spindle, oval, or spherical. They occur singly in the red cell. They do not multiply in the human blood. The young crescents develop in the viscera or bone, and their adult forms are seen in the peripheral blood, for the periphery offers the best opportunity for leaving the body—through the bites of mosquitos. The male form of the crescent has flagella (microgametes) which penetrate the sexual elements of the female crescents (macrogametes). This occurs outside of the body, either on the slide or in the body of the mosquito (Ross). After several days crescents, spherules, or spindles, appear. Crescents indicate that the fever has just begun. Fever rarely occurs when crescents alone are found in the blood. If fever develops with the presence of crescents, amœboid bodies, also, are nearly always found. The red blood-cell becomes copper-colored or brassy.

The parasite may be stained with methylene blue or hæmatoxylin, and counterstained with eosin.[1] For the beginner, at least, study of the fresh blood under an oil-immersion is the best method.

(*B*). The Life of the Organism Without the Human Body. —King (1883) first suggested that the *mosquito* was the intermediate host and sole source of infection. It takes the plasmodium from man and reinoculates him with it after the life-cycle within its body. Other analogous infections are known, such as the hæmosporidia of birds, frogs, bats, dogs, sheep, monkeys; *e. g.,* Texas cattle-fever inoculated by the tick, and the African tsetse disease (*trypanosoma*) in animals and man, which is inoculated by the tsetse-fly. Manson (1894), especially, then Ross, Grassi, Bastianelli, Bignami, Daniels, Stephens, Christopher, and others, have developed this most interesting subject. The Plasmodium malariæ has not been found except in man and in the mosquito. The mosquito is the primary and higher host, since the sexual process of reproduction in its stomach is biologically higher than the simple sporulation in man. The culex and the anopheles are the two chief genera. The culex does not contain nor convey the parasite. Its palpi are very short and its wings generally have no mottling. When sitting, its hind legs are carried above the body and its body lies parallel with the wall, or may sag from the blood it has sucked. Its eggs are laid in tanks or puddles. It is the ordinary house mosquito and is found chiefly in cities. The *anopheles conveys malaria.* Its palpi are almost as long as the proboscis; the wings are mottled; the rear legs usually depend, or touch the wall. Its body makes an angle of 45° with the wall. The eggs are laid in pools or sluggish streams with algæ growth, and the open country is its preferred habitat. In America, four species exist: (1) The most important is the Anopheles maculipennis; (2) A. crucians; (3) A. punctipennis; (4) A. argyrotarsis. When the anopheles bites persons whose blood contains sexually mature forms of the plasmodium (gametocytes), flagellation in the male form occurs and the flagella enter the female cells. After fecundation in the insect's stomach, the impregnated cell enters the muscular walls of the middle intestine, and, after two days, small, round, refractile, pigmented bodies appear, which, after a week, grow to 60 or 70μ in

[1] The Nocht-Romanowsky staining fluid, according to Ewing, is made as follows: (1) Polychrome methylene blue solution: To 1 ounce of polychrome methylene blue (Grübler) add 5 drops of 3-per-cent. solution of acetic acid (U. S. P. 33 per cent.), to neutralize the undue alkalinity. (2) Ordinary methylene blue solution: Make a 1-per-cent. (saturated) watery solution of methylene blue, preferably Ehrlich's rectified, or Koch's, by aid of gentle heat. This solution improves with age and should be at least a week old. (3) Eosin solution: A 1-per-cent. solution in water of Grübler's aqueous eosin. Then to 10 c.c. of water add 4 drops of (3), 6 drops of (1) and 2 drops of (2), mixing well. To use, the specimens, fixed by alcohol or heat, should be immersed for two hours, specimen-side downward; they will not overstain by twenty-four hours' immersion. They should then be washed in distilled water, dried slowly over the flame, and mounted in Canada balsam. The density of the blue may be varied to suit individual fancy, nor need the proportions be rigidly followed, but the accurate neutralization of the polychrome solution is most important.

diameter. They develop into myriads of sporoblasts which reach the salivary glands of the insect, whence they are again inoculated into man by biting. The sporozoids thus resulting from sexual conjugation in the mosquito, are equivalent to spores formed by asexual sporulation in the human blood. Entering the red cells in man, they follow the cycle of their type as above described, and produce either sporulating forms or sterile forms. The sterile forms in the human blood—*i. e.*, the forms which do not sporulate—linger in the peripheral blood with the teleologic purpose of preserving and continuing their life in the mosquito, and, on reaching its body, develop again the agents of reinfection. This knowledge concerning the mosquito explains many interesting and formerly incomprehensible details. We now know why those living in the first story of houses are more likely to be infected than those living in the upper floors, and why those who slept in houses facing inland were infected while those in houses exposed to the sea air escaped, etc. In Rome, the fever prevailed just outside the Porta del Populo, while the Corso, three hundred feet distant in the city, escaped.

Symptomatology.—1. FEVER.—Fever is, next to the finding of the melanin and the plasmodium, the most constant symptom, though malaria should not be divided into types by the fever alone. It comes on after an incubation varying irregularly from one and a half to fifteen days. *Fever bears a close relation to sporulation* (Golgi, 1885). The fever-curve is most variable, being irregular in some types or almost mathematically regular in others. It is usually irregular at first even in regular types. It may be remittent (subcontinuous), intermittent (tertian, quartan, quotidian), or continuous for a few days, when it ends by crisis or becomes remittent or intermittent before recovery. The paroxysm may be delayed beyond the time when it is due (*postponens*), or may anticipate this time (*anteponens*). In the algid variety there may be hyperpyrexia, or normal or subnormal temperature.

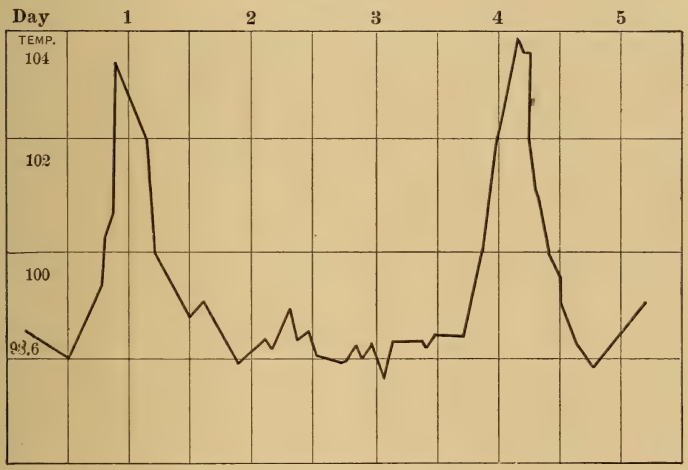
The classical divisions of the paroxysm are (*a*) chill, (*b*) fever, and (*c*) sweating, whose respective duration is in the proportion of 3–2–7 (Lorain). Any element of the paroxysm may be absent. Thus the fever or the rigor is absent in some virulent varieties and in persons partly immunized, and the sweating may be slight or absent. The paroxysm lasts ten (four to forty-eight hours), and in 91 per cent. of the cases occurs between 10 A. M. and 3 P. M. (*a*) The *chill* is sudden in its onset, often very severe, with headache, emesis, coldness, contraction and lividity of the skin (*cutis anserina*); the temperature by rectum is elevated, the pulse rapid, small, and hard; the respiration is rapid or even dyspnœic, and the urine is increased. The parasites are in the peripheral blood in the tertian and quartan types but largely in the internal organs in the æstivo-autumnal type. (*b*) The *fever* is high, perhaps not higher by rectum than during the chill, but is more evidenced by the red skin, thirst, delirium, and headache, soft, full, or dicrotic pulse, and other fever symptoms.

In this stage the young parasites are free in the blood and begin to attack new red cells. After their entrance into these cells the fever ceases. (*c*) In the *sweating stage* the young parasites are found in the red cells, and phagocytes take up the detritus and pigment. The sweat is usually profuse and somewhat characteristic, giving off an odor like that of semen. The patient may be exhausted, but postfebrile euphoria is usual.

Special Febrile Types of Malaria.—The quartan and tertian forms are regular and intermittent, and the æstivo-autumnal forms are more irregular and remittent.

1. The *quartan fever* is the rarest of all types ($\frac{8}{10}$ of 1 per cent.), is found in the temperate zones, and occupies a position geographically intermediate between the tertian and æstivo-autumnal varieties. It is caused by the quartan parasite, is the most regular of all types, and is called quartan because the simple form causes paroxysms every fourth day. The parasitic cycles are readily followed in the circulating blood, and the organisms do not accumulate in the internal organs. The fever is rather obstinate and recurrence is frequent, but

FIG. 10.

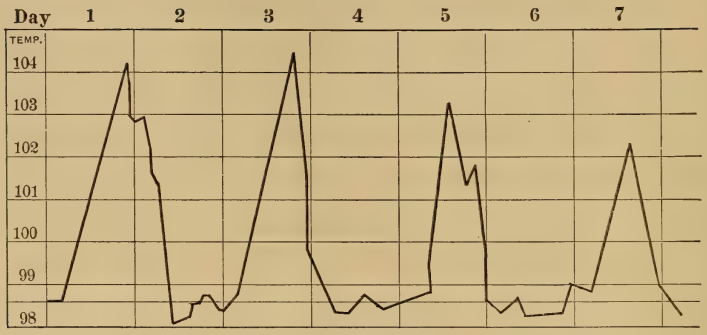


Quartan malaria. (Silvestrini.)

quinine exerts its specific action in a relatively short time. The type may be single, from one generation of parasites; double, from two generations; or triple, from three generations thus producing, respectively, temperature, every fourth, every first and second, or finally, every day of the cycle.

2. *Tertian fever* is most frequent in the temperate zones and is the most widely distributed variety. It is found in all malarial countries and constitutes 50 to 65 per cent. of all malaria. The organisms tend to accumulate in the internal organs. It is rarely pernicious. The type may be single, or more frequently, double from two genera-

FIG. 11.

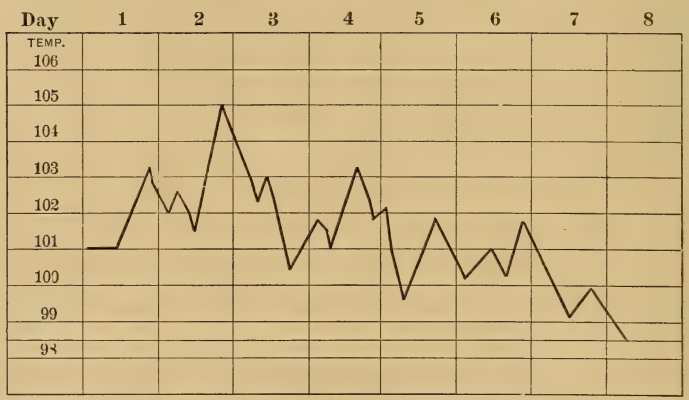


Tertian malaria.

tions of parasites. Almost continuous fever may be observed. It responds very readily to quinine or may recover spontaneously.

3. *Æstivo-autumnal fever,* caused by the æstivo-autumnal parasite, is often irregular, remittent, intermittent, or continuous (subcontinuous). It is characterized clinically by severe and recurrent fever, marked anæmia, frequently by absence of paroxysms (chills occurring in only 71 per cent.), and sometimes by pernicious symptoms. It was first called æstivo-autumnal by Marchiafava and Bignami, in contradistinction to the vernal or milder spring forms. *Sub-types:* (*a*) *Regular* intermittent æstivo-autumnal fever:—(i) The æstivo-autumnal tertian, in which the temperature is apt to be high, and the paroxysm doubly long. The typical curve may be mod-

FIG. 12.



Æstivo-autumnal malaria.

ified by the absence of a distinct initial elevation, by an exaggerated pseudo-crisis, by a briefer or longer duration of the attack, by anticipation or procrastination, by double attacks, and by marked oscillations. (ii) The æstivo-autumnal quotidian, in which the attack is usually short, but has an abrupt elevation of temperature.

This form often becomes irregular, has fewer symptoms than the other variety, is often mild and the patient may recover spontaneously. It is readily confused with typhoid. (*b*) The *irregular* intermittent æstivo-autumnal fever, which has marked irregular intermittency or remittency, or subcontinuous fever, because the attacks are duplicated, anticipated, or prolonged. The paroxysm may last twenty hours. The life-cycle of the parasite is often difficult to follow. The symptoms may be mild or very severe.

2. Blood-Findings.—The *blood-findings are pathognomonic; viz.,* parasites, melanæmia, anæmia and absence of leukocytosis are found.

(*a*) *Red Cells.*—Anæmia is always present from direct deglobulization by the parasite, and possibly from liberated toxins. *Acute anæmia* is naturally more marked and rapid than in any other acute infection. The loss is greatest in the first attack, especially in pernicious cases, and sometimes reaches half a million cells. Reduction to 500,000 red cells (Kelsch), thus giving the picture of pernicious anæmia, has been observed especially in pregnancy, and in children (Frayer and Ewart).

(*b*) *White Cells.*—The absolute and relative *leukopenia* is due to the collection of the white cells in the liver and spleen, to the destruction of the phagocytes, and to lesions of the blood-making organs. As in other anæmias caused by diseases of the bone-marrow and spleen, an increase of lymphocytes or large, mononuclear cells, with decrease of the polymorphonuclear cells, may be observed. Stevens finds the large mononuclears increased to 20 per cent. Leukocytosis in malaria denotes complications. An absolute increase is sometimes observed in the "black-water fever," but even here the increase is in the lymphocytes.

(*c*) *Melanæmia.*—Melanæmia (observed by Virchow and Heschl, 1847–50) is, *next to the presence of the plasmodium, the most constant change* in malaria. It probably occurs in every malaria. Melanæmia is present during fresh infection and may occur shortly after the fever has disappeared, as in the half-moon forms. After infection it usually disappears within forty-eight hours. It is *practically pathognomonic of malaria,* though it is said to occur in recurrent fever, in melanotic new growths, and in Addison's disease. It is found in the polymorphonuclears and sometimes in the lymphocytes. It is seen in the smaller capillary walls where their diameter suddenly decreases (lung alveoli, intestinal villi, appendices epiploicæ, renal glomeruli, cerebral convolutions, spleen, liver, and bone-marrow); the capillaries may be occluded by pigment. The lymph-glands are usually free of pigment. Besides this pigment the copper-colored hæmosiderin may be found not only in the vessels but in the parenchyma of the liver.

3. Spleen.—Enlargement of the spleen (first described by Andouard, 1803), is the third important finding. It is *palpable in 88 per cent.* of the cases and anatomically is rarely absent. Its size, hardness, and plumpness, are proportionate to the duration of the fever,

increase with its rise, and remit between attacks. In acute infections the spleen is increased three to five times its normal weight (five ounces); it is soft and friable; the veins are dilated, and the parenchyma is flooded with infected red and macrophagic cells; and there are occasional areas of focal necrosis. The Malpighian bodies contrast strongly with the pulp. A sticking pain over the organ, increased by moving the diaphragm, is due to capsular tension, or perisplenitis, and is often associated with pain and tenderness in the bones. As the disease subsides the splenic intumescence disappears. In chronic infections the spleen is permanently enlarged (fever-cake), reaching even into the pelvis. It may weigh two to ten pounds, the capsule is thickened, even calcified, perisplenic adhesions occur and friction is frequent. Sometimes a systolic murmur is heard (Griesinger). The organ is plump, and the acute hyperæmia and focal necrosis lead to increase of the connective tissue. The redundant pigment gradually decreases. Ultimately little remains of the pulp, follicles, and splenic function.

Rare complications are pressure on the intestines, nerves, and ureters, torsion of the pedicle of a wandering spleen, abscess, or rupture (of which Choux has collated 147 cases).

4. Skin.—The vasomotor constriction during the chill gives way, during the febrile rise, to hyperalgesia and vaso-dilatation. Miliaria appear in the sweating stage. Herpes is very frequent, mostly nasal or labial. Erythema is not common except from quinine; erythema nodosum has been observed in children. Roseolæ are most rare. Œdema and a cachectic hue occur chiefly in chronic cases. Slight icterus is not infrequent (urobilin icterus), and sometimes it is intense. Infrequent complications are urticaria, pemphigus, petechiæ, pigmentation, a measles-like eruption, furuncles, noma, gangrene, or erysipelas.

5. Circulatory Symptoms.—The heart is little involved, though myocardial degeneration with dilatation may occur. The pulse rises parallel with the fever, small at first from vaso-constriction, later dicrotic and full from vaso-dilatation. Endocarditis, angina pectoris, aortitis, or phlebitis are rare developments.

6. Respiratory Symptoms.—The rare dyspnœa and pulmonary congestion during a paroxysm are due to parasites and phagocytes accumulating in or even occluding the capillaries of the lung. Chronic bronchitis, emphysema, and chronic interstitial pneumonia, occur as complications. Plasmodia have been seen in the sputum. Bronchopneumonia is frequent in malarial cachexia. Genuine lobar pneumonia occurs only as a mixed infection. It is atypical, is often present without rusty sputum and without chill, and, in malarial cachexia, is especially fatal. Pulmonary tuberculosis and pleurisy are rare.

7. Digestive Symptoms.—The oral mucous membrane is dry; the tongue is heavily coated and the breath is foul. The parotid gland is sometimes swollen. The stomach may be the seat of erosions,

catarrh, hyperæmia, or of infarction with plasmodia. Anorexia, pyrosis, vomiting, and gastralgia, are common symptoms. Ulceration from amyloid degeneration is observed only in a few chronic cases. Hæmatemesis from chronic splenic tumor has proved fatal.

The Intestines.—Swelling of the lymphatic structures, erosions, catarrh, hyperæmia, thrombosis from parasite aggregation, meteorism, constipation, and rarely diarrhœa, are observed. Acute abdominal pain is not uncommon, but occurs without obvious cause. Pain may simulate peritonitis. Rarely the disease may resemble dysentery, with ulcers in the large and small gut.

The Liver.—In acute infections the liver is swollen, hyperæmic, or the seat of focal necrosis. Its macrophagic endothelial cells are swollen and its veins are full of pigment, though few parasites are present. Icterus may develop from sepsis or red-cell destruction; it is usually polycholic. Pigmentation from degenerated red cells is more common than actual melanosis. In chronic infections the liver may weigh eight or ten pounds, is hard and smooth, and has a thick capsule. The changes, *seriatim,* are focal necrosis, atrophy or partial regeneration, and pigmentation with hyperæmia. As a result of atrophy and regeneration, angioma, lymph-cysts, or abnormal lobules develop; hyperplasia of the perilobular connective tissue occurs with ultimate cirrhosis. Atrophic and hypertrophic cirrhosis may follow chronic malaria without necessarily being of malarial origin. Amyloid degeneration and simple atrophy from pylethrombosis are uncommon sequels. Acute ascites may result from pylethrombosis, from plugging of the capillaries by melaniferous cells.

8. Genito-Urinary Symptoms.—Polyuria is often observed during the paroxysm, despite the severe sweating. The urine is usually decreased in the æstivo-autumnal type. The post-malarial polyuria of convalescence is frequent. In one instance the author observed a veritable diabetes insipidus as described by Sydenham and Mosse.

The urea, iron, chlorides, and specific gravity, of the urine are increased. Phosphates are decreased during the paroxysm, and increased afterwards. *Albuminuria* is present in 25 to 40 per cent. of cases, and usually disappears with the paroxysm. Ehrlich's diazo reaction was obtained in 5 per cent. of the cases of Thayer and Hewitson. *Nephritis* occurs in 4.5 per cent. of cases, but is seldom fatal. Plasmodia are rarely found in the glomeruli or veins but frequently abound in the intertubular capillaries. Glomerular desquamation and epithelial exfoliation, endocapsular exudation, necrosis, and cast formation, develop in the convoluted tubules. Chronic parenchymatous and interstitial nephritis may result from previous malarial infection. The large white kidney, followed by secondary contraction, is considered by some to be a frequent sequel. Hæmoglobinuria, anuria, and uræmia, occur in blackwater fever (see Pernicious Forms). Gangrene of the genitalia is an uncommon sequel. Metrorrhagia and abortions occur frequently.

9. Nervous Symptoms.—The parasite and its presumed toxin especially affect the nervous system, whence van Swieten long ago called malaria a neurosis. There is scarcely a nervous symptom which is not occasionally observed in malaria. The sensorium, involved slightly in mild cases, is more seriously affected in the more severe types, the symptoms ranging from simple exaltation to coma and paralysis. Headache is usual, and neuralgias are frequent, especially of the fifth nerve. Cerebral confusion was noted by the writer in two ordinary tertian cases. In pernicious types it is not always easy to determine whether nervous symptoms are due to simple toxæmia or to cerebral thrombosis. The central nervous tissues are distinctly hyperæmic, and are anæmic only in the algid or choleraic types. The endothelial cells of the vessels are fatty, swollen, and pigmented. Thrombosis in grave cases, though it is usually caused by the rough infected red cells, may result from swelling of the endothelium. Thrombosis may lead to multiple small extravasations of blood. The infected erythrocytes never leave the vessels.

Symptoms of paralysis (hemiplegia, paraplegia, and monoplegia), or of motor irritation (tremor, chorea, contractures, epileptic attacks, or reflex spasms), may possibly occur in urgent types. Neuritis, neuroses, psychoses, ascending spinal paralysis, bulbar symptoms, symptoms resembling multiple sclerosis (Spiller), tabes or acute ataxia (Kahler and Pick), are rare sequences.

10. Muscles and Bones.—The muscles are often painful. Rheumatism is infrequent except in India. The bone-marrow is at first fatty and pigmented; the fat and pigment then disappear with marked vascularization, and, finally, with proliferation of the marrow-cells and connective-tissue thickening. Bone tenderness is often noted.

Relapses.—Relapses, caused by the awakening of an old infection, are almost invariable in all forms, but are most obstinate in the æstivo-autumnal type; the blood-findings are almost always positive. Reinfection is also frequent.

Chronic Malaria.—Untreated infection, or reinfection, persists for months, notably in the æstivo-autumnal variety. It is accompanied by irregular fever, marked anæmia, the parasite in the blood, and splenic and hepatic swelling. In severe chronic cases, the *malarial cachexia* develops, usually after years of infection. It is most marked in intensely malarial regions, in the æstivo-autumnal infection, and in reduced subjects. The fever is slight, irregular or entirely absent. The essential features of malarial cachexia are (1) anæmia from defective blood regeneration, with its general symptoms, such as œdema, rapid pulse, venous thrombosis, and hemorrhages in the skin, retinæ, stomach, etc.; (2) vascular changes, possibly due to pigment occlusion; (3) enlarged spleen and liver; and (4) gangrene, nervous symptoms, alimentary and respiratory catarrh, amyloid disease, liver hypertrophy, and extreme malnutrition.

Relation to the Other Diseases.—Malaria and typhoid rarely co-

exist; Craig saw but 8 instances in 5,000 personal cases. The detection of the plasmodia on the one hand, and typhoid bacillæmia or the Widal test, on the other hand, are necessary for diagnosis. *Surgical* operations may arouse a latent malaria. Sunstroke may initiate a pernicious attack. Erysipelas, septicæmia, dysentery, cholera, smallpox, tuberculosis, etc., very infrequently co-exist with malaria.

Pernicious Malaria.—Pernicious malaria, in the widest sense, is malaria which threatens life. Pernicious malaria constitutes but a small proportion of cases of æstivo-autumnal malaria. Very few pernicious cases have followed infection with the ordinary tertian parasite (Ziemann, French, and one case observed by the author). It is characterized by severe, obstinately recurring fever, pronounced intractable anæmia, pernicious visceral symptoms, frequently by abortive paroxysms without chills, by depression, tendency to recurrence, and the presence in the blood of the æstivo-autumnal plasmodium. The pathogenesis of perniciousness lies in the inelastic, irregular, infected red cells which cannot pass the visceral capillaries, and therefore slow the blood current. The melanosis, phagocyte aggregations, and endothelial swelling are of wholly secondary importance. In cases of capillary hemorrhages, only the normal red cells, not the parasite-laden red corpuscles, escape from the vessels. Regressional organic changes may occur in the cerebrum, gastro-intestinal mucosa, spleen, liver, and kidney. Accessory factors are (*a*) arteriosclerosis, renal disease, cardiac dilatation, alcoholism, sunstroke, overwork, malnutrition, and lack of acclimatization; (*b*) the number of parasites and their abundance in internal organs. Æstivo-autumnal parasites, with large blocks of central pigment, usually indicate many plasmodia in the viscera. The parasites are often few in the peripheral blood or in the blood of a splenic puncture, though they may abound in the brain at autopsy (Bastianelli, Marchiafava, and Bignami); (*c*) great activity in multiplication may be observed even in the peripheral blood where they are usually less abundant during the sporulating stage. Several plasmodia are sometimes found in a single corpuscle; the spores may number ten, twenty, or even thirty; (*d*) increased toxicity of the malarial parasites is purely hypothetical, as their toxin has not yet been isolated, but the necroses in the viscera and the great morphologic alteration in the "brassy" red cells, are suggestive.

1. The pernicious *typhoid form* (*perniciosa typhosa*), is inappropriately called typhomalaria. Its clinical picture is variable; chills are often absent; the sensorium is involved to a variable extent, and depression, delirium, insomnia, headache, and backache, occur. The fever is atypical, intermittent, or remittent (subcontinuous), resolves by crisis or by lysis, and is sometimes hyperpyretic or subnormal in register. Quinine generally disturbs or breaks the type. The pulse and respiration are increased. Vomiting of bile or blood, epigastric pain and tenderness, tympany, and thin, dark movements of the bowels may be marked. The spleen and liver are usually tender and

swollen. An irregular bronchitis, hæmoglobinuria and albuminuria are frequent. The mortality is 10 per cent., and, in adynamic forms, 25 per cent. Fatal cases usually terminate with nervous symptoms, and especially with coma. The differential diagnosis was impossible before the blood-findings were known, but was often made *ex juvantibus* by administration of quinine. Without blood examination malaria and typhoid may be confused; they very rarely co-exist; benign or malign malaria may resemble typhoid closely, with slow, dicrotic pulse, splenic tumor, headache, fever-curve, cerebral and abdominal symptoms, and even roseolæ and intestinal hemorrhage; on the other hand, erratic typhoid with remittent temperature, chills, and sweats, or typhoid in the latter part of its normal course, may simulate malaria.

Baccelli gives the following differentiation:

Malaria (Subcontinua typhosa).——*vs.*——Typhoid.	
Begins intermittently.	Progressively remittent.
Remissions very irregular.	Very regular, and in the morning; step-like.
Temperature may be high at first.	Considerable fever, reached only on the third or fourth day, or later.
Headache in beginning is rare, variable in site and intensity, pulsating and neuralgic.	Early, permanent, frontal, oppressive, characteristic.
Eyes dull, subicteric from beginning.	Glistening eyes; no icterus.
Stupor, dry tongue, and sordes, less.	Marked.
Breath nauseating.	Mouse-like.
Delirium from beginning; recurs.	In advanced stage; constant.
Lung congestion begins suddenly; foci change recurrently; dyspnœa from deranged circulation.	Slow development, hypostatic; dyspnœa rare (from tympany).
Nervous restlessness, jactitation.	Stupor, prostration.
Meteorism not characteristic; stools darker.	Meteorism and diarrhœa begin gradually; pronounced; ochre stools.
Liver congestion frequent; subicterus.	Less congestion, rarely icterus.
Herpes is frequent.	Rare. Roseolæ.
Cycle, none.	Very typical.
Blood; plasmodia and melanæmia.	Widal reaction and typhoid bacillæmia.
Recurrence.	Relapses less frequent.
Convalescence more rapid and easier.	Tardy.
In adynamic; icterus, hæmoglobinuria, leukocytosis, great anæmia.	Does not respond to quinine, which specifically reduces malarial symptoms.

2. Of *cerebral forms,* the comatose (*perniciosa comatosa*) is the most frequent. Melancholia, and impaired memory, may exist in the preliminary stage; later, contracted pupils, involuntary evacuations, lessened reflexes, or paralysis, may develop before coma intervenes. If the patient recovers, bradylalia, scanning speech, and insufficient motility of the lips or tongue, may be observed. The pulse at first is generally slow and regular, but as death approaches it becomes soft, thready and rapid. Death is usually cardiac. The respirations vary, being quiet or noisy, hurried, or of the Cheyne-Stokes' type. If fever is present it is irregular. The author has seen four such cases in Chicago. Apoplexy, uræmia, and sunstroke must be considered. The splenic tumor *per se* suggests a blood examination. The "meningeal type" occurs especially in children, and in these cases the slow pulse, headache, rigid and tender neck, stupor, convulsions, involuntaries, hyperæsthesia, and ultimate coma, can be correctly interpreted only after a blood examination. Delirium may be the main symptom, and may appear as a mania, as fixed ideas, hallucinations, or as melancholia. The rare convulsive type may be (*a*) generalized, and may simulate tetanus, hysteria, uræmia, or epilepsy especially in children, or (*b*) localized, with such symptoms as contractures, trismus, or opisthotonos. The paralytic form is the least frequent type. Hemiplegia with aphasia is more common than monoplegia, the bulbar form, etc.

3. The *algid pernicious type* occurs with cyanosis, collapse, Hippocratic expression, marked alimentary symptoms, and usually, early death.

4. After the cerebral type the *pernicious gastro-intestinal* variety is most frequent and occurs with vomiting, pain, epigastric distress, icterus, and dysenteric manifestations. In a soldier from Cuba, the author observed profuse hemorrhages from the bowel. A prompt response to quinine followed the detection of the æstivo-autumnal plasmodium.

6. The *pernicious biliary fever* ("jungle fever") occurs with vomiting, polycholia, icterus, nervous symptoms, hemorrhages, adynamia, œdema, and dark albuminous urine. The course is usually ten to twelve days, but even after the administration of quinine this type may be lethal.

6. The *hemorrhagic types* resemble Werlhoff's disease, and scurvy.

7. The *febris biliaris hæmoglobinurica* ("black-water fever") occurs in South America, in Africa, and in our Southern States. The factors in its production are syphilis, alcoholism, repeated malarial attacks, exposure to changes in climate, undue exertion, cold, and in some instances the use of quinine. The etiological role of quinine is questioned, and some other factor is apparently more potent. The cardinal symptoms of this type are (*a*) hæmoglobinuria; the urine is always albuminous, neutral, or alkaline, in reaction; hæmoglobin is detected only after the destruction of at least one-sixth of the circulating red blood corpuscles; (*b*) icterus, invariable ex-

cept in the mildest cases; (*c*) irregular fever; (*d*) the detection of the plasmodia; Stephens in 95 cases found the parasite in 96 per cent. before, and in 62 per cent. at the onset of the attack. The mortality of 40 to 50 per cent. is due to syncope, hemorrhages, anuria, and coma.

Diagnosis.—1. From Symptoms.—A probable diagnosis can often be made, as, for instance, from typical intermittent *fever* which occurs early in the day and is distinct from the vesperal hectic or septic fever. The three elements of the paroxysm are most marked in the benign malaria. In the æstivo-autumnal form the chills may often be absent. *Splenic enlargement,* and *herpes,* are always suggestive, but they also occur frequently in ephemeral fevers, pneumonia, meningitis, and influenza, and more rarely in typhoid or tuberculosis. Much emphasis was once placed on intermittent neuralgia, vertigo, herpes zoster, and alimentary and respiratory catarrh but intermittence is not synonymous with malaria.

2. The Diagnosis ex Juvantibus.—Laveran excluded malaria, when, for more than four days the fever resisted the daily administration of 20 to 30 grains of quinine. In tropical forms more quinine may be necessary for this purpose. However, other diseases than malaria may subside under the administration of quinine.

3. The Plasmodium.—The plasmodium may be mistaken for vacuoles resulting from mechanical injury to the red cells; particles of immersion oil; retraction of the hæmoglobin of the corpuscle; swarming particles from the red-blood-cells; blood plates which are sometimes mistaken for spores; the differentiation from these is easily made in the stained specimen; and for poikilocytosis. Possibly fewer mistakes are made when the inexperienced observer examines the fresh blood. Leukocytes containing pigment are distinguished by their large nucleus and by their amœboid movement, the latter of which is lacking in plasmodia sufficiently large to cause confusion.

Positive Findings.—The malarial parasite is found in the blood of malarial patients only. Osler found them in every instance, in 531 cases. It is usually stated that they occur in largest numbers before or at the time of the paroxysm though it must be admitted that the first examinations in early fever are often negative. Previous administration of quinine may render the findings obscure. Melanæmia is practically pathognomonic. Pigment lying free in the plasma is of no importance, for even the greatest care will not eliminate the possibility of contamination.

Negative Findings.—These are of relative value only, and depend upon the skill of the observer, and the time of the examination. The findings were negative in only 2 per cent. of Baccelli's series. Puncture of the spleen is dangerous.

Differential Diagnosis.—1. From *typhoid* fever (see Pernicious Malaria and Typhoid).

2. In *sepsis* (see table under typhoid), pyæmia, septicopyæmia, ulcerative endocarditis, liver abscess, hectic pulmonary phthisis, and suppuration in the intestinal and urinary tracts, may be confounded

with malaria. However, we find in malaria the plasmodium, melanæmia, response to quinine, and absence of leukocytosis; whereas in sepsis, leukocytosis, positive blood cultures and septic localizations are very frequent.

3. *Pneumonia* with remittent fever; irregular grippal temperature; recurrent fever; miliary tuberculosis; fever in malignancy, especially in sarcomatosis; pernicious anæmia; splenic anæmia; leukæmia; pseudoleukæmia; Weil's disease; yellow fever and acute febrile jaundice, may be confused with malaria, if careful and repeated blood examinations are not made. Pernicious malaria (*v. s.*) may superficially simulate apoplexy, sunstroke, meningitis, cholera, dysentery, or purpura. Among 1267 malaria patients, 25 per cent. were "masked," simulating tuberculosis, pneumonia, dysentery, appendicitis, etc. (Craig).

Prognosis.—The degree of infection, type of parasite, reinfection, health and habits of the individual, and antecedent renal or cardiac disease, are the most important considerations. The prognosis must be conservative in pernicious and cachectic forms. In Italy the yearly death-rate was 15,000, and the French lost 6,000 soldiers in Madagascar in three months. On the other hand, fully 20 per cent. recover when given rest in bed and a full diet, as is often shown in cases which are treated with placebos and saved for clinical demonstrations. Spontaneous recovery is not yet fully understood. The factors are the *phagocytes* in the spleen, bone-marrow, and to a less degree, the endothelium of the splenic, hepatic, and cerebral vessels, Kupfer's cells in the liver, and possibly the leukocytes.

Treatment.—1. Prophylaxis.—Prophylaxis embraces more than personal prevention. In Italy and other countries it is a national economic problem. Drainage, the filling in of stagnant pools and swamps and the planting of pines, eucalyptus trees and sugar-cane, have proved most successful, though sometimes accomplished at great sacrifice of life. The drainage of the great Bordeaux swamp in 1805 cost 3,000 lives. In digging canals or building roads, immunes should be employed, but whites may work successfully if they avoid excessive labor in the hot sun, alcohol, and exposure to the night air. Nocturnal infection by mosquitos may be minimized by using nettings on the doors and windows. The pools where the insects breed should be filled in, or covered with crude petroleum to kill the larvæ. The stocking of semistagnant streams with fish is also of value. The prophylactic use of quinine in daily doses of 5 to 10 grains is effective. Sick malarial patients and also the considerable number of those with malaria but without symptoms, should be protected from the bites of mosquitos which carry infection to others.

2. Specific Treatment.—*Quinine* is one of our few specifics. Cinchona has been used for ages in Peru and was known as the "tree of health" to Pizarro in the conquest of Peru. Introduced over one hundred years later into Europe by del Vego (1640), its alkaloid was discovered by Pelletier and Caventou in 1820. *It*

acts on the malarial organism while it is developing (Marchiafava and Bignami); when the parasite has transformed all the hæmoglobin into melanin, and segmentation begins, quinine is ineffectual. All such malarial manifestations as fever, acute splenic tumor, and anæmia, are cured by quinine except in pernicious forms. It acts as a direct protozoan poison. Binz thought it entered into such close union with the hæmoglobin that the parasite was suffocated. Just as syphilitics tolerate much mercury and iodide, so malarial subjects escape many or all of the symptoms of cinchonism, such as tinnitus aurium, cephalic distention, and slight deafness resulting from therapeutic doses (gr. x). (Deafness, disordered vision, flushed face, and cerebral congestion, vomiting, staggering, tremor, and twitchings, result from larger doses, and stupor, delirium, cardiac oppression, complete deafness and amaurosis, dilated pupils, convulsions, paralysis, hæmoglobinuria, dyspnœa, coma, weak heart, or hemorrhages result from toxic doses.)

Administration.—If idiosyncrasy exists, cinchonism is lessened by fluidext. ergotæ 3ss, or, better, by sodium bromide 3ss. Its antiperiodic action is enhanced by combining it with opium gr. ss. Laxatives are unnecessary. Pills are often insoluble, and the drug is therefore given *in powders involved in starch wafers,* or with one drop of sulphuric acid to each grain of the sulphate. Chloroform, coffee, cognac, and extract of glycyrrhiza but poorly disguise its bitterness. If the stomach rebels, a warm-water enema with equal parts of quinine and tinct. opii deodorat. may be used. In severe cases the hydrochloride (gr. vii) in 15 minims of water should be used hypodermatically, but, though every care be taken, abscesses develop too often. In pernicious types, Baccelli injects quinine intravenously (sterilized solution of sodium chloride, quinine muriate, and water, in the proportion of 1, 10, and 100, parts respectively).

Time of Exhibition.—If the fever recurs regularly, gr. xx. to xxx. should be given as the fever falls, to act on the growing young forms in the red cells, to be repeated before the next paroxysm, in order to have the drug in the circulation when the spores are scattered (Torti and Golgi). If the fever is continuous or sub-continuous, or if the type is severe, gr. v. should be given every four hours. Large doses are recommended because they are more effectual, and small doses are quite useless and sometimes only serve to aggravate existing malaria and develop quiescent forms (Steudel-Kuchel). In the treatment of black-water fever, one view maintains that it is caused by the parasite, and another that quinine is its cause. Quinine, however, should be given only when plasmodia are found, and should be withheld after they disappear.

3. Symptomatic Treatment of the Paroxysm.—During the vaso-constriction of the chill, morphia gr. $\frac{1}{4}$ and nitroglycerine gr. $\frac{1}{50}$, hypodermatically, and spirits of chloroform 3j., in hot whiskey and water afford great relief. The fever is short-lived and requires little interference other than the ice-cap, and cool sponging. The sweat-

ing requires no treatment. A weak heart should be supported by morphine, and strychnine hypodermatically, and saline enemata.

In chronic malaria, iron and full increasing doses of arsenic should be combined with quinine. Arsenic is said by Durham to act on the gametocytes. Celli finds iron and arsenic useless.

SEPTIC INFECTIONS.

Synonyms.—Sapræmia, septicæmia (sepsis), pyæmia, septicopyæmia.

Definition.—*Intoxications* are the result of the absorption of substances elaborated for the most part by microörganisms, but possibly also by living cells. *Infections* are always due to microörganisms. *Sapræmia* is the absorption of putrescent substances but not of the germs which develop them. *Septicæmia* (sepsis) is the presence in the blood of microörganisms (bacteriæmia),—which are usually pyogenic,—without hæmatogenous suppuration or metastasis. *Pyæmia* is infection, caused by microörganisms, which leads to hæmatogenous suppuration or metastasis. Septicopyæmia is co-existent septicæmia (bacteriæmia) and pyæmia (metastatic suppuration).

General Groups.—1. Some infections—such as carbuncles, puerperal parametritis, diphtheria, and tetanus—are wholly *local* so far as the causal microbe is concerned, and their symptoms are due to absorbed toxins (toxinæmia, toxæmia).

2. Other infections—such as typhoid, pneumonia, carbuncles, puerperal sepsis, anthrax, and gonorrhœa which cause *bacteriæmia* or *septicæmia—at first local,* may *become general* when their germs enter the blood. Some of these infections, especially typhoid and pneumonia, which were previously considered local, are always general.

3. Still other infections, often resulting from the same germs, cause *metastatic suppuration* (*pyæmia*). Among these are multiple abscesses from carbuncle, puerperal fever, typhoid osteomyelitis, pneumococcic arthritis, and gonorrhœal endocarditis. It is frequently difficult or impossible to preserve clinically these types which so often blend; what is seemingly toxæmia may prove bacteriæmia on examination of the blood; apparently pure bacteriæmia may prove pyæmia at necropsy; all three grades may occur successively; the primary infection—*e. g.,* with scarlatina, tuberculosis, and diphtheria—whether local or general, may be complicated by secondary, or mixed infection, especially by the streptococcus; again the symptoms vary, first with the variety of microbe and its virulence; second, with its localization (in the throat, lung, pelvis, etc.); and third, with the physiological resistance. Subjects of malignancy, cardiac, vascular, nephritic, cirrhotic, and other disease, readily succumb to an ultimate sepsis to which the name of *terminal infection* is applied. For these reasons we will discuss septic infections as a whole, and indicate such special etiological and clinical variations as seem necessary.

Etiology of Septicopyæmia.—The causal organisms of septic infections are numerous. The most common are the pyogenic cocci, the pneumococcus and the colon bacillus; less frequent are the gonococcus, anthrax bacillus, typhoid or diphtheria bacillus, Friedländer's pneumobacillus, meningococcus, bacillus pyocyaneus, influenza bacillus, Fränkel's bacillus phlegmones emphysematosæ, bacillus aërogenes encapsulatus, and possibly the micrococcus tetragenus, the spirilli and bacilli of Vincent, and the proteus group. Although puerperal fever, primary wound fever and severe late pyæmic infection were known to Hippocrates, and also to Willis (1682), Semmelweiss (1847), and others, it was reserved for the bacteriological era, building on Virchow's work, to clarify this group of affections. Though Wunderlich (1847) spoke of spontaneous pyæmia, and Leube (1878) of cryptogenetic (occult) septicopyæmia, careful clinical and necropsy investigations reveal the *atrium of infection* in 94 per cent. of cases; (1) *skin* lesions, such as furuncles, felons, and bites (staphylococcus more frequent than streptococcus); (2) *throat* (streptococcus); (3) *nose* (not frequent in sepsis, though in meningitis the atrium for the meningococcus and pneumococcus); (4) *ear* (pneumococcus and streptococcus); (5) *lungs* (pneumococcus); (6) *intestines* and *bile tracts* (colon bacillus; typhoid bacillus, gall-stone infection, appendicitis, dysenteric ulcers); (7) *urinary* tract (pyelitis from colon bacillus; vesical and urethral infections chiefly by the staphylococcus, far less by the streptococcus and gonococcus); and (8) *vagina* and *uterus* (chiefly streptococcus).

General Pathology and Symptomatology.—(*A*) General Toxæmic Symptoms.—1. *Fever.*—Fever is usually the first clinical symptom and most often occurs abruptly. The *virulence* of the germ and its toxin (rather than its variety) *governs the intensity of the toxæmic symptoms.* In general the fever tends to be remittent or intermittent. Lenhartz, in his work on septic infections, concludes that (*a*) the streptococcus infections usually produce irregular intermittent fever with moderate variations (sometimes remittent or in severe cases, continuous fever); (*b*) the staphylococcus usually causes weakly remittent or continuous fever; (*c*) the pneumococcus produces remittent or continuous fever; (*d*) the colon bacillus and gonococcus cause intermittent fever with great variations. Fever may be absent in very severe forms, especially before death.

2. *Chills.*—Chills occur both in septicæmic and in pyæmic forms, are usually irregular, but may occur daily or two or more times a day.

3. *Nervous Toxæmia.*—Pronounced toxæmia may appear as unrest, delirium, stupor, dry tongue, and the status typhosus, or it may resemble meningitis. The sensorium may be clear.

4. *Vasomotor and cardiac weakness* may be present, with increased, often irregular, or hopping pulse. 5. *Increased respiration* and bronchitis may be noted. 6. Albuminuria and acute nephritis are common. 7. *Septic diarrhœa,* in which the dark movements number six

to ten daily, may be observed. 8. Acute *splenic tumor* is present. 9. *Anatomically* we find cloudy swelling of the heart, liver, and kidneys; a vascular, soft, moderately swollen spleen; acute nephritis; hemorrhages into the skin, retina, and mucous and serous membranes, with or without bacteria; lung œdema, and, where there is marked anæmia, fatty heart, hemorrhage into the bone-marrow, and increased iron deposit in the liver.

(*B*) Blood-Findings.—1. *Bacteriæmia* is found in 50 per cent. of the cases clinically, and in 95 per cent. at autopsy (Lenhartz). Some circulating bacteria are apparently killed by the blood. In most streptococcic and staphylococcic infections, these organisms are found in the blood.

2. *Polymorphonuclear leukocytosis* is frequent, though not invariable.

3. *Anæmia.*—Marked anæmia is usual, and sometimes even resembles the pernicious form.

(*C*) Metastases.—They are apparent clinically, or first found at necropsy. Metastases occur through the veins or lymph-vessels. *Thrombo-phlebitis* develops at the seat of primary infection, and disintegrated thrombi, containing bacteria, escape into the venous circulation. They lodge in the lungs or, passing them, reach the left heart and arterial system. Purulent lymphangitis may, in the same way cause metastatic suppuration. If *"end arteries" are plugged,* infarcts result; if the arteries are not terminal, small foci develop. Not all infarcts suppurate. This difference is explained by the fact that the germs found in them are less virulent, or that the embolic material is bland or free of microbes. Suppurative foci occur in organs in which there are end arteries and in which the circulation is slow, especially, therefore, in the liver, kidney, and spleen. (1) *Cerebral metastasis* may cause *meningitis, brain abscess,* or extensive softening (encephalomalacia with aphasia and hemiplegia). (2) *Cardiac* metastasis develops in 21 (even 63) per cent. of the metastatic group; *endocarditis* is localized chiefly on the mitral (62 per cent.) and aortic (22 per cent.) valves; it is attended by a heart murmur in only 60 per cent., and often by *pericarditis;* the *heart muscle* is sometimes the seat of abscesses. (3) *Lung* localization may cause multiple or single abscesses, and often sero-fibrinous or *purulent pleurisy.* (4) *Renal* localization may result in nephritis or suppurating infarcts, with albuminuria, cylindruria, pyuria and bacteriuria. (5) *Splenic* localization usually results in enlargement, frequently in pain, and rarely in perisplenic friction. (6) Localization in the *bones,* may cause early fugitive bone pain, or later, fixed osteomyelitic tenderness over one or more bones, with inflammatory signs; in the *joints* (as in the post-febrile arthritides), it may cause simple pain, or serous or purulent synovitis; in the muscles it may produce abscesses, purulent œdema, or Wagner's polymyositis. (7) Localization in the *skin. Ecchymoses* occur in 50 per cent. of fatal cases; they are usually late, often symmetrical, and sometimes occur on the

nose, fingers, toes, or ears. *Pustules* and vesicles are less frequent; herpes occasionally develops. Scarlatiniform, morbilliform, roseolous or urticarial eruptions, are only toxæmic rashes. (8) Localization in the *eyes* produces retinal ecchymoses in 33 per cent. which were first described by Litten. They are round, irregular, or develop in streaks, with small white centers and appear late or before death; the white spots of Roth (8 per cent.) were once considered inflammatory, and are still unexplained. Least frequently eye localization results in panophthalmitis, of which 33 per cent. is due to ulcerative endocarditis. (9) The *peritoneum* is chiefly involved in puerperal forms. (10) The slightly enlarged *liver* is more often due to toxic parenchymatous degeneration and cardiac weakness than to abscess, which occurs in only 15 per cent. of cases of surgical septicopyæmia (Bärensprung). Intestinal infarction is rare.

Special Symptomatology.—This may be considered etiologically or topographically. Only the leading characteristics are presented.

(*A*) Etiological.—1. *Streptococcic infection* is the *most frequent primary* and *secondary septic infection.* It constitutes 66 per cent. of all forms (Mau), the coccus entering the blood before death in 33 per cent. of all fatal diseases. Its atrium is most often *the throat* and (in the female) the genitalia; it enters the veins in puerperal pyæmia more often than the lymphatics (erysipelas and puerperal sepsis); it more often produces septicæmia (65 per cent.) than pyæmia (35 per cent.); the lungs are usually unaffected; endocarditis occurs in 7 per cent.; its course is chiefly acute, though sometimes subacute (ulcerative endocarditis).

2. *Staphylococcic infection* usually *enters* by *the skin* and the furuncle is the typical primary local lesion. On the eye-lids, nose, or lips, this lesion is dangerous because infection may reach the facial vein, then the ophthalmic vein and the cavernous sinus. Furuncle of the neck may reach the transverse sinus. Infections of the skin are the most frequent cause of *osteomyelitis.* Meningitis is known to have followed a felon. The infection enters through the throat, with difficulty but then with increased malignancy (Kocher and Tavel). It travels by the veins or lymphatics; it produces metastasis in 95 per cent. and endocarditis in 6 per cent. of cases. Abscesses of the lung and kidneys are very frequent; the coccus is found very often in the blood and urine. Pustular skin eruptions are frequent. Staphylococcic infection is usually acute.

3. *Pneumococcic Infections.*—In all cases of pneumonia (*q. v.*) the pneumococcus appears in the blood (pneumohæmia). In actual sepsis following pneumonia, it enters the blood through the pulmonary veins. Infection through the ear or bile-tracts is less frequent; metastases occur in 25 per cent. and endocarditis in 6 per cent. Twenty-nine cases of pneumococcic meningitis were first reported by Nauwerk (1881), but it is yet unknown why lung infections so frequently cause brain metastases. The pneumococcus alone may produce pus in the joints, skin, thyroid, and serous membranes. Netter's state-

ment that pneumococcic sepsis is less severe than other types may be true in some cases, but it is often malignant. It may be associated with the pyogenic cocci.

4. *Colon sepsis,* which is less frequent, may follow wounds, suppuration in cholelithiasis, cystitis, pyelitis, or intestinal obstruction. Endocarditis is exceptional, and sepsis (78 per cent.) is more common than metastasis (22 per cent.).

5. *Gonococcic infection* is rather more benign than the other types. There may be gonorrhœal endocarditis (Bordoni, Uffreduzzi), pericarditis, and pleuritis, or the more common gonorrhœal arthritis. Thayer found the gonococcus in the blood (*v.* Gonorrhœal Infection).

Other forms are less frequent. These are typhoid suppuration in the pleura, thyroid, bones, meninges, muscles, ear, and parotids, sepsis from Friedländer's pneumobacillus, the meningococcus, etc. Pyocyaneus sepsis is attended by irregular or continuous fever, rapid pulse, vomiting, diarrhœa, and an hemorrhagic diathesis.

(*B*) Topographical.—(1) *Ulcerative endocarditis;* (2) *cholangitic sepsis;* and (3) *otogenous sepsis* (*v.* Sinus Thrombosis, Meningitis, Brain Abscess) will be considered elsewhere. The two following surgical or obstetrical localizations are of importance to the general practitioner and internalist.

(4) *Osteomyelitis* is either a solitary metastasis or part of a generalized sepsis. Even when a single bone is involved, the blood often contains the causal microörganism. It is due to the staphylococcus chiefly; then to mixed infection with the streptococcus, which is found in all fatal cases, or with the pneumococcus; and but rarely to the typhoid organism. Fränkel has recently found that in most autopsies on typhoid and pneumonia cases, their causal bacteria are present in the bone marrow. Acute forms of osteomyelitis are most frequent in the growing bones of children. Trauma is a promoting element and the onset occurs with *chill,* fever, vomiting, diarrhœa, headache, and *bone pain.* It is usually first seen (and often mistaken) by the general practitioner. Chronic forms are quite infrequent, but Kocher, Jordan, and others, have described cases, which, lasting one year or more (even thirty), may simulate sarcoma, or syphilis.

5. *Puerperal fever* is caused by the streptococcus (95 per cent.) or rarely by other organisms. It is still disputed whether streptococci normally reside in the healthy vagina. Its varieties are (*a*) *toxæmia* or *sapræmia.* The symptoms are caused by toxins or putrid decomposition products respectively. Even in cases apparently of this group, bacteriæmia may be found (colon bacillus, saprophytes, or streptococcus). The rare puerperal tetanus is wholly toxæmic. (*b*) *Lymphangitic form*—Lymphangitis, from infected vaginal or cervical wounds, may cause, (i) moderate, benign, localized *parametritis.* (ii) General *septicæmia,* from vaginal or cervical wounds, which follows the lymph-vessels through the pelvic cellular tissue to the general circulation. The symptoms usually appear within a day or so after delivery. Death may result in two to fourteen days, or encapsulation

and massive cellulitis may develop. (iii) Lymphangitic *peritonitis* is more frequent (20 per cent. of fatal cases). It begins three or four days after delivery and is usually generalized and suppurative. Infection may travel directly to the peritoneum or, more often, by retrograde lymphatic routes. This form includes Virchow's "erysipelas grave internum puerperale." It is more common in labor at term than in abortion. The symptoms are chill, fever, tense pulse, vomiting, abdominal pain and distention, and often effusion, and diarrhœa. In many apparently purely peritonitic cases bacteriæmia is found. Early death is the rule, commonly in five to six days, but encapsulation is possible. (*c*) The *thrombophlebitic* (pyæmic) type occurs in 50 per cent. of fatal cases. It usually begins in endometritic ulceration, diphtheria, or ichorous inflammation, in contradistinction to the lymphangitic form of vagino-cervical origin. Infective thrombophlebitis travels (i) from the upper veins by way of the spermatic veins, of which the right empties into the inferior cava and the left into the renal vein; the phlebitis may actually extend into the vena cava; (ii) from the lower plexus, by way of the uterine veins into the hypogastric veins, and thence to the inferior cava. Bilateral venous thrombosis is common (25 to 66 per cent.). Metastases are common,—58 per cent. in the lungs, 25 per cent. in the kidneys, 20 per cent. in the joints and muscles, and 16 per cent. in the spleen. Ulcerative endocarditis, meningitis, and peritonitis, may be present. The course may be stormy from predominating toxæmia, or slower with metastases. The fever is irregular, the pulse rapid, the chills severe, the anæmia marked, and streptococcæmia is present in over 90 per cent.

Diagnosis.—The direct diagnosis rests upon the etiology, the symptoms of toxæmia and metastatic suppuration and the blood cultures. The *differentiation* from typhoid, malaria, and miliary tuberculosis, is fully considered in the table under typhoid fever. Solitary local or visceral symptoms, as osteomyelitis, arthritis, endocarditis, etc., may cause confusion if not viewed in their broad relations to a possible sepsis.

Prognosis.—The prognosis depends upon the organism, *its virulence* and *dissemination*, the patient's *physiological resistance* (whence the obviously poor outlook in terminal infections), and finally on the *surgical accessibility* of the primary lesion or metastatic foci. Only 17 per cent. of Lenhartz's cases with bacteriæmia recovered. A stormy onset and chills are ominous. A case of Lenhartz with empyema, lung abscess, panophthalmitis, diffuse venous thrombosis, and multiple osteomyelitis, recovered. Puerperal infections, with the large wound-surface and frequent hemorrhages, are severe, the mortality of all forms being 55 per cent., and of severe forms 65 per cent. (Curschmann). The streptococcus has been regarded with particular dread; however, Bertelsmann (1904) states that the prognosis is twice as favorable as in staphylococcic invasion.

Treatment.—Surgical prophylaxis, even in apparently insignificant

wounds, and obstetrical asepsis, is important, especially in subjects reduced by general diseases. Treatment of existing sepsis consists in giving the largest possible amount of food which can be digested; in free administration of salt solution by rectum or transfusion, in order to flush the toxins through the kidneys; and in the free use of alcohol, given as in typhoid or pneumonia, but in its maximum dosage because patients exhibit an exceptional tolerance to the drug; unquestionable benefit results. The treatment of hypostasis, diarrhœa, vomiting, and chills, is symptomatic. The temperature is seldom influenced by drugs, though quinine (see PNEUMONIA) is most safely given. The internal use of germicides is justly abandoned. We have not observed the slightest benefit from Credé's silver ointment; Credé's collargol has been used with benefit, intravenously, in doses of one to two and a half drams. Nuclein and 3j to 3ij of fresh brewers' yeast are also advised. The use of Marmoreck's antistreptococcic serum has given no essential reduction in the death-rate, and its suggestive effects in isolated cases is easily misleading. Lenhartz has used a serum from the blood of convalescents from sepsis with some success. Surgical intervention is indicated whenever definite accessible foci are localized.

PNEUMONIA.

Synonyms.—Fibrinous pneumonia, croupous pneumonia, lobar pneumonia, pleuropneumonia, pneumonitis, lung fever.

Definition.—An acute specific infection caused by the pneumococcus, self-limited in its course, characterized by lobar fibrinous inflammation of the lung, toxæmia, pneumococci in the blood, and solution of the fever by crisis. It is not uncommonly complicated by secondary infective processes.

Frequency.—Pneumonia constitutes 6.5 per cent. of all internal diseases, 4 per cent. of all diseases, 6.6 per cent. of all medical deaths (Liebermeister), and 8 per cent. of all deaths (E. F. Wells).

Bacteriology.—1. The *pneumococcus* or Diplococcus pneumoniæ (Plate IV.), was recognized by Fränkel (1884) and Weichselbaum (1886), who first demonstrated its frequency and importance; the diplococcus bears their name. It is an elliptical, lanceolate, encapsulated diplococcus; it is easily seen in cover slips, it readily stains by the Gram method, and presents many cultural variations. Its *atrium* in pneumonia is by way of *the respiratory tract,* although in sepsis (*q. v.*) it may enter the blood by other avenues. From the lung it may spread diffusely, chiefly through the bloodvessels or possibly by way of the lymphatics. It is found in dust, in saliva, and in 30 per cent. of healthy noses and throats, though usually with attenuated virulence. Again, the organism is virulent, and experimental inoculation has produced pericarditis, endocarditis, and empyema. It has been found in the blood, joints, brain, bone-marrow, etc., in otitis media, endocarditis, peritonitis, cholangitis, and endometritis; in the

urine and fæces; and it may pass from the mother to the fœtus. It may be a terminal septic infection. Until recently it was thought that typhoid and pneumonia were local infections with general symptoms, and that the presence of the microbes in the blood was a serious and true complication. The typhoid bacillus is found in the blood in 95 per cent. of typhoid cases, and Rosenow, of Chicago, has found the pneumococcus in the blood in about 93 per cent. of pneumonia patients (*v. i.*). Prochaska found the organism in all of fifty cases. It is assumed that a *pneumotoxin* exists and produces the toxæmic symptoms; when an antitoxin develops, the crisis occurs. *Immunity* does not result from one attack; as in erysipelas, recurrence is likely in 13 to 33 per cent.; ten, or even twenty-eight recurrences are known. The pneumococcus is probably the sole cause of typical pneumonia, but other microörganisms are sometimes found:

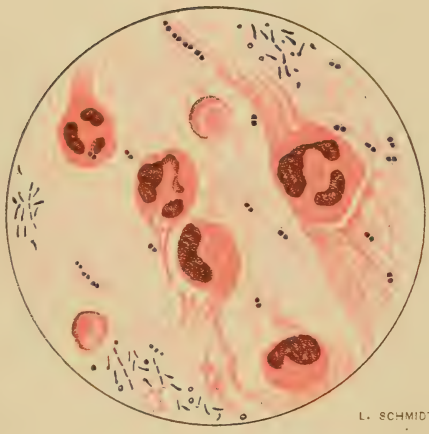
2. The pneumobacillus (Friedländer, 1883) is 1μ long, one-third as wide as long, with a capsule; it sometimes is found in the healthy nose, is larger than the pneumococcus, and does not stain by Gram's method. It has distinct biological and cultural characters, but its relation to pneumonia is probably that of a mixed infection only.

3. Other bacteria, such as the typhoid, colon, diphtheria, proteus, influenza, plague, and pyogenic organisms, have been found.

Indirect Causes.—(*a*) *Age.*—Pneumonia may be seen even in the new born. Predisposition to infection is great up to the sixth year, and then much less until the fifteenth year. After the fifteenth year its frequency increases after each decade. Over half the cases of pneumonia occur between the twentieth and fortieth years, and over 80 per cent. between the tenth and fiftieth years. (*b*) *Sex.*—Eighty per cent. of cases are males. The relation to trauma is in doubt, though contusions seem an occasional accessory factor. (*c*) *Cold.*—Cold and wet are predisposing factors. Pneumonia prevails especially in February, March, and April, when the weather varies, the temperature is low, the moisture is great, and the winds are high. Three-quarters of all cases of pneumonia occur in cold months. It is probable that dust disseminates the dried sputum. Pneumonia is half again as frequent in the cities as in the country. In the winter of 1903–4, when 4,000 persons died of pneumonia in Chicago, the author knew of practitioners in many towns not one hundred miles distant, who did not see a single case. (*d*) *Individual predisposition* is said to be a *more important factor than in any other acute infection.* Susceptibility to pneumonia is increased by debility, overwork, and alcoholism, though the disease also attacks the most robust individuals.

General Clinical Course.—After a very *short incubation* of a few hours to a day, the *disease begins suddenly.* In infants it often begins with convulsions—in the young with vomiting, and in older subjects with a severe chill, which often occurs in apparently perfect health. On the *first day* there is a sudden rise of temperature; the face becomes red, even livid; the eyes are injected; the pulse and res-

PLATE IV



The Diplococcus Pneumoniæ, Stained with Methylene Blue and Fuchsin as a Counterstain. Taken from the Sputum of a Case of Acute Croupous Pneumonia. (Simon.)

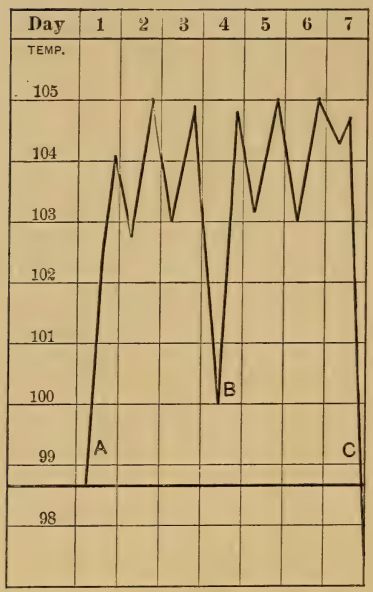
piration are accelerated; the patient lies characteristically flat in bed; there is much depression; thirst, headache, and a sudden, sharp pain in the side develop, accompanied by cough and increased respiration. On the *second day,* the cough, pain, respiration-rate and headache increase, with dilatation of the alæ nasi; the expiration is short and grunting, and speaking is difficult; leukocytosis develops. The sputum appears, or, if already present, becomes bloody; this, with the sudden fever, chill, pain, and increased breathing, practically establishes the diagnosis before signs of consolidation appear. On the second or *third day* there are evidences of lung consolidation, such as tympany which is soon followed by dulness, the crepitant rale, and bronchial breathing; high temperature develops with slight morning remissions; the skin becomes dry and red; there is increased pulse and respiration; herpes, anorexia, coated tongue, constipation, moderate delirium, febrile urine, and coughing attacks develop. The coughing is frequent but short in duration, voids a viscid, red sputum, and causes catching respiration, pain and broken sleep. The prognosis cannot be made from the symptoms up to this time, since the clinical course may be unfavorably modified (*a*) especially by collapse due to myocardial or vasomotor paralysis, or to embolism of the lung from right-heart dilatation; (*b*) by profound toxæmia; (*c*) by delirium tremens; or (*d*) by death, though at this stage it is rare. In favorable cases, when the toxæmia is spent, the crisis occurs, usually on the uneven days (the fifth, seventh, or ninth) with gradually decreasing dulness and disappearance of other local findings. A "critical" fall of temperature, pulse, and respiration-rate, and relief of nervous symptoms, and sweating, result. Lysis is less frequent. As a general statement, the *toxæmia is the dominant feature* of pneumonia, and the consolidation is decidedly secondary. Furthermore, the degree of toxæmia has no necessary or constant relation to the amount of consolidation, just as the toxæmia of typhoid does not depend on the presence or degree of the intestinal changes.

Analysis of Individual Symptoms.—1. Onset.—In over 75 per cent. *the onset of pneumonia is sudden. Chill,* convulsions, vomiting, and *fever,* after a short incubation, *usually mark the brusque onset.* Repeated chilly sensations, occurring when the heart is weak, do not as accurately designate the onset as does an initial rigor. A chill is present in 75 per cent. of the cases, and in no other acute disease except malaria is the chill so severe and constant; it lasts from a quarter to half an hour. The *onset is atypical* in senile pneumonia, and in pneumonia secondary to other diseases—*e. g.,* delirium tremens, typhoid, variola, and psychoses—the chill, cough, dyspnœa, pain, and sputum are often absent, so that the existence of pneumonia may not be suspected. *Prodromes* occur in 25 per cent. of cases (Grisolle), mostly in men between thirty and forty years, and rarely last more than five days; anorexia, depression, headache, bronchitis, backache, and low fever may be noticed. In one of the author's cases a

pneumococcus infection began in the throat, then invaded the larynx, and only after three weeks invaded the lung. In four cases, low fever and hæmoptysis for several days suggested tuberculosis.

2. Fever.—The usual course of the fever is, first, a *very sudden rise,* especially in menstruating women; the fever is then *continuous,* often with not more than 1° or 2° variation; in three-quarters of the cases the maximum fever is between 102° and 105°. It may then remit, which is of good prognostic import; remittent fever throughout the disease occurs in some cases. Recrudescences occur when consolidation passes from one lobe to another. The fever is lower or even absent in the aged, in alcoholics, and in cases of secondary pneumonia. Fever produces a marked loss of weight (twenty to thirty pounds). The rise preceding the crisis (*perturbatio critica*) may be mistaken for an ante-mortem rise. In both conditions there may be delirium, convulsions, chill, or an alarmingly irregular pulse. A pseudocrisis may deceive the physician, the fever resuming its original elevation. *Crisis* occurs in 80 per cent. of cases, usually on uneven days, as noted by Hippocrates, —the fifth, seventh, ninth, or eleventh days,—and usually between evening and early morning. It very rarely occurs on or before the third day. It marks the end of the toxæmia but not of the consolidation which requires several days more. In six to ten hours the temperature falls to or below normal, accompanied by sleep, euphoria, lessened pulse and respiration, lateritious deposits and reappearance of the chlorides in the more abundant, limpid urine, and "critical" sweating or even diarrhœa. The fall may measure even 10° or 11° (and the pulse may reach 30). Lysis occurs in 20 per cent. of cases. It is likely to occur when the fever lasts more than ten days, but lingering fever may indicate progression in other lobes and such complications as empyema or endocarditis. Lysis may be gradual and continuous or slow and remittent.

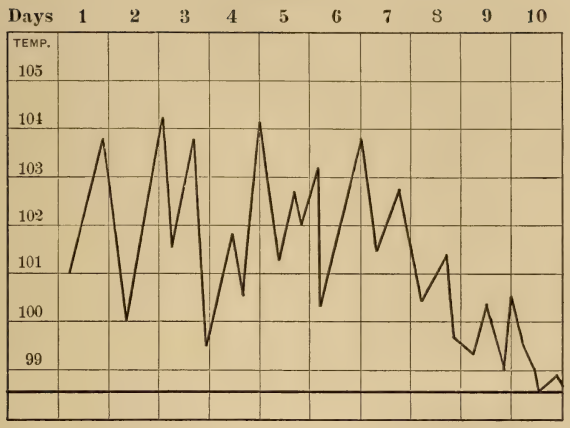
Fig. 13.



Fever-curve of typical case of pneumonia: A, sudden rise; B, pseudocrisis; C, crisis.

3. Respiratory Findings.—(1). *Pain.*—Pain in the side is generally pleural and its presence *in 90 per cent.* of cases makes it of diagnostic value, since it is the most frequent respiratory symptom. It appears early, is increased by coughing or motion, and is sharp and sometimes agonizing. It is usually well localized over the con-

FIG. 14.

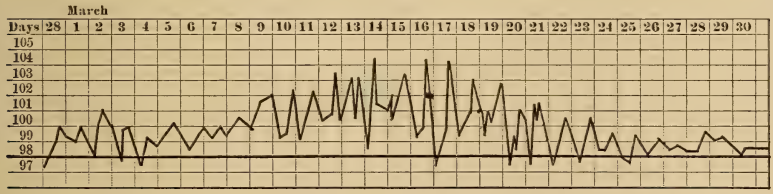


Pneumonia; remittent (subcontinuous) fever.

solidated focus, but in 8 per cent. is referred along the lower intercostal nerves to the abdomen, in children especially, and sometimes in adults. Appendicitis or cholecystitis may be thus simulated. Sometimes the pain is located on the opposite side of the chest. Pain is absent in central, secondary, and upper-lobe pneumonias; in the aged, and often in children; and in cases with great delirium.

(2). *Dyspnœa.*—Dyspnœa is frequent; it is caused by pain and especially *toxæmia,* but also by cardiac weakness, diminished respiratory area, bronchitis, and complicating pulmonary œdema.

FIG. 15.



Pneumonia fever curve; exceptionally protracted though uncomplicated fever.

(3). *The Respiration-rate.*—The respiration-rate is practically always *increased* to 24 or 40 (or more in children); and in two-thirds of all cases, it ranges between 30 and 50; it almost never rises to 60 or 80 except in the death agony, but such frequency is common in miliary tuberculosis. The author once saw a rate of 96 with recovery; the pulse was 90. Dilatation of the nares is said to be ominous, but to the author seems common in ordinary cases. *Alteration of the pulse and respiration ratio* was first noted by von Jürgensen; the normal ratio of 1 to 4 becomes 1 to 3 or 2. In one personal instance with the entire right lung consolidated, respiration was 18.

(4). *The Cough.*—The cough always irritates, is sometimes dan-

gerous, and seldom relieves the patient. It is usually present from the beginning of the disease to the crisis, is due to the bronchitis and almost invariably occurs in lower-lobe pneumonia, but is often absent in upper-lobe consolidation, in terminal or secondary pneumonia, in cases where there is much delirium, in the aged, and the young.

(5). *The Sputum.*—The sputum is at first transparent, mucous, and very scanty. It soon becomes *viscid,* and is difficult to raise and to expectorate, as it adheres to the mouth and lips. It is so viscid that the sputum-cup may be inverted without any flow of the sputum. In two days, it becomes *red and rusty* (80 per cent. of cases). Later, the sputum becomes more abundant, averaging between one and seven ounces daily. Increased fluidity may rarely result from weak heart, or decreased coagulability of the blood. Rusty sputum is often absent in children who swallow their sputum; in cases of upper-lobe pneumonia, in which there is less hemorrhage, less consolidation, and less cough to dislodge exudation; and finally in the

FIG. 16.

Fibrinous coagulum from a case of croupous pneumonia (Bizzozero).

senile and secondary pneumonias, possibly because of an early gray hepatization. The cause of unusual color in the sputum, such as lemon- or brick-color, is unknown; it is sometimes observed in pulmonary œdema, or in incipient gangrene and renders the prognosis uncertain. Safranin color (*sputum croceum*) indicates resolution. The rusty sputum is of great diagnostic value,—*e. g.*, in the beginning of the disease or in central pneumonia—and generally is of good import. *Fibrin threads* or "casts" of the bronchioles are usually

seen between the third and seventh days. On shaking the sputum in water and catching the shreds on a slide they appear clubbed at their alveolar ends. They are not wholly pathognomonic, since Laennec found them in tuberculosis and fibrinous bronchitis. They consist of fibrin, white cells, fatty cells and epithelium. Thinner casts, analogous to the cylindroids of the urine, and spirals are sometimes found. Microscopically the sputum contains blood cells, various epithelia, and the *pneumococci* (found first by Wolff in sputum and by Weichselbaum in the lung).

(6). *Lung Pathology.*—Laennec (1819) was the first to accurately separate pneumonia from pleurisy. He described three stages. (1) The stage of *hyperæmia* is very exceptionally observed unless the subject dies in twenty-four to thirty-six hours, because hepatization (*v. i.*) develops rapidly; hyperæmia is most often seen at the edges of the consolidated lung where the pneumonia is beginning to extend. The lung crepitates less than normal and floats; it is red, voluminous, and tears readily, since it has lost its elasticity, and on section exudes a sero-sanguineous fluid. Under the microscope, congestion, serum, and diplococci in the alveoli are observed. The alveolar epithelial cells swell and desquamate, which, according to some observers, are the primary changes. (2) In the stage of *red hepatization,* the lung is evenly red, friable, airless (not wholly so in pneumonia of an upper lobe), and sinks when put into water. Its weight is increased ten to fifty ounces. The lung has marks of the ribs on its fibrin-coated surface and is enlarged to the size of deepest inspiration (Rokitansky, who developed the gross anatomy of pneumonia). On scraping the cut surface, *fibrinous* casts or *plugs* of the bronchioles are observed. The lung is *granular on section* (Laennec), which results from fibrinous exudation into the alveoli. These granulations are seen best by oblique illumination. Under the microscope, coagulated blood and exudate, alveolar epithelium, injected bloodvessels, emigration of leukocytes, diplococci, and *meshes of fibrin,* are noted. (3) In the stage of *gray hepatization,* the granulations are grayish-yellow—not red because the blood is absorbed; they are larger and looser than in the second stage. The connective tissue is grayer than the granulations, and is sharply defined by the lung pigment. It has been claimed that death always occurs if this stage is reached. If the patient lives, the granulations disappear; the lung exudate becomes semi-fluid (purulent infiltration) and can be washed out; there is a fatty emulsion, and resorption occurs by the lymphatic vessels and not by evacuation through the sputum. Proteolytic enzymes (ferments), probably derived from the leukocytes, dissolve the exudate; the products of this autolysis are excreted by the kidneys.

Seat of pneumonia: Lichtheim's claim that the process begins at the hilum and extends to the pleura, has been proven by the *x*-ray. If it remains at the hilum it is called central pneumonia (1.7 per cent.); if the entire lung is involved, it is termed pneumonia totalis or massiva; the terms "double pneumonia" and "crossed pneumonia"

are self-explanatory. The lobes are involved in the following order of frequency: (1) Right lower lobe; (2) left lower; (3) right middle; (4) right upper; (5) left upper lobe. Jürgensen's collation of 16,614 cases of pneumonia shows that the right lung is affected in 53 per cent., the left in 37 per cent., and both lungs in 10 per cent. Fränkel found the upper lobes involved in 16 per cent. and the lower in 84 per cent.

(7). *Physical Signs.*—The physical signs develop in the lungs only after twelve to twenty-four hours, or later.

a. Inspection.—The decubitus is variable. It is often dorsal in lower-lobe, and erect in upper-lobe pneumonia; the patient at first lies on the sound side to avoid pain; on the diseased side later, for better expansion of the sound lung. The spinal column is concave toward the diseased lung for its better immobilization. The *excursion is less on the diseased side* because of pleuritic pain, and Litten's diaphragmatic sign is absent and the sound lung is vicariously active. The diseased side is larger by one inch. The accessory muscles of respiration are active and dilatation of the nares is seen. [Systolic pulsation of the lung occurs when the lingual lobe is consolidated, or, in the first stage, when the heart beats against semifluid exudation, Graves.]

b. Palpation.—This detects the *lessened excursion* more readily than does inspection; *vocal fremitus is usually increased* because of better sound propagation, fremitus being normally greater on the right than on the left side. It is decreased (i) by occlusion of the bronchus by fibrinous plugs, which are, however, usually dislodged by having the patient cough; (ii) by coincident pleural effusion; (iii) by great consolidation (Gerhardt), in which the chest wall becomes tense. Pleural friction may be felt.

c. Percussion.—(i) A *tympanitic note* is obtained in the first stage (Skoda's resonance), caused by decreased elasticity of the lung and therefore decreased tension and amplitude of vibration [due to the swelling of the alveolar epithelium and turgescent capillaries]. This condition may last as long as two days, and causes doubt as to the side involved. Careful watching of the tympany often leads to detection of extension, which is shown by relaxation of the lung tissue contiguous to areas already consolidated. Tympany in the later stages is not uncommon in upper-lobe pneumonia where exudation is usually incomplete, or in moderate compression,—*e. g.*, of right upper lobe when right middle and lower are consolidated. It is rare in lower-lobe pneumonias. Tympany may remain days or weeks after the crisis. (ii) *Dulness* is usually *found on the second day* and is the percussion sign of the second stage. When complete, it corresponds to the lobe confines, which are outlined in Fig. 33 under Pleurisy. The dulness is not absolute. It is usually first detected in the posterior axillary line in lower-lobe pneumonia and generally is more complete behind than in the front of the chest and more in a lower- than in an upper-lobe involvement. Dulness

is difficult to detect if the area is not at least 1 by 2 inches, or if it is deeper than 2 inches from the surface. It is absent in central pneumonia. It disappears with resolution to be replaced by tympany. Wintrich's palpatory percussion is very valuable. [Wintrich's change of pitch (on opening the mouth) is heard in upper-lobe pneumonia, where the percussion wave is conveyed to the bronchus more often than in lower-lobe pneumonia. Cracked-pot resonance over the edge of the consolidation bordering on sound tissue is due to irregular tension.]

d. Auscultation.—Decreased breathing is often noticed at first (Grisolle). Stokes diagnosticated the first stage by sudden local puerile breathing with fever and disordered breathing.

First Stage.—The *crepitant rale* of Laennec remains the auscultatory sign of the first stage; but, as Laennec stated, it is not absolutely specific, because it occurs also in tuberculosis, lung-œdema, infarct, hypostasis and bronchiolitis. It probably has its origin in the viscid exudate in the finest tubules and in the alveoli. It is coarser in the aged and in the children because the large tubes are more involved. It is heard at the height of inspiration as a succession or shower of delicate crackles. When not heard, a coughing effort may elicit the crepitant rale. It often disappears after a number of deep inspirations; *e. g.,* when it is demonstrated to a class, only the first students hear it. Dislodged plugs may occasion expiratory rales. The crepitant rale disappears during the second, and reappears in the third stage.

Second Stage.—The distinct, intense *bronchial breathing* is due to increased transmission of the breath sounds through the solidified lung. It sounds as though nothing intervened between the lung and the stethoscope and is especially clear and near when the ear is placed directly on the chest. It is absent or partial in central pneumonia, especially anteriorly, in coincident pleurisy, and in obstruction of the bronchi by mucus or fibrinous casts. In the latter case, several coughs usually loosen the obstruction and restore the bronchial breathing. *Bronchophony,* due to the voice sounds being better transmitted through the consolidated area, is heard when the patient counts or speaks, but especially after each involuntary expiratory grunt. *Ægophony* is occasionally heard, as well as Baccelli's phenomenon (auscultatory whispering). The crepitant rale disappears in this stage, except at the edges of the hepatization where extension occurs.

Third Stage.—The crepitant rale reappears,—the rale redux.

4. Circulation.—(*a*) The *pulse* rate is usually 100 to 120 or higher in children. It is also rapid in the aged, and in severe *toxæmia.* In adults, a rate of 130 to 140 makes the prognosis less favorable. If the rate is slow at the height of the disease, grave heart or brain complications are indicated (Traube). Irregularity and gallop-rhythm (one systolic tone and two diastolic tones) are of poor prognostic import. Antecritical irregularity frequently occurs. The pulse is usually deceptive and its fulness is of no value in prognosis.

It is not often dicrotic. Extensive pneumonias may compress the subclavian artery, making the pulse smaller in one wrist than in the other. (*b*) In the right *heart,* dilatation from toxæmia may occur; the second pulmonic tone is accentuated but this is not observed when the right heart becomes greatly weakened. There may be functional bruits especially in children. In very rapid cardiac action, the two heart tones sound alike, resembling the fœtal heart tones (embryocardia). (*c*) *Collapse* is usually due to myocardial or to central *vasomotor paralysis* and may occur at the onset, acme, defervescence or convalescence. (*d*) *The blood* shows anæmia, which may explain some cases of collapse. The fibrin increase is readily noted in fresh blood preparations. A polymorphonuclear *leukocytosis* of 15,000 to even 100,000 appears early, and lasts slightly longer than the fever. The leukocytes ran between 15,000 and 30,000 in 75 per cent. of Cabot's counts. The eosinophiles are decreased or absent until the infection reaches its height whence the favorable significance of their reappearance. The lymphocytes are decreased. During convalescence many myelocytes appear. The white count remains high in delayed resolution or complications. A low count is generally unfavorable. *Pneumococcæmia* (pneumococci in the blood) was, until recently, considered of bad prognostic import, but it is of no great prognostic value. Rosenow found pneumococci in blood cultures in 93 per cent., and in smears in 42 per cent., most abundantly on the fourth day, and few just before or at the crisis. Prochaska found pneumococci in 100 per cent., when two or three drams (10 c.c.) of blood were taken for cultivation and mixed with 98 or 99 parts of bouillon. Pneumococcæmia has been found before hepatization. Rosenow noted increased agglutinative power of the serum until the crisis occurs.

5. The Digestive System.—There is the febrile tongue, thirst, and anorexia. Vomiting occurs in 25 per cent. of cases and results from cerebral toxæmia, coughing or pharyngitis. The distressing, sometimes dangerous tympany and the occasional diarrhœa are toxæmic. Constipation is the rule. There is often intumescence of the liver. Fränkel found splenic tumor in 15 per cent. of all cases and in 40 per cent. of fatal cases.

6. The Skin.—The red cheek on the affected side was known to Aretæus. The skin is usually dry, but miliaria and sweating may be seen, especially in children. *Herpes* is found more often than in any other disease (30 to 50 per cent.). It appears from the third to fifth day, chiefly on the lips, nose, cheek or ear; Howard found causal changes in the Gasserian ganglion. Herpes is of diagnostic value, *e. g.,* from typhoid, and is generally of favorable import. Rarer eruptions are erythema, roseolæ, purpura or pemphigus. The lymph-glands are enlarged in 25 per cent. of cases.

7. The Urine.—Characteristic fever urine is found, and the urea is increased threefold after the crisis from over-production and accumulation during the fever and from the resolving exudate. The

chlorides are decreased from 225 grains daily to 30 grains, or they may be absent until the crisis or later, when they reappear in increased quantities (Rottenbacher); no satisfactory explanation for the decrease of the chlorides is known. Pick notes that the urine often becomes neutral or alkaline two days after the crisis, due to the sodium deposit in the lungs. *Albuminuria and cylindruria* appear in about 50 per cent. of cases; peptonuria and acetonuria are not uncommon, and the diazo reaction appears in 20 per cent. Hæmaturia is found in 1.6 per cent. Nephritis seldom occurs (1 to 2 per cent.), and uræmia is very uncommon. Pneumococci have been found in the urine and glomeruli (Koch, Nauwerck).

8. The Nervous System.—*Headache* is constant. There may be insomnia, restlessness, or stupor, and the latter is always ominous. *Delirium* (22 per cent.) occurs in adults, with upper-lobe pneumonia, alcoholism, or marked toxæmia. Delirium and other nervous symptoms resulting from exhaustion may occur even a week after the crisis (2.5 per cent.). Venesection in former times greatly increased the percentage of delirium. The patellar reflexes are often absent and the pupils may be dilated or sluggish. Convulsions, coma, and rigid neck, in children, result from toxæmia of the more sensitive young brain, from complicating otitis, from brain œdema, or from actual meningitis. Hemiplegia may result from brain œdema or anæmia, or arteriosclerosis and weakened heart.

Mechanism of Death in Pneumonia.—Death is rare in the first three days; within this time, its cause is usually some antecedent malady such as nephritis or typhoid. (1) *Toxæmia is all-important in its action on the heart, brain, and vasomotor nerves.* Death ensues with a rapid pulse, rapid respiration, algid extremities, and obtunded sensorium. (2) *Pulmonary œdema* is either mechanical (cardiac) or inflammatory—*i. e.*, the first stages of a developing pneumonia. (3) The *extent of pneumonia* is the least important factor. A massive pneumonia in a robust person causes less disturbance than a very small patch in a nephritic patient. There is no parallelism between the area hepatized and the resulting toxæmia. (4) *Pneumococci in other tissues, e. g.,* pneumococcic meningitis, endocarditis, or pericarditis.

Complications.—These are few but severe.

1. Pleurisy.—Dry pleurisy is so constant that the name of pleuropneumonia is rather justified, the pleura being covered with a very thick, tenacious, curdy exudate. It is usually due to the pneumococcus; if due to the streptococcus, it is a complication of great danger. Effusive pleurisy is said to occur in 5 to 15 per cent., though Fränkel saw serofibrinous pleurisy in but 1 per cent. in 1,000 cases. Maragliano aspirated his pneumonic patients and found some fluid in 66 per cent. Pleuritis is para- or metapneumonic, serofibrinous or purulent (2 per cent.). The presence of leukocytosis in a pleurisy may indicate a previous unobserved or overlooked pneumonia. Pneumonia may exist on one side and pleurisy on the

other (Traube). The author has seen two such cases. The prognosis is not necessarily unfavorable, even when operation for pneumococcic empyema is refused. Interlobular empyema is most serious, because it so often escapes detection. Pneumococcic empyema occurs in 2 per cent. of cases of pneumonia and is notable since the pneumococcus so seldom provokes suppuration in the lung. Its symptoms are discussed under pleurisy.

2. Endocarditis.—Acute endocarditis complicates pneumonia in 1 per cent. (Chvostek) of all cases and in 5 per cent. of fatal cases; autopsy figures are higher than figures in general practice. The pneumococcus is found in the vegetations. The *valvular exudate* is voluminous, though *ulceration,* valvular aneurysm and perforation are less common than in staphylo- and streptococcic forms. Endocarditis develops especially on old valvular lesions in the left heart and most frequently on the aortic valves (Netter, Heschl). The right heart is involved three or four times as frequently as in endocarditis due to other bacteria. Sixty per cent. of cases are associated with *meningitis.* Middle-aged adults are more often affected than the young, and women more often than men. Symptoms may be absent. Endocarditis is probable when a murmur, systolic or particularly diastolic, occurs with irregular and protracted fever, chills, sweats and embolism (50 per cent.). Symptoms may directly follow the pneumonia or develop after one-half to one week of normal temperature. The *endocarditis is malignant in 80 per cent.* of cases; in the balance, milder endocarditis occurs; 25 per cent. of all ulcerative endocarditides are pneumococcic. Fever is sometimes, and leukocytosis frequently, absent. (*Vide* Ulcerative Endocarditis and Septic Infections.)

3. Pericarditis.—Some figures place its post-mortem frequency at 5 to 15 per cent. but Fränkel estimates it at seven-tenths of 1 per cent. About 60 per cent. of cases are latent because the pericardial rub is frequently absent. Dry pericarditis occurs more often in children than in adults and, in all cases, chiefly at the height of the disease. Effusive pericarditis is less common than the fibrinous form. It is more common when the left lung is involved. It is dangerous though not necessarily fatal.

4. Arteritis and Thrombophlebitis.—These are far less frequent than in typhoid, but nearly every clinician encounters a few cases. Steiner collected forty-eight cases of venous thrombosis (1903); it most often develops in convalescence and heals usually (75 per cent.) though early embolism or permanent venous obstruction may develop.

5. Nervous Complications.—The chief nervous complications are delirium tremens, meningitis, or, less frequently, Landry's paralysis, cerebral embolism, encephalitis, neuritis, etc. *Meningitis,* first observed by Immermann and Heller, occurs in 5 per cent. of autopsies, and in three-tenths of 1 per cent. of clinical cases. It occurs in asthenic patients, in upper-lobe pneumonia, and often accompanies

endocarditis. One of the author's cases died at dinner a week after apparently complete convalescence, and the autopsy showed a quarter inch of pus over the convexity. The usual meningitic symptoms develop if infection reaches the base of the brain, but this is not usual [see Meningitis]. Lumbar puncture determines the diagnosis. The prognosis is considered absolutely unfavorable, yet Achard and the author have seen recovery.

6. Alimentary Complications.—These are few. Croupous gastritis and enteritis, parotitis or peritonitis, are not frequent. Dieulafoy described an ulcerative gastritis, caused by pneumococcic embolism and attended by gastrorrhagia. Icterus in frequency averages 1 per cent., though Chvostek saw it in 21 per cent. Its causes are (*a*) lessened excursion of the diaphragm; (*b*) hepatic stasis; (*c*) duodenal catarrh; (*d*) hæmatogenous jaundice resulting from absorption of the hemorrhagic exudate or toxæmic solution of the red blood-cells (hæmolysis). (See Pneumococcic Peritonitis.)

7. Lung Complications.—Suppurative mediastinitis was first observed by Weichselbaum, who regards it as the connecting link between pneumonia, meningitis, and pericarditis. Pulmonary induration (cirrhosis of the lung) may follow incomplete resolution, especially after repeated attacks of pneumonia (Charcot); it is usually febrile. Abscess is not frequent (5 per cent., Laennec, or once in 1,200 cases, Fränkel). Skoda emphasized the uncertainty of its signs and the difficulty in its diagnosis, for pus and elastic fibers in the sputum, and less frequently cholesterin and hæmatoidin crystals may be the sole signs, and continual coughing may be the chief symptom. Suppuration may result from the pneumococcus or from the pyogenic bacteria proper. *Gangrene* occurs in 1 to 2 per cent. of the cases. Clinically it is recognized by the horribly fetid sputum. Abscess and gangrene are more fully treated under Diseases of the Lungs.

8. Joint Complications.—Genuine rheumatism may occur with, before, or after a pneumonia. Ninety-two cases of pneumococcic arthritis (pseudorheumatism) were reported by Howard (1903). The arthritis may be serous or purulent. The knee is affected in 30 per cent. Arthritis is monarticular in 62 per cent. and fatal in 65 per cent. of cases (Herrick). The infection is hæmatogenous. Fränkel found the pneumococcus constantly in the bone-marrow.

Clinical Types and Variations.—These are as follows:

1. Abortive Pneumonia.—This type is recognized by the rusty sputum, fever and respiratory disturbance, and produces no hepatization.

2. Ephemeral Pneumonia.—This type is characterized by one day of fever, and very rapid resolution. In this type, the onset is brusque and a severe disease seems imminent but the symptoms abate surprisingly soon.

3. Relapsing Type.—After fall of the temperature, the entire cycle recurs (in 0.3 per cent., Wagner). Recurrent type (*v. s.*, Immunity).

4. Local Variations.—Apex pneumonia is often more severe and more atypical in symptoms and signs; it is frequently adynamic, and pulmonary and cerebral complications are rather more common. Migratory pneumonia travels from one part to another (the so-called erysipelas-pneumonia). Central and massive pneumonia (pneumonia totalis, affecting an entire side) will be considered under diagnosis.

5. In Children.—An abrupt onset occurs without chill before the seventh year, but with vomiting (50 per cent.) or convulsions (7 per cent.) and other cerebral or meningeal symptoms. Cough is often absent, no sputum appears, and the pain (third to fifth year) is referred to the abdomen but commonly without abdominal tenderness. Pain is a more important symptom than increased respiration, which occurs in any fever. The respirations may number eighty in cases under two years of age; in older children, fifty. Crepitant rales are usually absent because of the superficial breathing caused by pain. Latency of physical signs, such as absence of bronchial breathing, bronchophony or dulness, is sometimes noted. Localization is more often central, apical, or migratory. The fever may rise more slowly and lysis is more frequent than in adults. Rivière found croupous pneumonia as frequently (or more frequently) in children under two years as in those above two years. In 118 cases reported by Morse, 50 of them occurred in the first, and 68 in the second year of life.

6. In the Aged.—Pneumonia is frequently latent, insidious, and asthenic, with ill-marked signs, without chill, even without fever, and often without cough, pain, or sputum. It is strange that so severe an infection can exist with so few of its usual evidences. In this and the following type, pneumonia is declared by the physical findings only.

7. In Alcoholics.—The same latency or lack of type may be noted. In alcoholics the delirium tremens masks the lung lesion.

8. Asthenic Pneumonia.—In asthenic, "typhoid," or "toxic" pneumonia, toxæmia is dominant, there are high fever, enlarged spleen, albuminuria, icterus, mixed infections, severe complications, such as meningitis or gangrene, and high mortality. These cases contrast strongly with what we might call "walking pneumonia." The author had two patients who worked throughout the disease.

9. Pneumonia Secondary to Other Diseases.—Pneumonia may be secondary to emphysema, arteriosclerosis, hepatic cirrhosis, nephritis, diabetes, tuberculosis, typhoid, or other conditions. Anæsthesia by ether is thought to predispose to pneumonia by irritation of the respiratory tract. The nose and mouth should be cleansed before anæsthetization to remove native germs. The frequency of ether-pneumonia in abdominal operations suggests the possibility of embolism as a factor.

Various microörganisms are found—colon bacillus, streptococcus, etc. This type is commonly misinterpreted because it is atypical, ill developed, or masked by the primary disease. Pathologically, the

hepatization is rarely typical and is more lobular than lobar; the lung section is smooth and moist, and the exudation is more cellular than fibrinous.

10. Delayed or Imperfect Resolution.—Normal resolution is chiefly effected by lymphatic absorption and not by the sputum. (*a*) Resolution may require weeks or even months, especially in apical involvement, and it more often follows lysis than crisis. The toxæmia disappears but dulness and bronchial breathing persist. (*b*) Imperfect clearing up of the lung may be caused by abscess formation, old pleuritic adhesions, fresh pericarditis, or inflammatory involvement of the connective tissue and lymphatic vessels. Induration of the lung by organization of the exudate is usually febrile (Fränkel). (*c*) The so-called pneumonia migrans is pneumonia which slowly progresses from one part to another.

Diagnosis.—Mistakes are rare in the diagnosis of primary, frank pneumonia in adults—they occur mostly in cases of aged individuals, alcoholics, children, and pneumonia secondary to cancer, nephritis, diabetes, etc. Therefore, all cases with chronic disease must be closely examined and the back percussed—when the respiration, pulse, and temperature rise, or when, without these or other symptoms the subject is not doing well.

1. Onset Symptoms.—The chill in adults, or convulsions and vomiting in children, suggest the possibility of pneumonia. Chill, pain in the side, sudden high fever, and rusty sputum, are quite final, because pneumonia is practically the only disease in which they occur, except infarct of the lung—in which the same symptoms may obtain, but in which the sputum is reddish, not viscid, usually darker, and far more abundant. In 25 per cent. of cases the onset of pneumonia is not typically brusque and the physical findings of pneumonia are present without a suggestive history; indeed it may rather be suggestive of simple pleurisy, grippe, etc. The prodromes of pneumonia are too often disregarded by physicians. The prodromata may last two to seven days.

2. Diagnosis from the Physical Findings.—The physical findings are usually detected in the typical case upon thorough examination, and as a rule usually appear within two days after the characteristic symptoms of onset. They are the well known lessened expansion on the side of the lesion; increased vocal fremitus; distinct dulness; bronchial breathing; the crepitant rale; the grunting, abbreviated, expiratory "huh" sound, and a bronchial tone to the whispered or louder voice sounds. Typical as are these simple findings in their usual grouping, and easily recognized as they are when the student or practitioner has once heard and learned them, nevertheless many conditions modify their classical appearance and character. Thus the fremitus is decreased in massive consolidations which render tense the chest wall, in coincident pleural effusions or in fibrinous plugging of the bronchi. The latter may, however, usually be dislodged by repeated coughing. The dulness is found only in hepa-

tization which is nearly complete and is both preceded and followed by a tympanitic note. The crepitant rale is found only in the first and third stages, but in almost every case can be found in some part of the affected lung. Bronchial breathing is rudimentary or absent in incomplete hepatization, is often so in upper-lobe pneumonias, in complicating effusive pleurisy, and in obstructed bronchi. These physical signs are pathognomonic only in connection with the cyclic course, onset, disturbed pulse and respiration ratio, pneumococcæmia, diminished chlorides, herpes, leukocytosis and crisis.

3. Localization.—Massive pneumonia (total pneumonia of one side) not infrequently simulates pleurisy, because fibrinous plugging of the bronchi is not uncommon, and prevents bronchial breathing, bronchophony, and the crepitant rale; and again, the fremitus may be absent together with some slight luxation of neighboring viscera. Consideration of the onset, the later cyclic course, and the dislodging of the plugs by forcible coughing, almost invariably determine the diagnosis. Too much is generally said about central pneumonia. It exists beyond all doubt, but conservatism is necessary in its diagnosis. All pneumonias are central in their incipiency but rarely remain so. The author demonstrated two cases in which the symptoms were pneumonic, but the local findings did not appear for a week.

4. In Children.—Pneumonia in children may be mistaken for abdominal disease, pleurisy, or brain affections. The "cerebral pneumonia" of Rilliet and Barthez may be (*a*) convulsive, occurring under two years, with fever, vomiting, and convulsions; but the evenly high fever is very suggestive even if the cough and dyspnœa are obscured by the brain symptoms; or (*b*) meningeal, occurring between the second and sixth years; but the pulse is not slow, and there is no deep breathing, nor other signs of meningitis.

5. Differentiation from Pleurisy.—The differentiation is usually easy, but confusion is unfortunately common. (See page 145.)

In *lung tumors,* bronchial breathing, bronchophony, dulness, red (or green) sputum, are also noted, but the history and course differentiate. In *acute tuberculous infiltration,* the course differentiates it from upper-lobe pneumonia in which cough, sputum, and typical physical findings are absent. Many mistakes in both directions are made, simply by forgetting that pneumonia may occur in the apices, in which the incomplete exudation may impart a tympanitic note, and that the breath sounds are not always typically bronchial, at least anteriorly. In some cases tuberculous pneumonia may commence like lobar pneumonia (see Acute Pneumonic Form, under Course of Pulmonary Tuberculosis).

Prognosis.—Pneumonia is the most fatal acute disease and even has a greater death-rate than tuberculosis. In 1903, 4,629 persons died of pneumonia in Chicago, and, in 1904, 8,360 in New York City. From 1851 to 1860, the mortality, in Chicago, from tuberculosis, was 35 per cent. more than from pneumonia. From 1891

to 1901, the pneumonia death-rate was 9 per cent. more than the tuberculosis mortality.

PNEUMONIA——*vs.*——PLEURITIC EFFUSION.	
Onset sudden, with rigor, vomiting, herpes.	*Gradual;* chilly sensations rather than rigor.
Friction less common; may be present.	*Usually,* at some time in the clinical course.
Cyclical short course; high fever, crisis.	*Longer* duration, lower fever, lysis. Often tuberculous foci at apex, etc.
Sputum rusty.	*Mucous,* if any.
Leukocytosis.	Usually *absent,* unless purulent or complicating a pneumonia.
Ectasia absent.	*Present.*
Fremitus increased (coughing usually dislodging bronchial plugs).	*Absent* over effusion unless adhesions bind the lung to the chest wall; increased above over compressed lung or in the interscapular region.
Dulness, preceded and succeeded by tympany (tympany or partial dulness in upper-lobe pneumonia is not uncommon).	*Flatness,* increased downward; entering Traube's space on the left side. Level seldom shifts with change of position. (See Fig. 33.)
Crepitant rales, bronchial breathing (unless bronchus is plugged), and bronchophony.	*Decreased or absent respiratory murmur;* bronchial breathing in interscapular region from compression, or even through the fluid; ægophony.
No essential luxation of heart, liver or spleen.	*Luxation* of viscera named, is usual in large effusions.
Paracentesis negative.	Tapping nearly always decides doubtful cases; *fluid found* beneath the flat area.

There are various prognostic factors: 1. The VIRULENCE of the pneumococcus, which varies from year to year (5 per cent. mortality in one year, 31 per cent. in another, Brandes). The mortality of 85,000 cases in the German army was $3\frac{6}{10}$ per cent., but these were necessarily selected cases. In hospitals the mortality ranges between 20 and 40 per cent., from alcoholism, excesses, and overwork. In 465,400 collected cases, E. F. Wells found 20 per cent. the average death-rate.

2. AGE.—According to Fränkel, the mortality is; one to five years, 30 per cent.; five to ten years, 3 per cent.; ten to twenty years, 5 per cent.; twenty to thirty years, 8 per cent.; thirty to forty years, 25 per cent.; forty to fifty years, 39 per cent.; fifty to sixty years, 43 per cent.; sixty to seventy years, 53 per cent.; seventy to eighty years, 87 per cent. The aged succumb readily; long ago Cruveilhier spoke of pneumonia, in this connection, as the most formidable scourge of the aged, and Peter characterized pneumonia as

the natural end of old people. Morse found the mortality in children 36 per cent. in the first year and 19 per cent. in the second; Revière, 25 per cent. in the first, 15 per cent. in the second year, and 2 per cent. in older children.

3. Sex.—The mortality is 50 to 100 per cent. higher in women; in pregnancy the disease is always severe.

4. Location.—Right-sided is more fatal than left-sided pneumonia, and upper- is more fatal than lower-lobe involvement. Extension from lobe to lobe is serious.

5. Unfavorable Conditions.—These are hepatic cirrhosis and nephritis, in which the death-rate is almost 100 per cent.; delirium tremens and inveterate alcoholism, in which it is 45 to 50 per cent.; cardiovascular disease, scoliosis, and high altitude.

6. Unfavorable Symptoms.—These are long prodromata, hyperpyrexia, enteritis, marked jaundice, albuminuria, severe pain, marked cyanosis, pulmonary œdema, diffuse bronchitis, and a white cell-count under 10,000. Sears and Larabee found the mortality 9 per cent. with the respirations under 30, 15 per cent. under 40, and 60 per cent. over 60 per minute; and 50 per cent. with the pulse over 130. Over 50 per cent. of delirious subjects die.

7. Unfavorable Complications.—Endocarditis and meningitis are almost always fatal; abscess, gangrene, and arthritis are always serious; pericarditis is fatal in 50 to 75 per cent. of the cases.

Strictly speaking, death is due in almost all cases to *cardiac toxæmia*. Mechanical overwork of the heart or diminished respiratory space are of small importance.

Treatment.—1. Prophylaxis.—Prophylaxis concerns disinfection of the sputum, cleansing of the room [See Smallpox], isolation, and maintenance of the physiological resistance. Cleansing of the nose may be of preventive importance.

2. Serotherapy.—Serotherapy has not proved successful, and pneumonia remains a self-limited disease.

3. Hygiene.—The hygiene is the same as in typhoid. Quiet should be maintained, the mouth, especially, should be cared for, and the patient should be clothed with a light flannel jacket open down the back. The *fresh air treatment* works wonders in pneumonia; the bed should be placed out of doors or the windows widely opened and the bed protected by screens; mental and cardiac symptoms, particularly, are greatly improved.

4. Diet.—The diet consists of milk, eggs, and cereals.

5. Symptomatic Therapy.—(*a*) *The Chill.*—The chill, which is rarely seen by the physician, is managed as in malaria.

(*b*) *Cough.*—Expectorants are unnecessary, and codeine gr. $\frac{1}{4}$, and carbonate of ammonium gr. iij, should be given every two to four hours. If these are ineffectual, morphine is indicated (*v. i.*).

(*c*) *Pain.*—The chest should be firmly strapped with adhesive plaster, applied in several oblique directions so that the pieces cross each other. Morphine does not essentially depress respiration but

rather lessens the irritability of the respiratory centre and relieves pain. Poultices can be more easily endured than ice, but, like blisters, leeches, and the Paquelin cautery, are superfluous. In pneumonia, general is far more important than local treatment.

(*d*) *Heart.—Care of the heart is the main objective* in treatment. Constant watching is imperative lest the delirious patient leave his bed, and it is a safe rule to consider all pneumonics delirious. Sitting up to drink, defecate, or urinate is never permissible, as pneumonia is a disease of sudden and alarming changes and there is constant danger of sudden death. No therapeutic measure which depresses the heart should be employed, and therefore *acetanilide, antimony, aconite, and pilocarpine are always to be avoided.*

Cardiac Stimulation.—Strychnia.—Strychnia is the best heart tonic, and should be given in every case because of the cardiac and vasomotor instability which often develops without warning. Vasomotor paralysis and collapse are attended by accumulation of blood in the abdominal vessels; thus, pallor of the retinal vessels follows section of the splanchnic nerve. In the collapse caused by acute toxæmia, the vasodilatation is general, but particularly concerns the deep abdominal vessels. Strychnine is here indicated, not only because of its general effects but because of its powers of vasoconstriction on the deep abdominal vessels; the vascular tonus in the skin and brain is not increased, whereby the brain vessels are flushed and the bulbar vasomotor and other vital centres are stimulated. The drug stimulates the central nervous system; the digestive tract, the secretions, and peristalsis; the circulation, the heart-muscle, and chiefly the vasomotor centre; and the respiratory centre.

Administration.—The dose is gr. $\frac{1}{30}$ to $\frac{1}{20}$, every two to four hours. (For dosage in children, see SCARLATINA.) Some strychnine remains in the system from three to eight days, though some is destroyed at once by the liver, and some is quickly eliminated by the kidneys. If the kidneys or liver are diseased or functionally tardy, as in old age, the drug may accumulate. Full doses sometimes produce nervousness, irregular heart, palpitation, or renal-vessel spasm, as does digitalis. Hypodermic administration gives less chance for the liver to destroy the drug than does administration by mouth.

Ammonia.—The ammonia preparations are of great clinical importance. It temporarily stimulates the heart-muscle, increases its frequency, and constricts the vasomotor system, thereby raising arterial tension. Its superficial effects are local flushing, subjective warmth, tension in the temples, a sense of exhilaration, and increased secretion of urine, sweat, and mucus. The prime indication for ammonia is sudden and functional cardiac adynamia, which occurs at the crisis. It is a local irritant to the skin, subcutaneous tissues, and the respiratory and alimentary mucosæ, and therein lie its therapeutic value and its clinical disadvantages. Its application to the upper respiratory passages or its ingestion by mouth causes a sudden reflex

stimulation of the respiratory and circulatory centres and initiates centripetal stimulation, which travels from the throat, œsophagus, and stomach, to the brain, long before the drug is absorbed. Untoward action is frequently observed in gastric, intestinal, renal and bronchial irritation resulting from overdoses, and also in undue irritation or necrosis following subcutaneous administration. The corrosiveness and fugitive action of ammonium carbonate are both very relative contra-indications, and are obviated by administration of small and frequent doses, gr. iij every two hours.

Digitalis.—Digitalis, in doses of 75 to 150 (!) grains, was recommended by Traube and Petrescu. Such administration simply proves, in the author's opinion, that the digitalis is not absorbed. It is claimed that exudation is modified and that leukocytosis is increased. Personally, the author has never seen digitalis produce as good results as strychnine, but he has repeatedly noticed toxic symptoms appear at the crisis when the repeated doses of digitalis were absorbed together. Digitalis should be used only as a last resort; ɱij to v of fluidextract should be given hypodermatically. The author believes digitalin is almost inert (see TREATMENT OF VALVULAR HEART DISEASE).

Alcohol.—Alcohol was first recommended by Todd for fevers. The chief interest centres in its action upon the circulation. For years it has been called a heart stimulant, though no pharmacologist now holds that alcohol stimulates the heart as do digitalis, strychnine, and camphor. Direct cardiac stimulation by alcohol *per se* is impossible, except by preparations like champagne which contain other ingredients. The frequency of the heart's action is increased, but it is known beyond all doubt that the drug acts as a vasodilator or as a blood distributor. Some clinicians refuse to employ it because it is narcotic to the nervous system and to the respiratory, circulatory, and vasomotor centres in the lower brain, or because it is a vasodilator. It must be admitted that alcohol has some good effects. It is effective in all varieties of sepsis. It protects tissues from excessive waste and is well tolerated in nearly all cases. There is no danger of cardiac collapse when it is withdrawn in convalescence. Jürgensen gives it in all cases, using red Bordeaux wine as a routine procedure, especially if cool baths are employed. If the heart becomes weaker, Bordeaux is replaced by the stronger sherry or port; then by rum, cognac, champagne, or whiskey in hot water, and tea or coffee, which can also be given by rectum. Alcohol is invaluable, whatever its action; it is second only to strychnine and is indicated by collapse or cardiac toxæmia, in which hot whiskey and water unquestionably stimulate by reflex action on the throat and stomach.

Opiates.—It is the author's belief that morphine is not sufficiently valued as a cardio-vascular tonic. Many withhold opiates even when the drug is indicated by maniacal outbursts, active delirium, and harassing coughing or pain, because it depresses respiration, and ill effects upon the circulation are feared. When used freely, it de-

creases the irritability of the respiratory centres, lessens cough, dyspnœa, the attendant headache and insomnia, and it almost invariably acts as a cardiovascular tonic. Its effects on diseased kidneys are not to be feared with ordinary care in administration, as little morphine is eliminated by the urine. Morphine will of itself help to sustain life, and in critical cases, where the author has watched its effects for hours, its hypodermic use has strengthened and regulated respiration, even when Cheyne-Stokes' respiration was marked. When the heart weakens, its force will often be renewed by morphine. It is a most valuable adjunct to the true cardiovascular stimulants, either those which have continued action, as strychnine and digitalis, or those whose action is more fugitive, such as camphor, ammonia, or caffeine, which latter has an excellent effect, given by rectum, in neutralizing the effects of opiates.

Camphor.—Camphor is an excellent diffusive stimulant for the heart and brain (grain i hypodermatically in 10 parts of sterilized oil, every two to four hours).

Saline Infusions.—These are indicated in profound toxæmia with weak heart. Two drams of salt in one quart of water are the rough proportions of the physiological salt solution. They are the "lavage of the blood" of French writers, who combine them with venesection. Although once abused, *venesection,* which has been employed since the time of Hippocrates, has again come more into vogue. It is valuable at the very onset in the robust for severe pain, active delirium, and urgent dyspnœa, and later when great engorgement of the right heart occurs. It rather promotes heart thrombosis and delays resolution. Heart stimulants must be used with phlebotomy. Continuous use of the *salt solution by rectum* is valuable.

Oxygen.—Oxygen is said to relieve dyspnœa, but recent studies prove that it is often irritating and injurious. The mouth-piece should not be held closely over the mouth, but merely near it.

Atropine.—Atropine is valuable in collapse with low temperature and clammy skin. The daily dosage should not exceed one-fortieth of a grain.

(*e*) *Antipyretics.*—A high temperature is not *per se* injurious. Cantani proved that patients with fever between 103° and 105° progress most favorably and that those with temperature above or below these limits have a more severe course. Von Jürgensen insists that all measures or remedies which *suddenly reduce* the temperature must be avoided. They are especially injurious at the crisis, when the heart sometimes staggers under the sudden fall of the fever.

Hydrotherapy is opposed by many, though Liebermeister lessened the mortality in his cases by 9 per cent. The tonic vasomotor effects are thought to outweigh the element of shock. It is less indicated in children and is contraindicated in the very obese, the weak, and the aged. Cool sponging with water and alcohol is the safest method. *Quinine* gives no antipyretic effect except in large doses; Aufrecht, Jürgensen and Sée give thirty grains at 6 P. M., thereby inducing a slow reduction of temperature without cardiac depression.

(*f*) *Nervous Symptoms.*—These are toxæmic, but should be considered with regard to; (i) fever; the greater the temperature variation, the more marked are the nervous symptoms; febrile nervous symptoms are most efficaciously treated by baths or cool affusions; (ii) anæmia, the delirium of which is relieved by morphine, coffee, alcoholic and saline infusions; (iii) deficient oxygenation, in which cold baths, cardiants, and oxygen inhalations are indicated; (iv) insomnia, which is treated by morphia, bromides, hyoscine hydrobromide, sulphonal, and cool sponging, but never by chloral; (v) delirium tremens (*q. v.*), in which rest is absolutely necessary and is best secured by the drugs named under (iv) and by alcohol.

(*g*) *Iodides and Cod-liver Oil.*—These should be given in delayed resolution.

DIPHTHERIA.

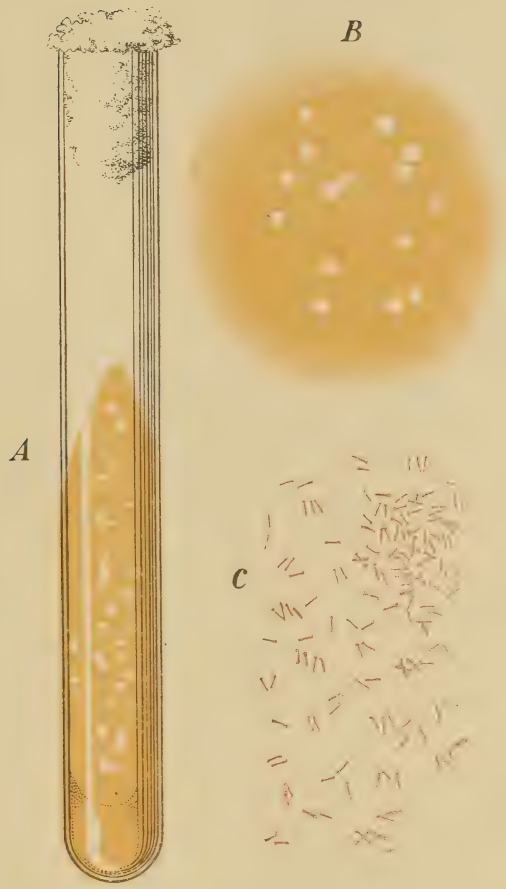
Definition.—An acute specific infective disease, which usually begins in the throat or upper air-passages, and is characterized by a local yellowish adherent membrane formed by the Klebs-Loeffler bacillus, and by constitutional symptoms due to toxæmia.

History.—Diphtheria was known to Hippocrates. Asclepiades, 100 B. C., performed the first tracheotomy. The name diphtheria literally means membrane and originated with Bretonneau (1821).

Terms and Restrictions.—True diphtheria implies a membrane formed by the Klebs-Loeffler bacillus whose absorbed toxins usually also produce toxæmia. There are, however, membranes not formed by this bacillus but due to the scarlatinal virus, streptococci, pneumococci, staphylococci, etc. While these are diphtheritic membranes in the old anatomical sense, they are not diphtheritic in the bacteriological sense. We must then term them *pseudodiphtheritic* or necrotic inflammation, or *diphtheroid.* Therefore, clinically, there are some discrepancies; (*a*) what seems diphtheria, anatomically, may prove not due to the Klebs-Loeffler bacillus; (*b*) some apparently simple throat inflammations, resembling angina or tonsillitis, may show no membrane or an atypical membrane, but bacteriologically are found due to the Klebs-Loeffler bacillus,—*i. e.,* are true diphtheria; (*c*) the diphtheria bacillus is found in 1 per cent. of perfectly healthy throats; (*d*) there are bacilli which closely simulate the diphtheria bacillus. The pseudodiphtheritic or diphtheroid membrane is most often due to the streptococcus and is seen oftenest in scarlatina, but also in typhoid, measles, Vincent's angina, syphilis and pertussis. It develops not merely in the throat, but wherever the true diphtheritic membrane may occur,—*e. g.,* the larynx, bronchi, eyes, skin, etc. As a working statement, most cases clinically resembling diphtheria are proved diphtheria by close and repeated bacteriological tests. G. Smith found the bacillus in 72 per cent. of 27,000 collected cases.

Bacteriology.—The diphtheria bacillus (described by Klebs, 1883, and cultivated by Loeffler, 1884) is 1.2 to 2μ long and 0.3 to 0.5μ

PLATE V



A. Culture of diphtheria bacillus.
B. Same magnified.
C. Diphtheria bacilli.

wide, with small round ends, or ends slightly enlarged. It stains readily by Gram's method, by Loeffler's methylene blue, and by carbol fuchsin. There are many irregular variations in form, such as branching types, but the serum culture always reveals the wedge- or mallet-like forms. Some are very long, others short and parallel. Their grouping is characteristic, like Chinese letters, in lines set asymmetrically at various irregular or slight angles. They are immotile and sporeless. Cultivated in all media, the diphtheria bacillus overgrows other associated bacteria on blood serum. On blood serum mixed with glucose bouillon, the culture is visible in eight to ten hours as yellow streaks, whose surface is slightly granulated and whose edges are somewhat thick. In twenty-four hours the growths have spread everywhere, with a wavelike surface, high, undulating edges, and a succulent, glistening appearance. The diphtheria bacillus is very resistant, and though it usually disappears in two or three weeks after the patient has shed the membrane, it may live for weeks or months in the throat (even six to fifteen months) or for a long period in clothing. It is found chiefly in the throat membrane, and is less frequently detected in the submucosa, heart (endocarditis), blood, lungs (bronchopneumonic foci), spleen, liver, brain, cord, kidney or urine. In one report, it was found in the blood and viscera eleven times in fourteen cases. Its presence in these parts has been explained by what we may call diphtheriæmia, by post-mortem migration, or by association with other bacteria, of which the streptococcus is the most important, being found in septic types, in bronchopneumonia, and in glandular suppuration. Symbiosis with the streptococcus, which is the most common of all secondary infections and enters by the throat mucosa, increases the virulence of the diphtheria toxin (Roux and Yersin). Less important are the staphylococci—which rarely enter through the throat, but when they do, are extremely virulent—the proteus, colon bacillus, and pneumococcus.

The *action* of the bacillus is (*a*) *local,* altering tissue, producing necrosis, and forming membrane; when inoculated in animals, local necrosis develops, whereon fibrin exudes and forms a membrane in which the bacillus is found; the contiguous tissues are œdematous; and (*b*) *general,* forming toxins which are absorbed by the blood and lymph-vessels. Roux and Yersin (1888) found that the toxins injected alone, after killing the bacillus, produce no membrane or necrosis, but toxæmia and even paralysis precisely similar to the post-diphtheritic paralysis. According to Ehrlich there are two toxins; one, the *toxin,* produces the ordinary acute phenomena of diphtheria; and the other, the *toxon,* produces the later anæmia and paralysis. The chief receptors of the virus are the nervous and lymphatic tissues.

The *virulence* of the germ varies. This is tested on the guinea-pig by inoculating it with $\frac{1}{200}$ of its body-weight of a forty-eight-hour bouillon culture, and by noting the results: (*a*) Death in three days or less results from fully virulent cultures; (*b*) death in three to five

days from one of medium virulence; (*c*) death after a longer time, or only local changes, from weak cultures.

Some bacilli, known as the pseudodiphtheria bacillus, bacillus xerosis, etc., microscopically resemble the Klebs-Loeffler bacillus. An interesting and vital point is that pseudodiphtheria bacilli may become virulent and may kill; they are not neutralized by the diphtheria serum but only by the pseudodiphtheria serum (Alice Hamilton, Spronek, Ruediger).

Dissemination.—Diphtheria is disseminated (1) by patients acutely ill with the disease; the nasal, throat and laryngeal secretion or membrane may be coughed into the face or mouth of one of the family, nurse, or attending physician. Infections in which the patient is not kept in bed are especially dangerous, for children going to school may impart to others a most virulent diphtheria, though the same bacilli have produced in them little or no constitutional reaction; (2) by healthy individuals, in 1 per cent. of whom the germ is found without causing diphtheria, since individual predisposition is almost as necessary to infection as virulence in the microbe; (3) by fomites, especially damp cloths, in which the germ may live for months; cultures have been made from dry cloths after five months; the germ lives in gelatin from twelve to eighteen months; and (4) as the germ may be cultivated in dust, it is probable that it may be spread by the wind. Although the bacillus has been cultivated from cheese, milk, and eating utensils, many question this mode of propagation. It is not demonstrated that domestic animals convey the disease. Diphtheria is endemic in all thickly populated districts, and from these, epidemics develop from time to time. Unlike scarlatina, which is growing milder, diphtheria epidemics are apparently increasing in virulence, although far less malignant than in the middle ages and earlier modern times.

Age.—Most cases occur between the second and fifteenth years, and most deaths occur between the second and fourth years. Jacobi has seen three cases in the new-born, but infants at the breast are far less exposed than creeping children whose hands and mouths come into close contact with dirt and dust (Jacobi). It develops more frequently in girls than in boys. Adults are frequently affected.

Season.—Most cases occur in the cold months. Predisposition, bad teeth, stomatitis, hypertrophied tonsils, nasopharyngeal and bronchial catarrh, measles, and nervous affections, are predisposing factors. Some individuals are temporarily, and some permanently, immune. One attack confers no immunity, and in this respect diphtheria resembles erysipelas, pneumonia and rheumatism.

Symptoms.—There are *three major localizations of diphtheria,* the *throat,* the *nose* and the *larynx.*

1. Pharyngeal Diphtheria.—The typical case is pharyngeal.

(*a*) The *simple localized diphtheria* begins after an incubation of two to seven days with fatigue, pallor, coated tongue, anorexia, dysphagia, pain beneath the angle of the jaw and vomiting. Chilly

sensations, fever and thirst, pharyngeal voice, and sometimes abdominal distention, ensue. Infants refuse the bottle on account of nasal obstruction.

The *pharynx* is moist, glistening, and characteristically purple-red, with later, areas of gray, yellowish, or dirty green color, which vary in extent from small white bands to irregular, gelatinous elevations or masses, located on the tonsils, epiglottis, palate, pillars, arch, or pharynx. Except in the very incipiency of the disease, they are adherent, and, on removal, leave red, bleeding spots which are soon covered again with membrane. The pharyngeal tonsil alone may be involved, but may easily be overlooked without systematic examination. The breath is septic in odor. The nose is not wholly free and some discharge is the rule. The glands at the angle of the jaw are tender, and *fever* reaches 102°, may be rather irregular, or may be absent. In 90 per cent. of patients who recover, the fever does not exceed 102°. The patient usually recovers in seven to ten days. Otitis, suppurative adenitis, vomiting, and albuminuria are less common than in the more severe forms.

Wagner, Weigert, and Oertel, have shown that the *diphtheritic membrane is composed:* (i) Of necrobiotic tissue. The superficial epithelium, and, later, the deeper structures, are necrosed; the bacilli are found in the superficial layers and streptococci and staphylococci may be found more deeply situated; the necrobiosis may be also noticed in the connected lymph-glands, and in the internal organs (Weigert's coagulation necrosis, or hyaline transformation of the dead cells). (ii) Somewhat deeper, the membrane is composed of more or less marked fibrin exudation, which is probably a conservative, walling-off process, and also pervades the tissue initially necrosed. (iii) In the deepest part of the membrane, emigration of leukocytes occurs. In most adults the membrane is thrown off by the fourth or fifth day; in children by the seventh or eighth day.

The form described is *the classical type of mild local infection.* Even though the bacilli may be virulent, the tissues and blood resist the infection. *Several deviations from this type* are often clinically misinterpreted: (1) In "catarrhal diphtheria" the membrane is often absent or atypical and pultaceous. This type is especially common in families where the more severe forms are seen. The bacilli, redness, œdema, and swelling, are present. (2) Diphtheria may resemble follicular pharyngitis, and between them there is no absolute distinction except bacteriologically; in diphtheria the red is brighter, the tendency to fuse is greater, and the spots are yellow, gray, or green, while in angina, the color is duskier, the areas more discrete, yellow, and pus-like. (3) Follicular tonsillitis may be very closely simulated, and then is only distinguished bacteriologically. (4) In chronic diphtheria, there is a variable deposit upon a red, relaxed, œdematous pharynx. The glands may be swollen, but usually there are no toxæmic symptoms. (5) The latent diphtheria of Heubner, which occurs in marantic subjects, may easily escape detection.

(*b*) The *diphtheritic general infection* is difficult to describe in all its variations. It begins with high fever and such *febrile toxæmic symptoms* as headache, depression, insomnia, rapidly increasing anæmia, anorexia, dysphagia, and vomiting. Albuminuria and nephritis may appear later, with rapid respiration and the heart movement following the fever, the tones being dull and often accompanied by a systolic murmur. The pharynx is dark red, swollen, and glistening, and a membrane is seen on the tonsils or pharynx. This membrane is thin and gray; it begins in the form of streaks, or as a thin veil over the pharynx; it tends to spread very rapidly and sometimes diffusely and with offensive secretion. The hard palate and the superior part of the soft palate are usually normal. The neck is tender from glandular intumescence. The nose is often closed by a purulent secretion and diffuse membrane, and oral breathing results. There is a type peculiar to young children who, with initial nasal symptoms and without appreciable throat symptoms, suddenly develop laryngeal stenosis, and, in the pre-antitoxin days, almost invariably died. In severe cases the heart is irregular, with gallop-rhythm; there are throat paralysis, infiltration of the neck, dyspnœa, cyanosis, albuminous urine, tympany, swollen liver and spleen, antemortem fall of temperature, and death resulting from pneumonia, cardiac insufficiency, nervous symptoms or paralysis or nephritis.

(*c*) *Septic diphtheria,* according to Baginsky, is due to the diphtheria toxins, and is therefore seldom observed since the introduction of antitoxin. Other writers maintain that streptococcus and staphylococcus infection determine this septic type. The yellow-gray exudate becomes a dirty green or brown from hemorrhage, is ichorous and horribly fetid, and may reach the nose or larynx, causing erosions, necroses, and stenosis. It often involves the lymph glands (adenitis) and connective tissue (periadenitis). The tongue is dry; respiration difficult; and nephritis, generalized hemorrhage or local arterial ulceration, failing pulse, low temperature, swollen joints, and intumescence of the abdomen, liver, and spleen, usually result fatally. In 1881, 94 per cent. and, in 1884, 66 per cent. of cases of this type died (Glaeser).

The initial, atypical *fever in diphtheria* may be maintained by otitis, pneumonia, adenitis, and other complications in the severer types of diphtheria, or by concomitant diseases, such as measles, scarlatina or tuberculosis. The temperature of sepsis may develop. A pre-agonal perturbation may be observed, or there may be antemortem collapse. As in perforative peritonitis, algidity is due to febrifuge toxins. These statements apply chiefly to cases untreated with antitoxin.

Leukocytosis of the polymorphonuclear type develops in 90 per cent. of diphtheria cases. When absent, the infection is either very severe or very slight. It does not correspond to the severity of the disease nor to the fever curve. It disappears in favorable, and persists in severe, cases.

2. Nasal Diphtheria.—Diphtheritic rhinitis, catarrhal or croupous, due to the diphtheria bacillus, may usher in the disease or accompany the benign or more usually, *the severe forms.* The serous and often sanious or bloody nasal discharge excoriates the alæ nasi, lips and cheeks; membrane is often expelled; infection may travel to the eyes, lachrymal sacs, antrum or ears. Cervical adenitis is frequent, for the nasal lymph supply is most abundant and, as Jacobi pointed out, enlargement of the angular glands is always suspicious. The prognosis is nearly always grave, and convalescence very tardy. In contrast with this severe form, usually diphtheritic, though sometimes diphtheroid (streptococcic), there is a *fibrinous rhinitis* usually subacute or chronic and generally observed in children, which is due to the Klebs-Loeffler bacillus (82 per cent.), or to the streptococcus (18 per cent. of the cases); the membrane is abundant. This form is obviously dangerous to other children, since the remarkable absence of constitutional reaction, despite the menacing discharge, does not prevent play or school-going. It responds readily to antiseptic douches. Cultures should be taken, before adenoid operation, in all children with nasal discharge.

3. Laryngeal Diphtheria or Croup.—Most cases (85 per cent.) of croup are diphtheritic, though some are streptococcic; their symptoms are identical. *Frequency:* Diphtheria involves the throat in 84 per cent. and the larynx in 16 per cent. of the cases. *Forms:* (*a*) Catarrhal laryngitis, due to the diphtheria bacillus, produces symptoms of short duration, usually two or three days; they may be severe or even stenotic. (*b*) *Diphtheritic laryngitis* (*croup*) is descending in 97 per cent. of cases—*i. e.,* secondary to pharyngeal or nasal diphtheria; even in the rare and seemingly primary cases there is usually some pharyngeal involvement. The child is usually *hoarse* at night or early in the morning, coughs the *croupy cough,* and then after twelve to twenty-four hours is distinctly dyspnœic. Aphonia is common but not invariable. The *stridor* becomes menacing and then we know that the enduring croup is diphtheritic. The *stenosis is due to membrane* in the epiglottis, true cords, trachea, or even in the bronchi. The membrane is less adherent than in the pharynx, and sometimes more pultaceous. The accessory muscles of respiration come into play and there is inspiratory retraction of the thorax and epigastrium, especially in rhachitic children. Excursion of the larynx up and down, extreme restlessness, and temporary improvement after expectorating the membrane, may also be noted. The stenosis is accentuated by the central, immovable position of the vocal cords, by swelling in the subchordal tissue, and by muscular spasm. The cough is usually rather hoarse than metallic, is often periodic, is increased after tracheotomy or intubation, and often evacuates both the membrane and tube. Cough may be wholly absent because of decreased reflex bronchial excitability. Fever is usually absent. The pulse rate increases with augmentation of the stenosis because the vessels contract from central stimulation. The *respite following the raising*

of the membrane is usually transient, exacerbations occur, and finally, unless intubation or tracheotomy is performed, dyspnœa becomes continuous, with stagnation of secretion in the tubes, absence of the vesicular murmur, stridor, extension downward of the membrane, loss of strength, increasing rapidity and weakness of the pulse, cold clammy skin—in short, the complete picture of carbon-dioxide poisoning and total asphyxia develops. In some instances, perichondritis, deep necroses, or gangrene may develop. (In reading older medical works and pathologies, the student may be confused with the use of the terms "croup" and "diphtheria." Anatomically it was held that croupous and diphtheritic membranes differed chiefly in degree—croupous being more superficial and benign, diphtheritic being more deep and malign. In the present connection we use the term "croup" as synonymous with laryngeal diphtheria.)

4. Other Localizations.—(*a*) *Diphtheria of Skin.*—The diphtheria bacillus may produce membrane on the eye, lips, nose, navel, neck, genitalia, and extremities. It is usually associated with pharyngeal localization. The skin is hard and infiltrated, the secretion is corrosive, the contiguous lymphatics are involved, and sometimes cellulitis or phlegmon may result. Other instances of so-called "wound diphtheria" are due to the streptococcus—the most common cause of skin membranes. In tracheotomy, the membrane which forms in the wound is mostly streptococcic.

(*b*) *Diphtheritic Vulvo-vaginitis.*—Genital involvement ranges under 1 per cent., is dangerous, and usually follows severe pharyngeal diphtheria; it consists of diffuse membrane, painful rhagades, ulcers, and foul secretion, with intumescence of the lymph glands. A diphtheroid (usually streptococcic) form is also encountered.

(*c*) *Diphtheritic Ophthalmia.*—This localization is (*a*) false or diphtheroid (due to pus cocci) or (*b*) genuine or diphtheritic. Diphtheritic ophthalmia, chiefly in young children, occurs in 3 per cent. of cases. It may be primary in the eye, or secondary to nasopharyngeal diphtheria.

(*d*) *Diphtheritic Otitis.*—This does not occur in over 5 or 6 per cent. of the cases (as against 33 per cent. of otitis in scarlatina and nearly 100 per cent. in measles). The orifice of the Eustachian tube or the middle ear may be reached by the diphtheria bacillus which usually travels from the throat.

Complications and Sequels.—*The most important are heart paralysis, nephritis, and diphtheritic paralysis.*

1. Cardiac Complications (Diphtheritic Heart Paralysis).—*Anatomically,* the heart muscle is the seat of early changes. It is soft, thin, friable, light brown or yellow in color, and fatty; it is the occasional seat of endocardial and myocardial hemorrhages, acute interstitial myocarditis and fragmentation or myolysis, which Eppinger, in describing eighteen cases of sudden death, considers the usual cause of death. Pericarditis, with septic or hemorrhagic effusion, is not frequent. Permanent changes due to endocarditis (in 2 per cent. of fatal cases) or to myocarditis are uncommon.

Clinically, the tones are muffled. The first tone is impure or obscured by a systolic murmur (90 per cent. of cases); the second pulmonic is accentuated, the *rhythm is disturbed* (60 per cent.) and sometimes there is gallop-rhythm and embryocardia. The pulse is soft, dicrotic, increased from 80 to 120 (or 150), but it may be slow. *Heart-failure is more frequent than in any other acute infection,* not excepting pneumonia. Syncope on sitting up is fairly common. Diphtheritic heart paralysis occurs (1) early in severe or gangrenous types, or (2) late (in 10 to 20 per cent. of diphtheria cases) after three to four or six weeks, when convalescence is apparently established. It is marked by copious vomiting from vagus neuritis, pain in the chest and epigastrium, cardiac dilatation, cold extremities, pallor, dyspnœa, cyanosis and convulsions. The pulse is often irregular, fast, or with equally ominous significance, slow. Heart failure is explained in various ways (*a*) acute interstitial myocarditis (Rhomberg); (*b*) myolysis (Eppinger); (*c*) sudden centric vasomotor paralysis (Hasse); (*d*) neuritis of the cardiac plexus or of the vagus or phrenic nerves; (*e*) cardiac thrombosis causing cerebral or pulmonary embolism; 33 per cent. die.

2. Diphtheritic Nephritis.—(*a*) Albuminuria, resulting from renal degeneration, occurs in 50 per cent. of all cases and in 100 per cent. of severe cases. (*b*) In *light* forms of *nephritis* there is some albumin; the specific gravity and quantity of the urine are about normal, and there are leukocytes or epithelial cells with indistinct nuclei, the cells being highly refractive and coarsely granular. Blood is rarely found. There are some hyaline and granular casts. These findings are caused by the toxins and correspond in time and degree to the diphtheritic local process in the pharynx. (*c*) The *severe forms* of *nephritis* almost always occur in the severe general infection or in septic types and are characterized anatomically by diffuse parenchymatous changes, necrosis, hemorrhage, and exudation. Albumin, epithelium, casts, and cylindroids, are abundant. Hæmaturia is rare, although hemorrhage into the kidney substance is frequent anatomically. Although nephritis influences the prognosis, diphtheritic nephritis rarely produces hydrops or uræmia and "is not an independent factor as in scarlatina." It very rarely becomes chronic.

3. Diphtheritic Paralysis.—The paralyses occur clinically and experimentally from toxæmia in 3 to 10 per cent. of cases. The percentage, apparently, has not been reduced by the use of antitoxin (*v. i.*). The frequency and intensity of the paralysis is usually directly proportional to the intensity of the diphtheria; the causal diphtheria, in some cases, has apparently escaped detection.

(*a*) *Early Diphtheritic Paralysis.*—This occurs in septic cases at the acme. Dysphagia is its most common form. There is danger of inhalation pneumonia, especially in stupid cases. The patellar reflexes may disappear. Most cases die.

(*b*) *Post-diphtheritic Paralysis.*—This, the typical variety, occurs after two or three weeks and usually follows throat lesions since laryn-

geal cases are likely to die early; Baginsky saw it principally in adults and Rolleston mostly between the years of three and six. Gowers claims that all throat troubles leading to paralysis are diphtheritic, though Bourges and others cite cases of paralysis after simple angina. The most common paralysis is that of the palate, which causes dysphagia, anæsthesia of the throat and nasal speech. From central or peripheral change the pupils become wide and react to light but not to accommodation (Donders). The arms frequently escape. Ataxia may be the first symptom. The reflexes disappear early whether actual paralysis is reached or not, and the muscles react slowly, if at all, to the faradic current, although the reaction of degeneration by no means runs parallel to the degree of loss of voluntary motion. The muscles waste considerably. The palsied limbs are sometimes painful and the muscles and nerve trunks are often tender. The trunk, bladder and rectum are more commonly involved than is usually admitted. The sensory nerves suffer less, sensation being largely retained above the knees. Sensory disturbance may be noted in the rectal, genital and vesical twigs. The cranial nerves (strabismus, ptosis, diplopia), the phrenic nerve and the vagus (recurrent paralysis, superior laryngeal, cardiac branches), may be involved; fearful abdominal pains with threatening collapse attributable to sympathetic involvement, and sometimes psychical disturbance may be noted. Anatomically, disintegration of the medullary substance, multiplication of nuclei in Schwann's sheath, granular degeneration, and even total loss of the axis cylinder and sometimes foci of hemorrhage, are present, the changes being almost totally parenchymatous. Diphtheritic neuritis (actual nerve inflammation) is seen almost wholly in the palate and is due to local invasion from the inflamed throat. The patient recovers in two to three weeks if the pharynx alone is involved, and in three to eight months if the limbs are affected. Death results in 18 per cent. of the cases, chiefly from vagus involvement.

(*c*) *Organic Central Changes.*—The brain is slightly congested and œdematous. Degenerated areas may occur in the crura, medulla, or pons. Accidental findings are cerebral hemorrhage, embolism from heart thrombi, and acute encephalitis (possibly terminating in focal sclerosis). The literature contains 85 instances of hemiplegia (Rolleston, 1905). In the spine, hemorrhage, exudation into the nerve roots, meningitis, and degeneration of the motor cells and their roots, have been noted. The degenerative cerebrospinal lesions not infrequently occur with multiple neuritis.

4. Respiratory Complications.—The bronchi and trachea may remain free. There may be membrane formation (in 50 per cent. of fatal cases) or purulent secretion. Lobar pneumonia is a genuine complication, but *bronchopneumonia* is present in 50 to 80 per cent. of the fatal cases and is caused by the Klebs-Loeffler bacillus alone, or by the pus or pneumonia cocci, by the colon, proteus or pyocyaneus bacilli. Anatomically, some cases are due to downward extension of

the bronchitis; some are hæmatogenous infarcts or abscesses; others are due to inhalation, and still others, seen after intubation or tracheotomy, rather resemble lobar pneumonia and are elevated and granular in appearance. Increase of pulse and respiration-rate, local consolidation, and accession of fever are observed. Pleurisy (serous, hemorrhagic or purulent) is not often encountered.

5. Alimentary Tract.—*Mouth.*—Fissures, sordes, membrane or ulcers on the lips, suppuration in the tonsils and in the retropharyngeal space, gangrene and deforming cicatrices sometimes occur. The œsophagus is usually intact, but the diphtheritic process, in septic cases, may reach downward and produce stenosis.

Stomach.—Epigastric pain and vomiting are most often cardiac; death is usual in patients who develop vomiting late in the disease. The rare hemorrhage and membrane formation usually escape clinical recognition.

Intestines.—Constipation is usual; in septic forms, thick, foul or even dysenteric evacuations may be exceptionally observed. Anatomically, there may be membrane formation and swelling of Peyer's patches. The liver is swollen from fatty or cloudy degeneration or from congestion, in which latter case it also becomes painful. Icterus is rare. Splenic tumor is found in septic types especially.

6. The Skin.—The skin is usually normal, except for the febrile hyperæmia, cardiac pallor, croup-cyanosis or septic grayness. The following alterations may be observed: Herpes, sweats, petechiæ, symmetrical gangrene and septic dermatomyositis; they usually occur with adenitis, nephritis, pneumonia, ulcerative endocarditis and scarlatiniform or morbilliform erythemata. Scarlatina or measles may actually complicate diphtheria.

7. The Glands.—The anterior cervical glands are swollen, tender and painful. In the most severe forms periadenitis follows with diffuse involvement of the cellular tissue (angina Ludovici), section of which evacuates serum, seldom pus. Glandular and articular changes are less frequent than in scarlatina. Muscular degeneration, interstitial infiltration, and tendency to separation of the muscle-fibers (myolysis or fragmentation) are observed in the heart or even more clearly in the diaphragm.

Diagnosis.—To the naked eye a *thick, adherent membrane, which is not removable* without hemorrhage and which leaves ulceration (Bretonneau), is sufficiently characteristic. The enlarged glands are of diagnostic value. A *positive bacteriological diagnosis* is important, in such cases as have actual membrane, diffuse reddening, and tonsillitis; but three or four negative examinations are necessary before negative conclusions can certainly be drawn. The statistics of Heubner and of Baginsky show positive findings in from 92 to 98 per cent. of cases in which the clinical diagnosis of diphtheria has been made. These figures are probably most nearly correct, although others find the diphtheria bacillus in a notably lower percentage. The nose, nasopharynx and pharynx must always be examined in

sick children and adults. In suspicious cases, particularly when cultures cause considerable delay, antitoxin should be given at once.

The health departments in large cities provide cultures, stations at which cultures and antitoxin may be obtained, and central and subsidiary laboratories for diagnosis. The Chicago Health Department has an able corps of physicians for free treatment, consultation and intubation. It is easy to obtain, on sterile cotton on a probe, a smear from the membrane or suspicious focus, to rub it on the serum without breaking its surface, and to put the tin box containing it in the incubator or under the axilla until the brood oven can be reached. In half a day characteristic cultures are obtained. Direct smears from the throat on a slide very often show the bacillus. In small places communal laboratories can be maintained easily, and competent young practitioners found to do this, and other clinical microscopy. In laryngeal diphtheria the bacillus is usually found on the pharynx, but not on the tonsils. Repeatedly negative findings are of value in the *pseudodiphtheritic* or diphtheroid pharyngitis, rhinitis, laryngitis, and bronchitis,—*e. g.,* membranes due to strepto-, staphylo-, and pneumococci, aphthous growth, syphilis, and the colon bacillus. The *scarlatinal* membrane has been considered. In *Vincent's angina* (*q. v.*), angina diphtheroides or angina ulceromembranosa, described by Vincent (1898), there is a whitish-yellow or grayish-brown membrane which may be confused with diphtheria and syphilis and which is caused by the symbiosis of two otherwise innocuous parasites; the bacillus fusiformis (6 to 12μ, staining readily with methylene blue and fuchsin), and the spirochæta dartcola (twice as large, and not growing in cultures). The infection is usually mild.

Course and Prognosis.—Aside from complications, the average course is one week, or possibly two. It is more favorable in diphtheria of the tonsils and skin than in that of the nose. The prognosis always must be guarded. (*a*) Without serotherapy the danger is great, since light cases may become severe or extend to the larynx. (*b*) Epidemics vary in virulence, 30 to 50 per cent. mortality having been observed twelve to fifteen years ago. (*c*) According to Glaeser, in the first year the mortality is 77 per cent., and sinks with each year until it is, at ten years, 20 to 25 per cent.; after ten, 2 to 5½ per cent.; after thirty and forty, 1 to 3 per cent. (*d*) Other factors are debility, wide extension of the infection from part to part, adenitis, and croup; the general diphtheritic infection or septic forms with hemorrhage or gangrene; heart and renal complications; rapid and weak or slow pulse; ecchymoses and late vomiting; or involvement of the brain or nerves. (*e*) In 946 fatalities studied by White and Smith, about 50 per cent. died of bronchopneumonia, usually after intubation or tracheotomy; 25 per cent. died of cardiac complications, and 25 per cent. of early severe toxæmia, though some succumbed to asphyxia or late paralysis. (*f*) Coincidence with other infections is ominous, such as measles, in which the exanthem may become hemor-

rhagic, and a lethal respiratory catarrh usually results; coincidence with typhoid, frequently with gangrene or laryngeal stenosis; and with scarlatina, summer diarrhœa, and tuberculosis. The disease is less severe in conjunction with varicella and pertussis. Diphtheria belongs to the recurrent infections.

Treatment.—1. Prophylaxis.—For the physician, this begins with the case in hand. Isolation must be absolute. In needy families the isolation hospital offers the best solution. In Boston it has reduced the mortality of diphtheria to one-third or one-sixth of its former rate. All suspicious throat cases should be isolated pending their immediate clinical and cultural evolution. Not only should a sick child be isolated, but other children in the family should be kept from school and away from playmates. Of course, the sick one must be isolated until the throat gives two negative cultures; three weeks is the average isolation. The physician may protect himself by examining the throat through a piece of glass, and may avoid carrying the germs by slipping on a surgical gown and washing both face and hands with bichloride of mercury or similar solution. In fatal cases the body should be wrapped in a sheet saturated with bichloride, and buried privately in a tightly closed casket. Those exposed to infection may receive antitoxin, and should gargle the throat once with ½ of 1-per-cent. solution of silver nitrate, or often, with bichloride of mercury ($\frac{1}{5000}$). The clothing, blankets, rugs, etc., must be steamed. After convalescence, the walls of the room should be rubbed with bread which is then burned, the woodwork should be washed with 2-per-cent. lysol solution or bichloride of mercury and the room should be fumigated (see Smallpox). Under prophylaxis may be included the building up of the general physiological resistance and the treatment of chronic catarrh of the nose, throat and bronchi.

2. Local Treatment.—Though local treatment of the throat is less important than before the days of serotherapy, the antitoxin does not always kill the bacilli, and does not affect the associated streptococci. Caustics are never necessary. In children such mild antiseptics as 3-per-cent. boric solution and 3- to 10-per-cent. potassium permanganate solution, should be used lest they be swallowed. The child is held in the nurse's arms with its arms and body firmly wrapped in a sheet. The mouth is made to open by closing the nostrils, and a piece of wood or cork is inserted between the teeth. The throat is then swabbed. It is a harsh procedure and the effects are apparently more than offset by the resultant exhaustion. In adults 1 to 1000 bichloride, 25-per-cent. hydrogen peroxide, or 1-per-cent. carbolic solutions, or iodoform salve, may be used, or Loeffler's solution, which is as follows:

℞ Mentholis ..... 10 parts.
Toluolis ..... 30 parts.
Liq. ferri chloridi ..... 4 parts.
Alcoholis absoluti ..... 60 parts.

Loeffler's solution should be kept well corked in a dark bottle. Local injury, such as opening up new wound surfaces for infection, must be avoided. In nasal irrigation the tube is held horizontally, so that fluid may return by the other nostril. A kettle containing a weak carbolic solution should be kept steaming in the room.

3. Serotherapy.—Behring (1890) borrowed Ehrlich's results with ricin and abrin to establish the units of immunization, and (1893) used the diphtheria antitoxin on diphtheria patients. The method of action of an antitoxin is vital or active (Buchner, Roux, Martin) though some (Behring and Kitasato) consider that it is chemical or passive.

1. *Technique.*—A syringe with an asbestos plunger should be used. The syringe need not be boiled each time, but should be cleaned after each injection with water until the serum is washed out; it should then be washed with carbolic acid or alcohol, which should be thoroughly removed each time lest it coagulate the antitoxin at the next injection. Syringes are generally supplied with the serum. With ordinary surgical antisepsis, inject the antitoxin into the subcutaneous tissue of the thigh in adults, and into the interscapular region in children. In very severe infections, intravenous injections give results eight hours earlier. The unit is 1 c.c., which counteracts ten times the minimum dose of diphtheria poison fatal to a 300 gm. guinea-pig.

2. The *dosage* is rather empirical, depending on the localization, age, and severity of the disease and the effects produced. After administration the discharge should lessen, the membrane exfoliate, the fever fall, and toxæmia decrease. At least twice as much antitoxin is necessary for cure as for immunization. After eight hours, three times as much is necessary as at the beginning; after twenty-four to thirty-six hours, eight times as much. As we cannot measure the toxins, so we cannot exactly specify regarding the antitoxin dose. Personally, the author prefers *an initial dosage of 3,000 to 5,000 units to insure results and to avoid delay and frequent repetition.* The decreased bulk of present preparations also allows a larger dosage. In severe cases twice the above amount should be given. McCallum, above all others, has emphasized the importance of full doses, and he speaks of doses of 10,000, 30,000, or even 50,000 units in desperate cases, because results must be produced. Sutherlin gave a total of 498,000 units in one case!

3. *Effects.*—(*a*) The local changes are lessened, the mucosa becomes redder, and the membrane more yellow and it exfoliates in one-half to three days, with decrease in glandular swelling. Extension afterward is rare. (*b*) The fever falls by crisis or by lysis, unless there are complications. A temporary rise in temperature sometimes occurs. Antitoxin is given until the fever falls. (*c*) The pulse is lessened, and hyperleukocytosis is decreased. Light cases rarely become severe, and nonseptic rarely become septic, unless there is co-existing tuberculosis, syphilis, etc. The kidneys are never injured.

The author believes that paralysis and heart failure are less frequent when the administration of the antitoxin is prompt. Both of these complications are thought to occur in the same proportion as in pre-serum days. But even if their percentage is not absolutely reduced, many patients now live who would previously have died before developing paralysis. (*d*) The mortality is reduced from 41 (46 per cent.) to 15 per cent. (Siegert's collection 42,000 cases); to 10 per cent. (Wenner, 9.8 per cent., 132,500 cases, American cities); or to 6.7 per cent. (Chicago Health Department Report). According to Bayeux, the mortality in 230,000 diphtheria patients was 55 per cent. before, and 16 per cent. after, the introduction of antitoxin. The fatal cases and severe complications are lessened in direct ratio as the antitoxin is given early; if on first day of disease, less than 1 per cent; second day, 2 per cent.; third day, 8 to 10 per cent.; fourth day, 14 per cent. Failure to use antitoxin is criminal neglect; 50 per cent. of croup cases regress with the use of antitoxin, and heart lesions are reduced 50 per cent. in fatal cases. (*e*) Antitoxin is a prophylactic, and should be given in special instances; *e. g.,* to weak or very young children (in smaller doses). It protected for two or three weeks in all but 0.7 per cent. of 1,000 cases (Zuppinger). (*f*) The sequels are: (i) Abscess formation; (ii) exanthemata, which are local (on the fourth or fifth day) or general (in one to two weeks), are due to individual peculiarity or some unknown element in the antitoxin, and appear as urticaria, morbilliform, scarlatiniform, or petechial eruptions; antitoxin rashes occur in 33 per cent. of the cases, and are said to be decreased by calcium chloride, gr. xv, for three days after the injection (Netter); (iii) nephritis, always due, however, to the diphtheria itself; (iv) sudden death which results from vomiting into the larynx, shock, lymphatic diathesis, and from other disease; sudden death is exceedingly rare; (v) tetanus has been inoculated. In a few cases where antitoxin was injected for prophylaxis, the author has seen extremely severe symptoms; pallor alternating with flushing, chills, fever, renal suppression, œdema of the skin and pharynx and very severe cardiac weakness; these cases to a certainty had no diphtheria, for throat cultures were first taken; the statement, therefore, that antitoxin *never* injures does not hold for all prophylactic doses. This "hypersusceptibility" or anaphylaxis, has lately been studied by Rosenau and Anderson, who refer the reaction merely to the foreign proteid introduced in the injection.

4. General and Symptomatic Treatment.—A rich *fluid diet* of milk, eggs, etc., with tonics, cinchona, wine, etc., is given. In marked dysphagia rectal feeding is indicated. Forced feeding by the stomach tube or nasal catheter is likely to injure the throat.

Sepsis.—Concentrated diet by mouth and rectum, alcohol *ad libitum,* oxygen, saline infusions or enemata, strychnine, iron, and anti-streptococcic serum should be given; all measures are usually of no avail if tens of thousands of units do not give relief. Credé's collargol is given intravenously in severe cases; Netter, in 586 cases, found that it reduced the death rate by 8 (to 20) per cent.

Paralysis.—General stimulation and alimentation are indicated; electricity is of little or no avail; massage, baths and strychnine are useful in the later stages.

Heart Failure.—The ice-bag is indicated for stormy heart action; strychnine, iron, oxygen, digitalis, coffee, alcohol, aromatic wines and camphor should be given. (See SCARLATINA, for dosage table.) For severe abdominal pain and vomiting, usually an evidence of cardiac failure, the above remedies, and champagne with menthol and carbolic acid, are indicated. The heart must be watched far into apparently complete convalescence.

Nephritis.—Nephritis cannot be prevented. Alcohol, tincture of ferric chloride, beef tea and eggs are to be avoided; vichy, cardiants, milk diet, baths, laxatives, and care of the skin are indicated.

Local Diphtheria.—Two per cent. nitrate of silver in balsam of Peru affords relief in vaginitis; ice applications, atropia, and silver in ophthalmia; over inflamed glands, a 10 per cent. ichthyol salve may be applied; local diphtheria necessitates antitoxin, irrespective of its location.

Croup.—Mercurial inunctions are given, together with steam sprays or tents and ice locally applied; for emergencies, the hot water may be turned on in the bath-room, after closing the door (Jacobi); or the child may be held over a pail of water into which live coals are thrown; emetics may be administered, with which wine is given because emetics often fail in carbon-dioxide narcosis (Stricker); retraction of the chest walls, suffocation, cyanosis, and cold extremities, necessitate intubation or tracheotomy.

O'Dwyer's Intubation.—Advantages.—It is bloodless, easier, and quicker than tracheotomy; there is less wound infection, and it is less objectionable to the family.

Disadvantages.—Membrane may rise beneath the tube, and tracheotomy may therefore be necessary after intubation. The tube may sometimes produce trauma, false passages, necrosis, ulcers, perichondritis and stricture. The tube is usually replaced two, three, or four times, and rarely need remain more than five or six days when the serum is used. It is removed by the extubator, or by pressure. Size of the tube: 5 to 6 millimeters, one to two years; 6 to 7 millimeters, two to four years; 7 to 8 millimeters, four to six years; 8 to 9 millimeters, six to eight years.

Tracheotomy.—Tracheotomy is indicated when the heart is weak or when great asphyxia, much secretion, or gangrenous, diffuse or septic infiltration exist. In the after-treatment, the patient requires a moist atmosphere, and cleaning of the tracheotomy tube every second day, is important. The tube should not be permanently removed until all stenotic symptoms and all plaques disappear. In cases treated by serotherapy, this may be done on the fourth day as against the sixth or seventh in cases treated before antitoxin was introduced. The prognosis of tracheotomy depends upon possible coincident wound-infection with erysipelas or diphtheria, phlegmon,

thrombophlebitis, mediastinal suppuration, ulceration of trachea, inhalation pneumonia, cardiac collapse, and nephritis. Rapid rise of temperature is ominous. Antitoxin has reduced the mortality from 66 per cent. to 35 or 25 per cent.

CEREBROSPINAL FEVER.

Definition.—Cerebrospinal or "spotted" fever is an acute specific infection, caused by the meningococcus, characterized anatomically by a purulent or sero- or fibrino-purulent exudation in the cerebrospinal meninges and marked clinically by headache, rigidity of the neck and spine, hyperæsthesia, vomiting, Kernig's sign and often by various paralyses.

In 1805, Vieusseux, of Geneva, first described epidemic meningitis; in 1806, it was seen by Danielson and Mann in New England, and they performed the first autopsies (Osler).

Bacteriology.—The *Diplococcus intracellularis meningitidis* of Weichselbaum or meningococcus resembles the pneumococcus and gonococcus. It lies in pairs or even tetrads, its contiguous sides are flattened, and it resembles two coffee grains with their flat sides apposed. It is wider and less lanceolate than the pneumococcus. It is usually intracellular, being enclosed in the polymorphonuclear leukocytes. Unlike the pneumococcus, it usually has no capsule, although one may develop in serum cultures. It decolorizes by Gram's method. Cultures do not show very active growths; it develops best on agar or blood serum as white, viscid, shining colonies and it grows poorly on gelatin. Subcutaneous inoculations are usually negative, therein differing from the pneumococcus, but it produces inoculation-meningitis and peritonitis. The meningococcus is found in the nose, from which it is thought to invade the brain. Lord found it in the nose, in cases with and without meningitis. Von Lingelstein found the meningococcus in the nasopharynx in 94 per cent. of cases in the Silesian epidemic (*v. i.*) and every time in 68 personal observations. He asserts that the disease is communicable only by direct contact with the mucus from the nasopharynx of patients or of those in contact with them. Its presence has been demonstrated in the blood in 25 per cent. of cases by Elser, kidneys, joints, spleen, ear, tonsils, lymphatic nodes, pleura, lung, heart valves, pericardium and more rarely in the sputum and urine; it probably travels by way of the lymph vessels.

Predisposing Etiology.—Children and young adults are especially predisposed; it occurs more often in males, is apparently more frequent in the country, although it is often endemic or epidemic in barracks, prisons, hospitals and tenements. The disease has closely followed the movements of armies or regiments. Though not very directly contagious, its contagiousness seems proven beyond question. As many as seven members of a family have been successively stricken. Most cases are seen in the late winter and early spring; the author

has always seen some cases in these months. 2,594 deaths occurred in New York in 1904 and 1905. In a Silesian epidemic in 1905 there was a mortality of 57.6 per cent. of 3102 cases. Trauma, overwork and depression are but predisposing causes. Like pneumonia, it often attacks the robust and may recur; Councilman collected five instances of recurrence. The disease is also seen in the horse and goat.

Pathology.—The findings in the main are those of the suppurative form, *viz.*, those of an acute fibrinopurulent, or less often a seropurulent leptomeningitis. The cerebral dura is seldom, and the spinal dura often, involved. The inflammation follows the lymphatics and vessels penetrating the brain and cord, resulting in encephalitic or myelitic foci, small abscesses, parenchymatous and interstitial changes. The axis cylinders are sometimes lost. Flexner and Barker observed rows of cells "like miniature tubercles," of which some were two to eight times the size of the leukocytes. The exudate is rich along the fissures, fossa of Sylvius and at the base about the chiasm or the surface of the pons and cerebellum. Hydrops ventriculorum is fairly frequent, in cases of long standing. In other organs the changes are those of acute infection, the muscles are brownish-red, dry and degenerated, there is parenchymatous degeneration in the heart and kidneys, and sometimes acute splenic tumor, arthritis, pneumonic foci or swollen glands in which the coccus may be found.

Symptoms.—The period of *incubation* is not definitely known, but lasts from a few hours to several days (three to eleven). Prodromes are vague, as malaise, or headache. Strümpell and others observed initial coryza. The onset may be gradual or sudden, sometimes with a chill and usually with fever. In general, the course is febrile, attended by brain irritation; by an almost constant triad of symptoms, *headache, rigid neck and hyperæsthesia;* by vomiting, mental, motor and sensory symptoms, and in fatal cases death results from paralysis, convulsions or coma.

1. General Nervous Symptoms.—(*a*) *Headache* is almost invariable, though rarely absent in very young children, or alcoholics. It develops early, is most severe and is at first localized but soon becomes generalized. Though it varies somewhat in intensity, it is constant, with exacerbations, and provokes the short plaintive, clear meningeal cry, *cri hydrencéphalique* which is largely heard in children (Coindet, 1817). The headache is throbbing or lancinating, even under narcotics, and persists after delirium and even coma have set in. Its cause may be compression of the dura, changes in the brain, inflammation of the nerve trunks, or ventricular exudation. (*b*) *Hyperæsthesia* or hyperalgesia affects the special senses, causing intolerance of light and noise, or general sensation—affecting the arms most, the trunk less and the legs least. It is rarely absent, always suggestive, and probably due to root neuritis. (*c*) *Rigidity of the neck and spine* is caused by inflammation of the nerve-roots, possibly from lesions in the pons or medulla. The *head is retracted,*

sometimes so severely that the occiput lies between the shoulders, producing dysphagia and decubitus. The entire body may be lifted by the head. Marked *opisthotonos* may be present. The most common attitude is the lateral, with the head retracted, the arms flexed and the knees drawn up. Flexion and extension of the head are impossible. Retraction is one of the cardinal triad. Closely associated and caused in the same way, is severe *spinal pain with tenderness,* chiefly in the neck and loins, which is felt rather to the side of the spine than directly over it. It may radiate to the trunk and in 33 per cent. of cases to the extremities. (*d*) *Contractures of the limbs* are caused by root inflammation, increased pressure, or lesions in the pyramidal tracts. *Kernig's sign* (see DIAGNOSIS) comes under this head. (*e*) *Mental symptoms* often occur early, especially in children, neurotics and alcoholics; they include unrest, insomnia, delirium (sometimes with periods of normal intelligence), and mania which is followed by stupor and incomplete coma. (*f*) *Vomiting,* in the initial stage, is due to vagus irritation; later it results from ventricular hydrops or increased cerebrospinal pressure. It is of the cerebral type, *i. e.,* it occurs without nausea, without relation to eating, is often projectile and is not amenable to gastric therapy. Vomiting is more typical and common in children. The tongue may later become coated and foul. (*g*) The *pulse* at first is increased, and is sometimes disproportionately rapid, considering the fever. Sometimes it slows later because of brain pressure but ultimately it is fast again. It may be irregular in rhythm and oscillating in rate. Friis noted variation from 84 to 144 within a minute. (*h*) *Respiration* is irregular in 20 per cent. of cases; Cheyne-Stokes' breathing is not common, save toward the end. Deep breathing with apnœic pauses (Biot's breathing) is quite common, though less so than in the tuberculous type. (*i*) *Convulsions* are most common in the young; they may occur at the onset or later. They are ambiguous from their frequent occurrence in other diseases of childhood.

2. FOCAL NERVOUS SYMPTOMS.—These are partly irritative and partly paralytic. (*a*) *Symptoms relating to the cranial nerves* are as follows: These nerves are most involved in basal localization, while the limbs are most affected in that of the convexity. The optic nerve is frequently inflamed, if the disease lasts over four days. The disk is swollen and its margin is "washed" (see Plate VII, Fig. 4). Neuritis is less frequent than in tumor; hemorrhage and choking are uncommon. The ocular muscles may be involved. The pupils at first are usually small, often unequal, and sometimes variable like the pulse. Later they widen spontaneously or on movement of the rigid neck, and irritation of the skin, but do not react to light. These as well as the following changes, result from cortical or basal disease. Ptosis, diplopia, nystagmus, and strabismus are frequent and often vary from time to time. Complete paralysis of the third nerve is not common. Involvement of the fifth nerve is unusual, although trismus, grinding of the teeth and trigeminal neuralgia have been

noted. Involvement of the facial nerve is frequent, ranking next to that of the third nerve. If diseased at the base, its paralysis is usually total; if at the cortex it is of the cerebral type, and its upper third escapes. The paralysis varies from time to time (*v. s.*). The facial expression shows suffering, the brows are slightly elevated and the forehead is corrugated. The *risus sardonicus* is not frequent. The auditory nerve is often bathed in pus, and hemorrhagic inflammation in the labyrinth results from extension. The tongue on protrusion may deviate to one side; dysarthria or dysphagia are sometimes seen. (*b*) *Symptoms relating to the limbs* occur as follows: Paralysis of the limbs is not very frequent, although noted in some epidemics. Hemiplegia is rarely complete, occurs with or without participation of the tongue and face, and is caused by involvement of the cortex, changes in the pyramidal tracts, and sometimes apparently by reflex inhibition. It is frequently accompanied by rigidity of the limbs and aphasia. It is most frequent in children, occurs early or late and may be temporary or permanent. Monoplegia is not common and most often concerns the face or arm. Choreiform or athetotic movements, twitchings and tremor are occasional. Convulsions are frequently Jacksonian in type. Sometimes the paralyses are spinal, as paraplegia, which also involves the functions of the bladder and rectum. The tendon reflexes, as the patellars, may be increased at first and are often decreased or abolished late in the disease. The reflexes of the skin are variable.

3. General and Somatic Symptoms.—(*a*) There is almost always *fever*. The temperature rises suddenly, is usually irregular or remittent, follows no definite cycle, and lysis is usual in cases which recover. A temperature of 107° may be registered before death. (*b*) The *blood* shows leukocytosis, which is early, constant and of no prognostic import; the white cells may number 25,000 to 40,000; the meningococcus is found in 25 per cent. of cases. (*c*) The *abdomen* is often retracted, and is scaphoid, although less so than in the tuberculous type. It may be due to retraction of the abdominal walls or intestines from irritation of the vagus [Traube]. (*d*) The *spleen* is often palpable. (*e*) The *urine* is febrile, albuminous (33 per cent.) and may show peptonuria, hæmaturia in malignant cases, and rarely glycosuria or polyuria (Traube). (*f*) The *nutrition* suffers, which Leube explains by cerebral influences. (*g*) *Skin:* The *tâche cérébrale* described by Trousseau has no differentiating value, for it occurs in many febrile and other conditions. Eruptions are more common in the epidemic than in the sporadic cases, and vary with the individual epidemic. *Herpes* varies with the epidemic (40 to even 90 per cent.); it is most frequent on the face, but may develop anywhere; it spreads more rapidly than in any other disease (v. Ziemssen); in one case the author saw it extend from the lip to the ear, neck, chest and shoulder. *Purpura* [*"spotted fever"*] occurs in severe types. Erythema, roseola, urticaria and gangrene are uncommon.

Clinical Forms.—Hirsch distinguishes (*a*) the *acute* or subacute type, the ordinary form in which the acute symptoms of onset are followed by the general nervous symptoms enumerated, then by focal nervous symptoms, somatic symptoms, death in coma, or slow recovery; (*b*) the *foudroyant form,* with violent onset, rapid course and death in a few hours (three to thirty-six) with apoplectic symptoms, or purpura, high fever, irregular heart and early coma; (*c*) the *abortive form,* with low fever and symptoms so mild that the disease is recognized only by its association with an epidemic; the ambulatory type is included here; and (*d*) *Heubner's chronic form,* which lasts two to six months, with marasmus, and recurrent fever.

Complications.—These vary with the epidemic. Pneumonia, bronchitis, tonsillitis, pleurisy, parotitis, endo- and pericarditis, arthritis and nephritis are the most common. Pneumonia usually is due to the pneumococcus, but Councilman found the meningococcus in small isolated foci in the lung. The arthritis is either poly- or monoarticular, simple or suppurative, and may heal even when suppurative; it occurred in 25 per cent. of Flexner's and Barker's series.

Sequels.—Mental alteration, paralysis, contractures, obstinate headache, optic atrophy, deafness or Ménière's syndrome may remain after recovery. Chronic hydrocephalus, paroxysmal headache, vomiting, pains, and convulsions may result (v. Ziemssen). In an Heidelberg epidemic, Moos found as sequels deaf-mutism in 60 per cent., absolute deafness in 31 per cent. and an atactic gait in 50 per cent. of the cases.

Prognosis.—The average mortality is 37 per cent. (Hirsch in 15,632 cases), but varies with the epidemic, and may reach 75 per cent. Fifty per cent. of the deaths occur in the first five days. Recovery is exceptional after deep coma, repeated convulsions, high fever or paralysis of the vagus. Obstinate vomiting, disturbed respiration, sudden drop of temperature and inanition are ominous signs.

General Diagnosis.—The *first step* is the diagnosis of meningitis, which in general has an acute or subacute onset, with signs at first of general, then local inflammation of the surface of the brain. The *second step* is determination of the variety (suppurative, epidemic, tuberculous, primary or secondary), cause, and localization.

Cardinal Symptoms.—The important *early triad* of (*a*) headache, (*b*) retracted rigid neck and (*c*) hyperæsthesia, develops early. (*d*) *General symptoms,* as vomiting, abdominal retraction, pulse and respiratory changes, fever or convulsions, are important in their grouping. (*e*) *Lumbar puncture* (Quincke, 1890) is of great diagnostic value. Its *technique* is as follows: The patient lies on the side with the knees drawn up and the body bent as far forward as spinal rigidity allows; with or without chloroform, an aspirating needle or small trocar is introduced in the median line in children, and a quarter to half an inch to one side in adults, between the third and fourth lumbar vertebræ (Quincke) or between the last lumbar

vertebra and sacrum, and is pushed forward toward the median line and a little upward, one and a half inches deep in children and 1.5 to 2.5 inches in adults, until it passes into the canal. Fürbringer prefers to have the patient in the sitting posture. Not more than 4 to 5 drams (15 to 20 c.c.) should be withdrawn, lest fatal results ensue. Negative taps result from needle occlusion by clots. *Results:* (i) Normally, the pressure with the patient in the lateral position, equals 100 to 150 mm. of water and the escaping fluid only trickles out. In meningitis, the *pressure is greatly increased* (200 to 700 mm.) and the fluid often spurts out. (ii) The normal fluid is clear, has a specific gravity of 1,007–09 and contains little albumin ($\frac{1}{2}$ to 1 pro mille). Though the fluid may be clear in all forms of meningitis, it is usually clear in the serous form, but *cloudy, flocculent* or sometimes bloody in other forms; in the tuberculous variety it is usually clear or slightly yellow with a whitish clot forming on standing; in the epidemic form, it may be turbid or clear, with a yellowish clot; in the purulent type it is opaque, and in rare cases pure pus. The specific gravity is about 1,010 in the serous, 1,011 in tuberculous, and 1,015 in the purulent or epidemic variety, although these figures are not absolute. The percentage of albumin is small in the serous, 2 pro mille in tuberculous, and 3 or even 9 pro mille in the suppurative forms. (iii) *Bacteriologically* in the purulent type the streptococcus, staphylococcus, pneumococcus, typhoid and colon bacilli are found; in the epidemic variety, the meningococcus which was positive in 100 per cent. in the last New York epidemic, is present; in the tuberculous type, tubercle bacilli exist in 66 per cent. of cases as an average (81 per cent. Fürbringer, and 100 per cent. Holtzmann); even in this form, mixed infection with the pus organisms and meningococcus may be seen; the tubercle bacilli are often found on cover slip preparations. (iv) The *cellular morphology* (*cyto-diagnosis*) is a valuable aid. In the purulent and epidemic forms leukocytes are largely polymorphonuclear; in the tuberculous form the lymphocytes predominate according to Widal, Sicard, and Rivault (see CYTODIAGNOSIS, PLEURISY).

FIG. 17.

Kernig's sign, showing retraction of head and back and prominence of knees in the erect posture, *i. e.*, the impossibility of extension of knees (Dieulafoy).

(*f*) Kernig (St. Petersburg, 1882), described a sign known as

Kernig's sign. When the patient sits or the thighs are at right angles with the trunk, in 75 to 95 per cent. of all meningitis cases it is impossible to extend the knees, as shown in Fig. 17. The legs and thighs are flexed (flexor contracture) and the knees cannot be pressed down, *i. e.,* cannot be extended when the patient is sitting but this is possible when he is lying. It indicates involvement of the spinal meninges, degeneration of the pyramidal tracts or irritation of the posterior sensory roots. It lasts well into convalescence, whence, as Netter states, "a retrospective diagnosis may be made." It occurs at times in other conditions, but in infancy is almost invariably meningitic. It is sometimes unilateral.

(*g*) *Local signs* usually follow the general signs, and are more important. They include the pupillary changes, strabismus, facial paralysis, and hemiplegia. Often their late development lessens their value for early diagnosis.

Diagnosis of the Variety (*v. s.*).—This is more difficult. Cases of tuberculous meningitis sometimes increase when the epidemic form prevails, and the meningococcus may be found in these cases. Sporadic cases, due to the meningococcus, may be indistinguishable from primary pneumococcic meningitis, save by lumbar puncture.

	Epidemic Form.	Tuberculous.	Suppurative.
Onset:	Sudden, with few prodromes.	Longer prodromes; family history; other evidences of tuberculosis.	Sometimes primary but oftenest secondary to ear disease, acute infections, trauma, pneumonia.
Fever:	Fairly high; pulse more rapid than in tuberculous type.	Less; inversed type frequently; sometimes absent.	Especially high, with chills, perhaps pyæmic type.
Eruptions:	Herpes and purpura very common.	Rare.	Rare—polymorphous septic rashes.
Leukocytosis:	Regular and marked.	Exceptional.	Frequent.
Rigidity of neck:	Most frequent.	Spinal symptoms somewhat less frequent.	Sometimes absent (pneumococcic form).
Other symptoms:	Peptonuria.	Choroidal tubercles.	Peptonuria.
Course:	Rapid—more recoveries.	Subacute, slower, fatal.	More acute, very rarely recover.
Lumbar puncture:	Meningococcus; leukocytes polymorphonuclear.	Tubercle bacilli; mononuclear.	Pyogenic cocci, pneumococcus; polymorphonuclear.

Diagnosis of Location.—(*a*) Localization on the *convexity* is characterized by more frequent delirium, Jacksonian convulsions,

mono- or hemiplegia, less cranial nerve paralysis and less optic neuritis. (*b*) Localization at the base is characterized by more frequent cranial neuritis, and optic neuritis. (*c*) The *ventricular* form rarely involves the cranial nerves.

Differentiation.—1. From Nervous Toxæmic Symptoms.—Jenner pointed out that the headache ceases when delirium begins in acute infections, *e. g.,* the pseudo-meningeal symptoms of pneumonia, typhoid (*q. v.*), sepsis, etc. Meningitis with focal symptoms may be most confusing (see Uræmia), but the albuminuric retinitis, cardiovascular changes, albuminuria, casts, and deficient elimination are usually distinctive. Acute *inflammation of the labyrinth* may resemble meningitis, for it sometimes produces headache, vomiting, fever, convulsions, stiff neck, and even optic neuritis. If labyrinthitis involves the meninges, the facial nerve is likely to be affected. Such conditions resembling meningitis Dupré called meningismus, and Bouchut pseudomeningitis.

2. From Brain Disease.—See *syphilitic meningitis, brain abscess,* cerebral hemorrhage, and encephalitis. Great difficulty is sometimes experienced in differentiating between slow tuberculous meningitis and *tumors of the brain* (*q. v.*), as tubercle of the pons, or rapidly growing glioma. Moderately severe optic neuritis may suggest tumor or meningitis; intense neuritis, with choking, swelling and hemorrhage is indicative of tumor. Paralysis is more sudden in meningitis than in tumor.

3. From Hydrencephaloid (*see* Brain Anæmia).

4. From Hysteria.—Hysteria is marked by psychical alteration, limitation of the field of vision, anæsthesia and other stigmata. Hysterical strabismus is always spastic and convergent, and is often associated with small pupils; meningitic strabismus is often divergent (always a sign of organic disease), and the pupils are unequal.

Treatment.—Prophylaxis; the nose should be douched with a weak resorcin solution.

Flexner's Serum has given 75 per cent. of recoveries in 400 cases. The mortality varied directly with the time of administration; given on first to third day, it was 15 per cent.; fourth to seventh, 22 per cent.; after the seventh day, 36 per cent. mortality. The serum results are (*a*) the meningococci become more intracellular; (*b*) the exudate becomes less turbid or purulent; (*c*) leukocytosis decreases rapidly, even critically; and (*d*) complications are few and recovery is usually complete. The patient should be kept quiet in a dark room, the head elevated without flexion, an ice-bag applied to the head and spine, or an ice-pillow used. *Nutrition* should be maintained by feeding with the nasal tube or by nutrient enemata, if retraction of the neck causes dysphagia. The *bowels* should be mildly relaxed, and the bladder should be watched. *Vomiting* is treated as in acute gastritis (*q. v.*), but therapy is usually unavailing, for the cause is cerebral irritation; of drugs the bromides are in general most serviceable. Feeding by the rectum, sucking of ice, and the remedies in-

dicated for the headache give the greatest relief. Counterirritation by blisters and the use of the cautery are dangerous because the skin sloughs readily. *Headache,* restlessness and convulsions necessitate the use of opiates. The hypodermic use of morphine, or the use of the tincture of deodorized opium by mouth, is necessary, and free from harm; no other analgesic is equally potent. Ergot, to relieve congestion, never replaces the opiates. Atropine reinforces the analgesic action of opium. Chloral is used chiefly for the convulsions and for unrest. *Warm baths* were first recommended by Concetti and Aufrecht, and are considered almost specific by Netter. *Stimulation* is indicated when the heart weakens, and coma is imminent, until which time alcohol should be withheld. *Resorbents,* as iodides, are of little value. Mercurial inunctions are inefficacious although serous meningitis seems apparently benefited by them.

Lumbar puncture can permanently relieve only the simple or serous forms. Sudden death is much less likely to occur from repeated punctures than in uræmia or brain tumor. 20–40 cc. are withdrawn.

INFLUENZA (LA GRIPPE).

Definition.—A specific infective disease, caused by the Bacillus influenzæ, propagated by direct human intercourse but also by means of fomites; endemic over the entire world and almost yearly epidemic somewhere, characterized by its unparalleled rapidity in extension, the large number of people attacked, its protean symptomatology and its particular affinity for the respiratory tract.

Varieties.—(1) Influenza vera, (*a*) pandemic; (*b*) endemic or epidemic; due to Pfeiffer's bacillus. (2) Influenza notha s. nostras, pseudogrippe, catarrhal fever of unknown or variable bacteriology.

Pandemics originate in Asiatic Russia. No other pandemic has ever been so widely distributed as influenza, which spreads very rapidly but not faster than the fastest trains and steamers. The rapidity of its dissemination is due to the enormous virulence of the virus; to the universal susceptibility; and to the fact that many moderately sick individuals frequent cars, conveyances, churches, schools, and places of business. Epidemics develop gradually, usually following pandemics; they occur every few years whereas pandemics are separated by decades. According to Hirsch, the first epidemic occurred in 1173. The first pandemic seems to have been in 1510.

Etiology.—Though no age confers immunity, most cases occur between the twentieth and fortieth years. Physicians are very often attacked. The immunity usually conferred by one attack is less enduring and absolute than in other infectious diseases.

Bacteriology.—The organism discovered by Pfeiffer (1891–2) is the smallest bacillus cultivated. Its ends are rounded and it resembles a diplococcus when two bacilli lie end to end. It has no capsule, is non-motile, it lies free in the sputum in the early stages and later is found in the leukocytes, but often disappears early

from the sputum. The bacilli lie in lines "like schools of fish" (Finckler). Ten minutes are necessary for staining with carbol fuchsin. The bacillus is aërobic, short-lived, and very susceptible to drying. Culturally it grows characteristically on blood-serum as closely compressed yet discrete colonies, clear as water, and almost microscopic in size; it grows at body temperature and is easily overgrown by saprophytes. The germ has also been found in rare instances in the blood, pneumonic and encephalitic foci, peritoneum, and other parts, and possesses rather marked pyogenic characters. Its *atrium* is probably through the upper respiratory tract.

Symptoms.—There is no uniform clinical picture of influenza and certainly there is scarcely an acute infection with such varied grouping of symptoms and such varied sequels and complications.

After a *short incubation,* of one to three days, the grippe begins suddenly with chill and fever, headache, anorexia and depression, pains in the back and legs, respiratory catarrh, and intense nervous and sometimes digestive symptoms.

Types.—The form of infection varies greatly. In the *simple toxæmic type* there are coryza, moderate soreness of the throat, possibly some respiratory symptoms as cough and the toxæmia is evidenced by fever, some depression, headache and pains in the back and limbs. In the *severer toxæmic form,* the above named symptoms obtain but the patient suffers more profound depression both with the fever, and after it. Prostration, neuralgias and possibly cardiovascular atonicity mark the tardy convalescence. The *intensely respiratory* variety is attended by fever and toxæmia but the bronchitis is severe, perhaps intractable, and is not uncommonly followed by pneumonia: 75 per cent. of all influenzas are characterized by respiratory inflammations. The *gastro-intestinal* grippe is less a type *per se* than influenza attended by gastric intolerance and diarrhœa.

The *duration* is one to three days and the diagnosis is usually easy without the detection of the bacillus. Recurrences are frequent; most frequently, an initial respiratory catarrh is seen with nervous manifestations in the recrudescence but the order may be reversed. In rare instances influenza may cause chronic symptoms. Convalescence is marked by great nervous depression, neuralgias, and tardy return of vigor.

Special Symptoms.—1. Fever.—The rise is usually sudden to 103° or 104°; exceptionally it rises gradually. It is entirely atypical. Remittent fever is more common than continuous fever, which may suggest typhoid. It is occasionally intermittent, resembling malaria. Hyperpyrexia is observed particularly in encephalitis; some of these cases and certain nervous forms are afebrile (Eichhorst). The typus inversus has been noted (Pribrâm). The fever averages one to three or four days, and rarely lasts a week. Sometimes fever returns as a recurrence or relapse. Fever may last longer as a result of complications. Remarkable loss in weight may attend the fever.

2. Nervous System.—The nervous system is second in importance

only to the respiratory tract. The most frequent disturbances are functional and psychical but gross anatomical lesions are sometimes found. Headache is observed in nearly 100 per cent. of cases, and often is very severe. *Influenza comatosa* results from very acute intoxication, which may cause epileptiform seizures, tremors, or severe delirium. The post-influenzal *psychoses* are due to cerebral toxæmia or exhaustion and are observed five to eight times as frequently as in any other acute disease. An hereditary tendency is observed in 62 per cent., and most cases are seen in the young. They may occur from the initial intoxication but usually follow the attack. The majority are of the hypochondriacal type (exhaustion psychoses). Suicides were increased 25 per cent. in Paris during the last pandemic. In *encephalitis grippalis,* of which twelve cases are recorded, there are acute foci of inflammation usually discrete and multiple; they almost always occur in the gray substance of the cortex or ganglia and vary in size from the "flea-bite" spots of inflammatory hemorrhage with surrounding softening to the size of a cherry or pigeon's egg. The inflammation is due to embolism by the Bacillus influenzæ (Pfuhl and Nauwerk) or to encephalitis, which in turn is either primary, or secondary to foci in the nose, ear, and lung. Mono- and hemiplegia or sudden apoplectiform onset may mark the process, or there may be diffuse cerebral symptoms without definite focal localization. Encephalitis is usually fatal but recovery is recorded. *Meningitis grippalis* generally develops early. The virus reaches the brain by the blood stream or by contiguity through the *basis cranii.* It is basal or cortical. Lumbar puncture may show the grippe bacillus. Mya (1904) reported three cases of suppurative meningitis in which recovery occurred; the bacillus was found by lumbar puncture. *Spinal complications,* such as transverse myelitis, and acute poliomyelitis, have been observed. *Neuritis,* is usually post-influenzal. Pains are frequent in the back, legs and joints; neuralgia affects most often the fifth, sciatic, and intercostal nerves; neurasthenia, hypochondriasis, and hysteria may be observed as sequels.

3. The Respiratory Tract.—This is the atrium of the bacillus and is involved in 75 per cent. of cases.

Epistaxis is uncommon, though coryza is seen in 70 per cent. of cases. The bacillus may be found in the conjunctiva or the frontal or other sinuses. *Angina* is seen in 33 per cent. and *tonsillitis* in 60 per cent. of cases. *Laryngitis* (10 per cent.) occurs with hoarseness. *Tracheitis* (65 per cent.) causes pain, tenderness and severe paroxysmal cough, induced by irritation at the tracheal bifurcation.

Bronchitis is either diffuse, or more characteristically, occurs over one lung or one lobe. At autopsy are found great congestion, tendency to extension, hemorrhage, round-cell infiltration into the mucosa, and exudation and thrombosis in the smaller vessels, which causes necrosis of the mucosa. The sputum, according to Pfeiffer, is characteristically nummular and greenish-yellow. It is often blood-stained, contains the bacillus, is viscid and is often abundant. Rales

are most often found above the liver and near the lingual lobe. We also may observe dyspnœa, which is nervous or congestive in origin, without appropriate physical findings. Cyanosis is particularly dangerous in the young, aged and decrepit.

Acute *bronchiectasis* is not uncommon, but usually escapes clinical recognition.

Influenzal *pneumonia* occurs in 6 per cent. of the cases and is the *most important complication.* Pfeiffer's bacillus is its most frequent cause (Leichenstern and Wassermann). The pneumococcus, streptococcus, and staphylococcus, alone or combined with the Bacillus influenzæ, are the cause of some cases. A grippe epidemic may double the number of *lobar pneumonia* cases. When it complicates grippe, it is less completely lobar and granular.

Influenzal pneumonia, strictly speaking, is exclusively *catarrhal* and shows no fibrinous exudation or granulations, but merely infiltration of cells, which commences about the bronchi as multiple distinct foci with normal lung between them. It often causes minute abscesses. Mixed infection is of great importance in variations from this type. Clinically, the pneumonia is usually *atypical* and begins insidiously without a chill, but with increase of the prior cough and dyspnœa. It usually develops during or after the influenzal attack, although pneumonia may be the first sign of influenza. Its symptoms are more pronounced than its physical signs, and the disproportionate cyanosis and dyspnœa sometimes suggest miliary tuberculosis. Foci of dulness may be found but distinct bronchial breathing is rare; bronchophony is a much more reliable symptom. The heart is often weak and a peculiar redness of the face and head, profuse sweats at the beginning, and characteristic paroxysmal coughing are symptoms not seen in ordinary pneumonia. The foci are bilateral or multiple in 60 per cent. of the cases and the apices are involved very frequently. The symptoms and signs may intermit, or recur, as the consolidation wanders from place to place. The sputum is purulent or bloody. *Issues:* (1) In solution by lysis; (2) in death from early œdema pulmonum; the patient may die later, with early gray hepatization; the mortality is 20 to 30 per cent.; (3) in chronic influenza pneumonia, with hectic fever and suppuration, simulating tuberculosis; during a grippal epidemic, an increased mortality from tuberculosis is noticed; (4) in abscess or gangrene of the lung.

Pleurisy may follow pulmonary lesions or even the bronchitis. Primary pleurisy occurs in 27 per cent. of the fatal cases of influenza. It may begin early and its symptoms are frequently severe and protracted.

4. Circulation.—The heart-muscle is directly injured, especially its nervous apparatus. The lesser circulation is most involved. Weakness of contraction, syncope, even death, may occur during convalescence, especially from preëxisting fatty, valvular, or arteriosclerotic changes. The pulse is labile, often arrhythmic or dicrotic; it may be rapid, and out of proportion to the fever; cyanosis, dyspnœa

and bronchiolitis are frequent in such cases. A slow pulse occurs in 50 per cent. of the severer types, from toxæmic stimulation of the vagus. Acute endocarditis sometimes follows suppurating foci in the lungs; three cases are recorded in which the influenza bacillus was found. Acute phlebitis is seen mostly in severe cases with weakness of the heart, most frequently in the upper extremities. Arteritis is rare, occurs mostly in the popliteal vessels, and may end in gangrene. Leukocytosis occurs in not more than a third of the cases. Hemorrhages into the skin, serous and mucous membranes indicate severe intoxication.

5. Alimentary Tract.—Digestive symptoms are not important. The coated tongue is rarely dry or typhoidal. Vomiting (34 per cent.) which is sometimes bilious and persistent, and grippal hyperæsthesia of the stomach may be observed. Constipation is more frequent than diarrhœa. There are the usual febrile degenerative changes in the liver. Icterus is rare (2 per cent.). The spleen is enlarged clinically in but 15 per cent. of cases.

6. Genito-Urinary Tract.—Albuminuria may result from toxæmia or stasis (10 per cent.). Altered red blood-cells in the urine are rather frequent. Acute glomerulonephritis occurs in 1 per cent. of cases. Diabetes mellitus is said to have followed influenza; if influenza complicates an old diabetes, the prognosis is doubtful. Senator noted the frequent occurrence of the diazo reaction and of indicanuria. Cystitis, hæmaturia, menorrhagia, abortion and orchitis are but occasional complications.

7. Skin, Muscles, Joints.—Urticaria, herpes (5 per cent.), redness and hyperidrosis, miliaria, erythema nodosum or multiforme, scarlatiniform or morbilliform eruptions on the face or chest, or purpura, may be noted. Skin eruptions occur in 16 per cent. of the cases. Polysynovitis (7 per cent.), pyarthrosis and myositis are not common; bone involvement is infrequent.

Eye involvement (7 per cent.), conjunctivitis, keratitis, herpes, swelling of the lids, and otitis media (35 per cent.), which is usually purulent, and often hemorrhagic, are due to the pyogenic cocci, pneumococci and the influenza bacillus.

Diagnosis.—At the time of an epidemic the diagnosis is usually easy. Respiratory catarrh, headache, pains in the joints and back, nervous depression and tardy convalescence are significant. The diagnosis of the toxic, nervous and alimentary forms is more difficult and many errors may be made. The importance of bacteriological diagnosis has been over-rated, but positive results are decisive.

Differentiation.—Typhoid Fever.—The diseases may be confused when atypical severe influenza begins with gradual, or step-like rise of the fever and typhoid symptoms, such as tympany, enteritis, intestinal hemorrhage, swelling of the spleen and roseolæ. At autopsy, swelling and even ulceration of the stomach mucosa and Peyer's patches occur (Jürgens). However, coryza, conjunctivitis, the existence of an epidemic and early catarrhal symptoms, indicate influ-

enza, and the serum test and other cardinal typhoid findings (*q. v.*) decide the diagnosis.

Simple Coryza, Bronchitis, etc.—These are distinguished from true influenza by the sudden rise of fever in influenza, its nervous manifestations, pains in the head, bacteriology, spleen and exanthems. Lord found the bacillus of Pfeiffer in 30 per cent. of bronchitis cases in Boston, in which there was no suspicion of influenza.

Measles.—The early diffuse respiratory catarrh in each disease may cause confusion, and doubts may be increased by measles-like eruptions in influenza, but the typical measles fever curve (the fever rising again, after the prodromes, with the exanthem), and Koplik's spots, determine the diagnosis on the third or fourth day.

Miliary Tuberculosis.—This is suggested only in severe respiratory types; the history is usually distinctive.

Meningitis, apoplexy, the acute psychoses and acute poisoning are rarely simulated by influenza.

Prognosis.—(1) The morbidity is enormous, reaching 50 or even 75 per cent. of the population (pandemic of 1889–1890). (2) The mortality is $\frac{6}{10}$ to 1 per cent., but statistics are unreliable since the diagnosis of influenza is often carelessly made. It is certain that influenza increases the total number of deaths from pneumonia and tuberculosis, especially in the weak and aged. In some cases the course is chronic, the bacillus being found in the sputum for weeks or months, especially in tuberculous subjects, who are remarkably susceptible.

Treatment.—There is scarcely any *prophylaxis* except that persons should avoid crowds. Isolation should be practised in asylums, hospitals and prisons. Quinine is very uncertain. Gargles and nasal douches may possibly prevent infection and are distinctly indicated in grippal patients. There is *no specific treatment* and symptoms must be met as they arise. In every case, influenza should be treated as a serious disease and complications should be avoided by means of rest in bed and restricted diet. The treatment is much like that of a severe cold.

1. Pain.—At the onset, Dover's powder and acetphenetidin (phenacetin) āā grains x, at once, are given for pain. Phenacetin has been used to excess, but is superior to acetanilid and is less depressing. If these measures do not relieve the pain, the salicylates (or acetylsalicylic acid, aspirin) should be used as they are in rheumatism. Cold baths for high fever are poorly tolerated by the patient.

2. Catarrhal Symptoms.—For conjunctivitis, boric acid solution is excellent; for coryza, douches with Dobelle's solution, tincture of belladonna and of aconite in two- and one-drop doses, respectively, should be used every half hour for about three hours, and the alkaloid cocaine gr. j to albolene ʒj should be used locally; for pharyngitis, silver nitrate (20 per cent.) should be applied once or twice; for tonsillitis, nitrate of silver should be introduced into the follicles which are first opened and touched with Dobelle's solution; for bron-

chitis (*q. v.*), codeine or morphine is indicated; for enteritis, bismuth and paregoric āā 3j should be given after each bowel movement.

3. Severe Nervous Symptoms.—These are somewhat relieved by warm baths. Neuralgia necessitates the use of acetphenetidin, gelsemium, quinine, camphor, iron and arsenic. Headache is treated as it is in typhoid; it is increased by alcohol.

4. Heart.—The heart may require strychnine.

5. Convalescence.—Care in convalescence is important; cases of chronic tuberculosis or bronchitis require high altitudes.

PERTUSSIS (WHOOPING-COUGH).

Definition.—A specific, frequently epidemic, infection of the upper air-passages, occurring chiefly in children, and characterized by a cyclic course and a severe convulsive cough, which ends in a long-drawn, spasmodic inspiration or whoop. Cullen's definition was Morbus contagiosus, tussis convulsiva strangulans cum inspiratione sonora iterata, saepe vomitus."

History.—Pertussis was described by Ballonius (1578) and Willis (1674).

Etiology.—(*a*) *Age.*—It usually occurs in children and is most frequent at the fourth year. It is extremely infrequent after the twentieth year, though cases occur in middle and advanced life. (*b*) *Sex.*—Sixty-six per cent. of cases occur in girls. (*c*) *Anæmia* and respiratory catarrh are predisposing factors. (*d*) Most cases occur in March and April.

Koplik, Affanassiew and Hensel have described a facultatively aërobic bacillus which has small rounded ends, is but little larger than the influenza bacillus, and which occurs in clumps, but the microorganism has not been proven causative. One attack confers almost absolute immunity. *Incubation* lasts from two to five to eight days.

Symptoms.—1. Stage of Prodromes.—The *stadium prodromorum s. catarrhale* is characterized by headache, backache, photophobia, conjunctivitis, coryza, sneezing, angina, and by a cough which becomes dryer and harder toward the end of this stage. In a few cases there is moderate fever. This stage averages one week (one-half to two weeks), but is often shorter when marked epidemics prevail.

2. Convulsive Stage.—The *stadium convulsivum s. spasmodicum* dates from the first "whoop." The fever, aside from complications, ceases with the onset of this stage. The *seizures* are paroxysmal, convulsive, and are accompanied by dyspnœa and vomiting. As a rule the child is well except for the paroxysm, which has for an aura, tickling in the larynx or back of the sternum, thoracic constriction, vertigo, or a creeping sensation, and then the child braces himself, and terrified, runs for support. Then the explosion comes in the form of *three to ten or more short expiratory coughs* following *in rapid succession* until the breath is lost, when the "*whoop*" is

heard as a long, rapid, deep, sharp, singing or whistling inspiration. Then a thin, tenacious mucus is expectorated and vomiting constitutes the crisis of the paroxysm. The laryngoscope sometimes reveals laryngeal or tracheal catarrh. In severe cases there may be apnœa, great cyanosis, and usually diminution of the vesicular murmur. One to two minutes elapse between expiration and inspiration, and inspiratory and expiratory spasms or generalized convulsions may develop. The eyes are injected, the nose runs, the jugular veins are large and the skin is clammy. Involuntary evacuations occur; the pulse is small; ecchymoses sometimes occur in the conjunctivæ, scleræ, neck or face. After the attack, consciousness returns, respiration is fast, and there is fatigue, stupor, sweating, and pain in the abdominal or other muscles from the strain of coughing or occasionally from actual muscle rupture. The attacks occur largely at night but especially toward morning. In the free interval there may be euphoria or great depression, stomach disturbance, inanition, or diarrhœa. Leukocytosis is observed early in the disease, is unusually marked, and the increase is chiefly in the lymphocytes (Frölich). Frank S. Churchill states that lymphocytosis occurs in 85 per cent. of cases, and in 90 per cent. during the catarrhal stage. The number of the attacks averages about twenty daily.

3. Stage of Decline.—The *stadium decrementi* follows after three or four weeks, but may require months. The attacks become less frequent, but this stage is longer in proportion as the case is mild. From its complications (*v. i.*) it is the most dangerous stage.

Course.—The incubation lasts one week; the catarrhal stage one to two weeks, the spasmodic, three to six weeks, and the decline, three weeks. It is modified by the following complications:

Complications.—1. Increase or Extension of the Usual Symptoms.—*Catarrhal inflammation* is common in the initial stage,—stomatitis, laryngitis, catarrhal diarrhœa (10 per cent.), and otitis media (10 per cent.). Bronchitis (bronchiolitis) is not common in the spasmodic stage. It may occur with fever, a pulse of 120 to 150, respirations of 30 to 50, irregularity of breathing, many rales, cyanosis and atelectasis. *Bronchopneumonia* is the *most frequent and severe complication,* occurring particularly in rhachitic children between the third and fifth years; it most often occurs in the convulsive stage. The onset is gradual with fever of 100° to 102°, a pulse of 130 to 160, respirations of 30 to 80, with cyanosis and dyspnœa. The mortality may reach 25 per cent. and an almost absolutely bad prognosis must be given in the very young. Sometimes the pneumonia greatly lessens or entirely prevents the spasms (Trousseau). *Lobar pneumonia* is much rarer. Exudative pleurisy is a rare but very severe complication, and is seen almost exclusively in older children or adults. Endocarditis, pericarditis, meningitis and nephritis are very infrequent. *Spasm of the glottis* in nervous or scrofulous children rarely occurs before the fourth year. Its incidence is largely nocturnal, and it may cause death from asphyxia

even in the lightest cases. *Convulsions* of the carpopedal type, especially in nervous and hydrocephalic children, may occur at any time but are most frequent at the height of the apnœa, at the time of the first teething, and in the second to fifth week of the disease. The eyes are rolled upward and outward, the pupils are dilated, and coma follows.

2. Mechanical Complications.—*Hemorrhages* may occur into the *skin,* conjunctivæ, nose, ears and throat. Hemorrhage in the brain has produced paralyses, of which Valentine summarized 83 instances. Detachment of the retina and extravasation of blood into the lungs, stomach, and intestine, are rare. There may be *œdema* of the eye-lids, anasarca, rupture of the tympanum, or rupture of the *frenulum* of the tongue, the *ulceration* of which is almost constant. *Vomiting* occurs regularly at the end of the paroxysm as a mechanical crisis to the attack. Other mechanical complications are cardiac dilatation, pulmonary emphysema (less often interstitial than alveolar), bronchiectasis, pneumothorax, involuntary evacuations, prolapse of the rectum or uterus, aneurysm, muscular ruptures, hernias, rib fracture, and spinal deformity.

3. Toxæmia.—Marked toxæmia is rare. Parenchymatous degenerations are found in the fatal cases. Nephritis is not common.

Sequels.—The hysterical coughing and spasm which sometimes follow pertussis may very closely resemble it; rhachitis is an incidental sequel; *tuberculosis* may involve the lungs or peritoneal or mediastinal lymph glands, the latter signalized by retrosternal dulness; it occurs especially *after pertussis with measles,* both of which infections predispose to tuberculosis. Preëxisting tuberculosis renders the prognosis of whooping-cough very unfavorable. Tuberculosis, as a complication, develops in the third stadium, in children between the sixth and tenth years of age. The heart-muscle may suffer permanent weakness.

Diagnosis.—Diagnosis is easily made (1) by the definite stages of the disease; (2) by the presence of an epidemic; (3) by the absolutely characteristic convulsive seizures; (4) by stigmata, such as sublingual ulceration, swollen upper eye-lids, œdema of the face, petechiæ, and tender areas in the larynx; (5) pressing the tongue down and back with a spoon often elicits a characteristic attack of coughing. The stridor may be absent in nurslings and adults. The diagnosis may be difficult in intervals between paroxysms in the early or late stages. Intercurrent—especially febrile—diseases, like pneumonia or measles, may render pertussis atypical.

Differentiation.—In *croup* there is fever, aphonia, metallic inspiration, lividity and constancy of the symptoms, *i. e.,* no interval.

False croup (laryngismus stridulus) beginning with acute catarrhal laryngotracheitis, occurs at night in healthy children. *Spasmodic cough from disease in the bronchial glands* more closely resembles pertussis than any other cough.

Barthez and Sannée give the following differentiation:

Whooping Cough	*vs.*	Enlarged Glands.
1. Contagious, epidemic.		1. Isolated, not contagious.
2. Three periods, second paroxysmal.		2. No distinct periods.
3. Paroxysmal cough with whoop, vomiting and viscid expectoration.		3. Paroxysms, without the whoop, expectoration or vomiting.
4. Respiratory sounds normal.		4. Signs of enlarged glands sometimes present.
5. Respiration normal in interval; apyrexia if simple.		5. Asthma in some cases alternating with paroxysms. Febrile movements with recrudescence in the evening, sweats, progressive wasting, etc.
6. Voice natural.		6. Sometimes a change in voice.
7. Acute.		7. Chronic.

Spasmus glottidis occurs in nervous children at dentition, in rhachitis and tetany; it is a sudden spasmodic closure of the glottis without prodromes, but with dyspnœa, cyanosis and convulsions. *Asthma periodicum* (Millar's asthma) may develop in healthy children from the second to the sixth year with hoarseness, moderate fever, dyspnœa on the second day, with convulsions, cyanosis, and repeated seizures. Recovery occurs, or death, within the first eight days of the disease. *Hysterical "after"-pertussis* is characterized by the absence of catarrhal symptoms, fever, vomiting, sublingual ulceration, œdema of the face, petechiæ and a tender point in the larynx, irritation of which induces an attack. Hysterical imitation is associated with the stigmata of hysteria (*q. v.*), the influence of suggestion and a longer course than that of pertussis proper.

Prognosis.—Whooping-cough, with its complications, ranks third in the mortality of the English vital statistics of children. Hirsch states that, in Prussia, 85,000 deaths occurred from pertussis from 1875 to 1880, and in England, from 1857 to 1867, 120,000 died from the disease. The mortality averages 3 per cent., but varies with the virulence of the epidemic, and may even reach 15 per cent. The longer the time of development of the convulsive stage, the better is the prognosis, and the clearer are the intervals between paroxysms. The outlook is more unfavorable in girls, in the poor, in nurslings and in children under five years, especially in colored children. The mortality of cases in the first year is about 27 per cent.; in the second year, 14 per cent.; from the second to the fifth year, 3 per cent.; from the fifth to fifteenth year, 1.8 per cent. (Hagenbach); Toplitz found 41.7 per cent. of pertussis deaths in children under one year, and 48 per cent. in children between one and two years of age. According to Trousseau, when more than forty attacks occur in one day, a grave prognosis is indicated and when over 60 attacks occur a fatal outcome is certain. The writer, however, has seen recovery in cases with over 100 paroxysms daily. Coincident *measles, pneumonia* or bronchitis, especially if of early development, weak heart,

purpura, continued emesis, *glottis spasm,* eclampsia, *sinus thrombosis,* skin emphysema, encephalitis, brain hemorrhage, asphyxia, and the sequels of scrofula and *tuberculosis* render the prognosis very uncertain.

Treatment.—1. PROPHYLAXIS.—Isolation is difficult, but is indicated, particularly in tuberculous children. It is never advisable to expose a child to an epidemic. The greatest danger of infection exists during the catarrhal stage. The sputum, and the washing of clothes, etc., should be cared for on antiseptic principles. It was thought that vaccination mitigated or aborted pertussis (Archer), but in eleven personal unvaccinated cases, vaccinia had no prophylactic effect.

2. MEDICATION.—There is no specific, as proved by the multitude of vaunted remedies. All drugs should be employed cautiously. The following are of value: *Quinine,* the foremost remedy, was introduced by Binz, who gave 1½ times as many grains *per diem* as the child was years old; to children under two years, he gave ⅛ grain for each month. Children seldom object to the taste of quinine.

Widerhofer's formula runs as follows:

℞ Extr. belladonnæ ..... gr. iss.
Quininæ sulph. ..... gr. viij.
Sacchari albi ..... gr. xxx.
M. et divide in pulv. no. X.
S.—One powder three times daily.

Camphor, recommended by Jacobi, may be indicated as a stimulant in asthenia and in increasing bronchitis, ¾ grain at a dose. Cold tars should not be exhibited. *Bromine* should be administered as bromide or bromoform (Stepp). It is not curative, but moderates the course of the disease. Bromoform should be kept bottled in the dark and administered fresh from the stock solution each time—one to five minims in simple syrup.

The *catarrh* may be treated with expectorants and alkaline drinks. A steaming kettle somewhat alleviates bronchitis, a tent being constructed over the bed. Intralaryngeal insufflations of resorcin (1 per cent. with starch) are recommended. Blenorrhœa should be treated with benzoic and tannic acids (āā gr. ij, t. i. d.).

For the *paroxysm* there is no specific. *Belladonna* is given by Jacobi in the form of the extract, one-sixth grain to a child of six to eight months, until flushing is observed. Symptomatically it is also useful for the clammy, cool skin and for collapse symptoms. Though children tolerate belladonna well, great care must be exercised in very young children. The same caution is necessary with ipecac, chloral and Henoch's combination of belladonna and opium.

Chloroform inhalations are dangerous. *Codeine* is less effective than *morphia. Chloroform* (Schilling) is given with advantage in dry coughs, and twice as many drops may be administered as the child is years old, in one dram of warm water. It is said that the paroxysm

may be averted by pulling the lower jaw downward and forward. Yeo recommends the following formulæ:

℞ Morphinæ hydrochloridi ..... gr. ¼.
Sodii bromidi ..... ʒss.
Aquæ laurocerasi ..... ʒij.
Aquæ chloroformi .....ad ʒjss.
Misce, fiat mistura.
S.—One to three teaspoonfuls for a dose, according to the age of the child.

℞ Chlorali hydrati ..... gr. xvj.
Sodii bromidi ..... gr. xxxij.
Aquæ chloroformi .....ad ʒij.
Misce, fiat mistura.
S.—One to four teaspoonfuls for a dose, according to age.

℞ Sodii benzoatis ..... gr. lxxij.
Sodii bicarbonatis ..... gr. xlviij.
Ammonii chloridi ..... gr. xxiv.
Aquæ chloroformi ..... ʒj.
Aquæ anisi .....ad ʒiij.
Misce, fiat mistura.
S.—One to four teaspoonfuls, according to the age of the child, in a little hot milk, every four hours.

Digitalis is used by Koplik and Knight for right-heart dilatation. O'Dwyer recommends *intubation* for the spasm of the glottis. This is open to the criticism that ulceration might occur from the long-continued pressure in the larynx. *Bronchopneumonia* (*q. v.*).

3. Hygiene.—In warm climates pertussis is rare and runs a mild course. A suitable climate is likewise prophylactic against tuberculosis. The attacks are often lessened, or the disease quickly ended, by removal to the country. Such irritants as beef tea or stimulants, dry bread, cookies, and overfeeding or drinking, provoke coughing and vomiting in the convulsive stage. Food should be given at frequent intervals in concentrated form—gruels, milk with lime-water, zwieback in milk, eggs, malt, meat-juice, etc. Older patients tolerate more solid food. Rectal feeding is sometimes advantageous.

EPIDEMIC PAROTITIS (MUMPS).

Definition.—An acute specific infection, contagious, febrile, often epidemic, and characterized by a primary inflammation of the parotid glands.

History.—Parotitis epidemica was known to Hippocrates, who described every essential point, but later it was confused with other swellings of the parotid gland. Its distribution is world-wide.

Etiology.—(*a*) Its precise cause is unknown, although various microörganisms have been described. (*b*) Most cases occur between six and fifteen years of age; isolated cases are seen in adults. (*c*) The sexes are equally involved. (*d*) Most cases occur in the cold, moist, spring months. (*e*) Contagion is more frequently direct, from person to person, than indirect by a third party or by fomites. (*f*) It prevails largely in epidemics. (*g*) Immunity is conferred by one attack. It rarely prevails with the exanthemata.

Symptoms.—After an *incubation* of two and a half to three weeks, the disease begins with fever, followed in a few days by *parotid intumescence and pain.* In some cases slight fever, chilliness, and redness of the throat may exist before fever and parotitis develop. In other instances the severity of the epidemic determines such severe initial symptoms as vomiting, convulsions, or facial spasm in children; or delirium, prostration, diarrhœa, or a typhoidal condition in adults. As a rule, the fever rises at the onset to 101° or even 104°, the pulse averages 100, and in two days the *characteristic parotitis* develops. Its signs are clearly inflammatory (swelling, pain, temperature, and interference in function). The parotitis is bilateral, for the inflammation of one gland is rapidly followed by parotitis in the other. Indeed, Bouchoud said: "Mumps have no singular"; in rare epidemics the parotitis is unilateral (Laveran). Its degree varies and sometimes there is partial swelling. We know little of its pathology beyond what Virchow described—hyperæmia of the parenchyma, interstitial swelling, catarrhal secretion in the ducts and inflammation of the contiguous lymph-glands. The swelling about the gland may reach the eyelids, forehead, mastoid region, or even the clavicle, making the features unrecognizable and even invading the pharynx and larynx. The swelling is due less to periparotitis than to pressure on the facial and other veins. It is soft and does not pit. Compression of the masticatory muscles by the swollen glands makes chewing or opening the mouth difficult; chewing also elicits pain because it excites salivary secretion which often cannot pass the turgid ducts. Compression of the Eustachian tube or external meatus may occasion tinnitus. In the early unilateral involvement, the face inclines to the affected side in order to relax the swollen tissues; in the later bilateral parotitis, the head is often bent forward to the same end. The salivary secretion may be normal, increased or decreased. Suppuration is due to mixed infection. Leukocytosis is usually absent.

In severe cases the *submaxillary and sublingual* glands are swollen (even in 50 per cent. of some epidemics); in atypical cases they may be the sole seat of the disease. The parotitic swelling reaches its acme in three or four days and an equal period is necessary for its subsidence. The *course* is *one week,* perhaps two. The fever is remittent or intermittent, and falls by lysis. One to two weeks are necessary for convalescence.

Complications and Sequels.—1. The most important are *orchitis* and *epididymitis,* which were described by Hippocrates. On the sixth to the ninth day fever returns (102° to 105°), sometimes with chills and local redness. Swelling and pain in one testicle develop, often accompanied by general symptoms. Orchitis usually follows the parotitis, whence it was formerly considered metastatic, but in some epidemics, orchitis precedes or even occurs without parotitis. We must then explain mumps as a general infection, the blood carrying the virus first and most commonly to the parotid glands and

later to the testicle. A urethritis is sometimes observed, analogous to the catarrh of the parotid duct. Testicular localization occurs almost wholly in the sexually mature, active testis and therefore is most rare in children or the aged. Orchitis or epididymitis is usually unilateral. Comby observed it in 30 per cent. of mumps among French soldiers. Acute hydrocele is due to the epididymitis. Suppuration from mixed infection is very exceptional. Testicular atrophy occurs in 50 per cent. and if bilateral may occasion sterility. In women, ovaritis and vulvovaginitis are infrequent. Mastitis is rather more frequent, and may also be observed in men and boys.

2. *Other glandular organs.* Of these the lachrymal glands are most often affected, then the thyroid, with symptoms of acute Graves' disease, pancreas (1.5 per cent., Simonin), and the thymus. The spleen is seldom swollen.

3. *Other complications* are very rare. These are delirium, meningitis, paralysis of the cranial nerves; polyneuritis, or the neuroses; middle-ear disease, eye inflammations, optic neuritis; endocarditis; synovitis; albuminuria or nephritis; bronchitis or bronchopneumonia; necrosis, suppuration, or gangrene of the parotid or testis.

Diagnosis.—The diagnosis is usually easily made. Osteomyelitis of the jaw, periostitis, lymphadenitis, thrombo-phlebitis of the external jugular, and furuncle of the external ear, should be considered and excluded. Epidemic parotitis must not be confused with *secondary parotitis,* which results from trauma, or pus burrowing from the middle ear, or with mercurial, iodide or lead parotitis, and purulent metastasis in typhoid, pneumonia, and erysipelas, in which the pyogenic virus enters by the ducts or settles from the blood-current. In *post-operative parotitis,* after laparotomy, trauma to the gland, from holding up the jaw, may promote infection.

Prognosis.—The prognosis is good, for but 7 deaths occurred in 58,331 cases. The course is lighter in children than in adults. Sterility may result from orchitis.

Treatment.—The patient should be isolated. The fever is controlled as in other fevers, and diet and hygiene are the same. Complications are treated *symptomatically.* The patient should be kept in *bed* for one and a half weeks. *Pain* often necessitates the use of opiates. The local parotid or testicular inflammation is self-limited, and resists all attempts at abortion or mitigation. *Local heat* is soothing and better tolerated than the ice-bag.

ACUTE ARTICULAR RHEUMATISM (RHEUMATIC FEVER).

Definition.—An acute, febrile, non-contagious disease, with multiple articular involvement and with a tendency to heart complications.

Frequency.—McCrae's statistics, from Johns Hopkins Hospital, show that rheumatism constitutes 2 per cent. of admissions, as against 3.5 to 7 per cent. in London hospitals.

Etiology.—The evidence is strongly presumptive that rheumatism

is an acute infection. This is shown by its occasional epidemiology; its clinical picture; its tendency to recur like pneumonia and erysipelas; the autopsy findings of exudation, hemorrhage, cloudy swelling, and acute splenic tumor; the frequency with which an atrium may be observed, such as an angina, and its indubitable close affiliations with endocarditis, pericarditis, and meningitis, whose mycotic origin is clearly established. The *rheumatism virus is unknown.* Birsch-Hirschfeld, Triboulet, Poynton, Paine and others, have found bacteria in the joints and blood, but the organisms are neither constant nor uniform.

Singer likens rheumatism to septicopyæmia in that common symptoms occur in each—temperature, anæmia, articular metastases, endocarditis, leukocytosis, and peptonuria. He places particular stress upon the detection of pyogenic organisms in the rheumatic joints, the initial angina, endocarditis, erythema multiforme, etc. Chvostek and Egger explain the presence of organisms by bacterial invasion during the death agony, since bacteria could not be obtained from aspiration of the joints during life. The rheumatic arthritis is sometimes explained by toxæmia rather than by bacteriæmia.

Some writers question the "unity" of acute articular rheumatism, because the clinical picture is not always the same. Age modifies the disease, as is shown by the predominance of chorea, pericarditis, and endocarditis in childhood, with striking synovial subordination. The "genius epidemicus" changes the symptomatology, and the frequency of complications. However, no greater variations are noted than in other diseases,—*e.g.,* in typhoid.

Predisposing Factors.—(*a*) *Cold;* 75 per cent. of cases occur in the first half of the year (McCrae); (*b*) heredity in 25 per cent.; (*c*) male sex (70 per cent.); (*d*) age; the greatest susceptibility exists between ten and thirty years (50 per cent.); M. Miller collected nineteen cases in nurslings; (*e*) trauma, overexertion, exposure, and occupation, occurring frequently in sailors, divers, bakers, and day laborers.

General Clinical Picture and Course.—The onset is usually sudden, with moderate fever, redness of the skin, considerable prostration, drenching sweats and polyarthritis. The joints are involved in succession, the inflammation lasting a few days in one joint and then passing to many others, with frequent heart localization and the fever ending in solution by lysis. In adults and adolescents there is febrile urine, constipation, conjunctival injection, coated, moist tongue, some irritability, restlessness, and fever which is renewed as fresh joints are involved. There is marked anæmia and loss of weight. If endocarditis occurs valvular disease is the usual sequence. The usual duration without therapy is three to four weeks. Recovery is usual, but death may result from hyperpyrexia and brain toxæmia. A tendency to recur and a family tendency to rheumatism and valvular lesions are noted. The articular disease is directly influenced by the salicylates, but cardiac localizations are much less directly affected.

Symptoms in Detail.—1. Prodromal Symptoms.—These are angina or tonsillitis (estimated at from 2 to 20 per cent.), regarding which there are two conceptions; (*a*) that the angina is primary and rheumatic, and (*b*) that the rheumatism following angina is pseudorheumatism (polyarthritis anginosa), characterized by exanthems, occurring chiefly in the arms and reacting poorly to salicylates. Other possible prodromes are laryngitis, chorea, erythema nodosum, albuminuria or gastro-enteritis. Diagnostic doubts are settled by the appearance of the polyarthritis.

2. Polyarthritis.—(*a*) *Localization.*—The joints most frequently involved are those of the lower extremity, particularly the knee (53 to 84 per cent.); and usually one side is affected after the other. The ankles, elbows and wrists are the next most frequent localizations. Though usually found in the large joints, it may early involve the smaller articulations, such as the small bones of the carpus—thought by Trousseau to promote endocarditis. Rheumatism may invade the spine, jaw, sterno-clavicular joints, the synchondroses of the ribs, the sacroiliac or pubic articulations, and the larynx. Occupation has a bearing on the disease; for instance, rheumatism may occur in the arms of washerwomen. If a joint of an upper extremity is first involved, the opposite arm is usually next inflamed. (*b*) *In general* the arthritis is *ascending,* fleeting and almost invariably polyarticular. The characteristic erratic character of the arthritis was mentioned by the oldest writers as Van Swieten, who spoke of it as "migratory inflammation" or "fleeting gout." Though usually fleeting in its nature, the virus may settle in one joint for a period, but persistency in one joint always suggests pseudorheumatism (symptomatic or secondary forms). (*c*) *Signs and Symptoms.*—Polyarthritis is a better term than polysynovitis because the inflammation pervades all structures of the joint, the periarticular bursæ, cellular tissue, muscular insertions and tendons,—indeed, the external œdema is usually greater than the synovial effusion. The joints are swollen, red, tender and painful. Pain is the most prominent and frequent joint finding. Lasègue attributes pain not to the joint but to inflammation in the associated muscles and tendons, affirming that if these be voluntarily relaxed or suitably supported, the joint can be moved freely in all directions. Lasègue's phenomenon is said to be absent in gonorrhœal and surgical arthritis. The contiguous tendon reflexes are lessened or abolished. (*d*) *Duration.*—The inflammation remains in one joint from one to eight days, seldom longer, and its involution is shorter than its evolution. (*e*) *Sequels.*—The joints become normal, in fact ankylosis usually reflects doubt upon a previous diagnosis of rheumatism. Suppuration is extremely exceptional, and results only from mixed infection. Spontaneous dislocations are rarities.

3. Temperature.—The fever is directly related to the intensity of infection, the number of articulations involved, and to the development and progression of visceral complications. As Wunderlich

noted in his celebrated communication on fever, the temperature is usually highest when the patient is first examined. The fever rises with each new localization, be it arthritic, endocarditic, or pericarditic. The fever curve is in no wise cyclic, beyond undulation with each new manifestation, and is rarely higher than 102° or 104°. The temperature usually drops when the sweats occur. Prodromal fever was early noted by Todd, Graves and Fuller. The fever averages ten days, defervescence occurring spontaneously or with the salicylate therapy. The fever is longer when treatment is commenced late. Kahler held that fever could occur independently of joint inflammation (febris rheumatica). Pleuritis, renewed arthritis, angina, erythemata, embolic and other complications, prolong the pyrexia. Simple fixation of the joints reduces the fever, tachycardia and tachypnœa (Pribrâm). Chills suggest septicopyæmia rather than rheumatism. Hyperpyrexia is exceptional. With Dr. Lackner, the writer saw a patient with a fever of 112°, who recovered. Severe nervous symptoms, coma, convulsions, rapid pulse, transitory paralysis, stertorous breathing and hyperpyrexia constitute the "cerebral rheumatism"; 50 to 60 per cent. of cases of hyperpyrexia occur in the first attack of polyarthritis, with lesser frequency in later seizures. It is less frequent since the introduction of the salicylates.

The pulse is usually said to exceed 100, although McCrae found that its rate was not over 100 in 59 per cent. of his cases. Dicrotism and arrhythmia may sometimes be noted before involvement of the joints. The respiration follows the fever; Pribrâm noted one case with 80 to 100 per minute.

4. Heart Complications.—These are almost an integral part of rheumatism and constitute *visceral rheumatism* as distinguished from articular rheumatism. Heredity seems to be a factor in certain cases. Heart lesions occur as frequently in light as in severe forms. They vary somewhat with the epidemic, are most frequent and most dangerous at the time of puberty and increase in frequency with repeated attacks of rheumatism. Lasègue says: "Acute rheumatism licks the joints, pleura, and even the meninges, but it bites the heart."

1. *Endocarditis.*—This, the most vital complication, was first described by Pitcairn (1788), and Bouillaud, Pidoux, Besnier and Homolle developed the subject. Bouillaud (1832) formulated the statement that "in every severe case of acute rheumatism, endocarditis was the rule." The frequency of endocarditis is about 20 per cent. Mackenzie finds endocarditis in 58 per cent. of first attacks, 63 per cent. of second attacks, and 71 per cent. of third attacks. In McCrae's series we might divide the cases into thirds; in one third the heart is normal; in another third, it is doubtful; and in the last third, it is diseased. Hospital statistics, however, represent the severest cases. Clinically, endocarditis often escapes recognition, or is latent;—*i. e.*, it is found more frequently at the autopsy table than at the bedside. It is usually verrucose, and rarely ulcerative even in the fatal cases. Accidental or "functional" murmurs must not

be called endocarditis. To be certain that an endocarditis exists, two points are absolutely necessary: (*a*) Continued observation of the patient after convalescence; and (*b*) attention to signs of valvular disease (*q. v.*), other than the mere murmur, such as dilatation, hypertrophy, pulmonic accentuation, etc. Trousseau called attention to the possibility of the functional murmur, but Potain, almost alone among the present French writers, properly estimates its importance. The diagnosis of acute endocarditis, consequently, must long be uncertain, yet therapeutically we must protect the patient, and ourselves, by keeping him sufficiently long in bed. We may interpret the disappearance of a murmur in two ways,—first, as the disappearance of a functional murmur when the fever falls, the toxæmia disappears, the heart slows, dilatation subsides, or the anæmia mends; or, second, as the healing of a slight endocarditis, though this is always the less probable issue.

Valves Affected.—McCrae finds the mitral valve involved in 95 per cent. of the cases, the aortic in 23 per cent., and both valves in 18 per cent. (Aortic lesions are usually estimated at 12 per cent.) The fever may be continuous with that of the arthritis but is often higher. Less often the fever is remittent with severe rigors and may require differentiation from malaria, pyæmia, or gonorrhœal endocarditis. A low remittent fever may suggest pulmonary or serous membrane tuberculosis. The endocarditis exceptionally antedates the polyarthritis (Jaccoud, Trousseau).

Masked or atypical rheumatism embraces rheumatism masquerading under the guise of an endocarditis or trifacial neuralgia and yielding to salicylate treatment (Immermann). Kahler's opinion of rheumatic fever without arthritis has been mentioned. It is claimed that rheumatic pericarditis or endocarditis occurs without articular inflammation—" polyarthritis rheumatica *sine* arthritide." We may call this the " rheumatic equivalent," borrowing from the nomenclature of epilepsy.

In *old heart lesions with renewed rheumatism,* lingering fever suggests the possibility of an exacerbation of an old endocarditis aroused by the last rheumatic attack. Various embolisms may occur. The bacteriological findings are most varied, and not knowing the actual cause of rheumatism, we cannot say exactly what is rheumatic and what is mixed infection.

2. *Pericarditis.*—Osler states that rheumatism explains more cases of pericarditis than all other causes combined. Its frequency is given as 5 to 10 per cent. Pericarditis is very frequently latent. In children, especially, a subacute or latent course is most common. Baginsky found that adhesive pericarditis was the most common autopsy finding in rheumatic children.

3. *Myocarditis.*—Disturbance of the heart in the acute stage is almost certainly myocardial, and toxic or mycotic. Acute interstitial myocarditis (in 28 per cent.), coronary disease, myocardial invasion by contiguity from endocarditis in the later stages, and sometimes

the use of salicylates, are possible causes. Recovery is usual, but chronic fibrous myocarditis is a not uncommon sequel. Acute myocarditis may produce a systolic murmur, suggesting an early endocarditis. Myocarditis may cause sudden heart pain with weakness, and possibly death,—the "heart rheumatism" of the laity. Transient palpitation, cardiac oppression, or tachycardia, may occur. Bradycardia with vertigo and syncope is probably myocardial.

As a practical point, it must be remembered that the right heart may be dilated as in other fevers and various abdominal affections; this dilatation disappears with convalescence. When accompanied by a murmur it may lead to an incorrect diagnosis of endocarditis. The bloodvessels are rarely inflamed, but French writers maintain that acute arteritis is common; severe pain over the artery is followed by paræsthesia and pain over its smaller branches in the extremities; pathologically only part of the vessel-wall is affected and healing usually follows, but gangrene may develop after complete arterial occlusion. Endophlebitis is somewhat more common.

5. Skin.—Sweating is most profuse, constant and characteristic; in odor it is sour or mouse-like (Schönlein); in reaction it is first acid and later neutral or alkaline. The skin is frequently macerated and *sudamina alba* are common (in 4 per cent., *sudamina rubra*). *Rheumatic nodes* (first described by Hilliers, Meynert, Hirschsprung, Barlow and Warner) are subcutaneous nodes, as large as buckshot, on the fingers, hands, wrists, elbows and spine. They may appear during or after an attack, or independently of acute seizures. They occur especially in children, but are also observed in adults, particularly in women. They are abundant, sometimes numbering even sixty, are symmetrical, hard, and are usually tender. Sometimes osseous or arterial endothelial proliferation is observed. Nodes occur most often in chronic cases or in cases with severe endocarditis or pericarditis. If very numerous or large they indicate an unfavorable prognosis. In contradistinction to these more fixed nodosities, Féréol has described more fugitive and less indurated nodes (*nodositiés cutanées éphémères*). Œdema of the skin may be septic, rheumatic, or angioneurotic. Erythema polymorphum is generally regarded as an independent affection but in rheumatism the nodose form occurs. Some cases are possibly septic or anginose eruptions. The salicylates relieve some cases and fail to benefit others. Morbilliform or scarlatiniform rashes are infrequent, and may be attributed to errors in diagnosis, or to drug eruptions. Purpuric eruptions are not uncommon. The intestinal hemorrhage, colic, and vomiting, with articular intumescence, described by Henoch, are entirely distinct from genuine rheumatism and will be considered under Purpura (*q. v.*).

6. Muscles and Bones.—*Myositis* is infrequent, though *myalgia* is common. Muscular atrophy may follow inflammatory extension from the joints to the muscles. "Muscle scars" are not frequent, and are much more often due to trauma, or other infections, than to rheumatism. *Periostitis* may occur, and some cases of albuminous periostitis are clearly rheumatic.

7. Respiratory Tract.—*Laryngitis* may be prodromal and an atrium for the rheumatic invasion, or may occur later as diffuse catarrh or as local nodes. The rare acute œdema of the larynx is most dangerous. *Pleurisy* may result from the rheumatic virus or secondary infections. Its frequency is 2 to 3 per cent. A primary pre-arthritic pleurisy is very rarely observed. Pleurisy and pneumonia are usually associated with heart disease. Pleurisy is found in 54 per cent. of cases of endopericarditis (Pribrâm). The writer believes that the so-called rheumatic pleurisy is almost always tuberculous (see Etiology of Pleurisy). *Pneumonia* occurs in but one-tenth of 1 per cent. of cases of rheumatism not involving the heart, and in 66 per cent. of cases with endopericarditis. Howard found lung and pleural complications in only 10 per cent. of cases with rheumatic endocarditis, in 58 per cent. of those with pericarditis, and in 71 per cent. of those with endopericarditis. Many cases are called pneumonia which are pulmonary infarction, stasis, or compression from pleural or pericardial exudates.

8. Digestive Tract.—Angina or tonsillitis occurs as a prodrome (*v. s.*) or as a later development. The tongue is coated and moist except in the severest cases where it is dry. Gastric symptoms are rare, though Damash considers them the most frequent prodrome. Constipation is the rule. Diarrhœa occurs as prodromal enteritis or as a rare complication. Peritonitis, icterus and liver enlargement are most exceptional.

9. Genito-Urinary Tract.—*Albuminuria* is mostly transitory. It occurs late in the course or after several attacks, and mostly in individuals over thirty years of age. The percentage ranges up to 34 (McCrae). Albumosuria is frequent, especially during resorption, whence it has a slight diagnostic value. Cylindruria may occur without albuminuria. Renal infarction usually occurs late in the course of cardiac cases. *Acute nephritis* is seldom seen ($\frac{3}{10}$ to 1 per cent.); it occurs early, and chiefly in endopericarditis, and is usually benign but may become chronic. But few of the author's cases in private practice show nephritis, though it is present in 50 per cent. of hospital patients; it is probably latent until awakened by the fresh toxæmia and is probably due to exposure and alcoholism. Hæmatoporphyrin has been found and the characteristics of fever urine, and reduction of the chlorides. Cystitis may result from cantharides blisters. Amenorrhœa is usual. Hemorrhagic endometritis and genital hemorrhage are infrequent.

10. Blood.—The blood in low dilutions, is said to respond to the Widal reaction. The red disks are reduced 25 to 30 per cent. and chlorotic characteristics are noted. The fever, the anæmia and the intensity of articular involvement run more or less parallel. The white cells, in moderate cases, range between 10,000 and 15,000, and higher figures (20,000) are only reached in extensive endocarditis, pericarditis, pleurisy, pneumonia, or in septicæmic or suppurative arthritis. The increase is in the polymorphonuclear neutrophiles

while other cells are normal or decreased. The eosinophiles are practically absent, but increase with betterment of the articular findings. If they are increased in a florid case, the prognosis is good, since the course will probably be mild. Bacteriological findings are negative.

11. Nervous System.—The relation of chorea to rheumatism is an unsettled question. *Chorea,* rheumatism and endocarditis are associated (in 50 per cent. of cases, Pribrâm), or coördinated, and possibly a common, though unknown, infection is the essential correlating cause. A constant anatomical cause of chorea, as arterial change, encephalitis, or embolism, is not known. Cases with rheumatic or endocarditic association usually run a slow and often a relapsing course; cases which react to the salicylates are probably rheumatic. *Cerebral rheumatism* is very infrequent; it may develop acutely at the acme of the disease, with psychical alteration, often with hyperpyrexia, sometimes with warnings, or again precipitately. Delirium, unrest, convulsions, and coma, frequently result in death. High fever may be solely a cerebral symptom, but cerebral symptoms may occur without high temperature. The course is *foudroyant* or lasts several days, and even a week. The author has seen two cases of recovery after mania, convulsions, meningeal symptoms, and coma. Chronic *psychoses,* hallucinations, mania, melancholia or convulsions, may occur in convalescence. They are usually due to inanition and the patient generally recovers. Brain *embolism* (from endocarditis), hemorrhage and rheumatic meningitis, are infrequent. *Spinal rheumatism* may be confused with myelitis, septic or gonorrhœal meningitis, multiple neuritis, local myopathies adjacent to the joints involved, or cerebral embolism. In Kraus' clinic the author saw one fatal case characterized pathologically by meningomyelitis. *Multiple neuritis* is often rheumatic; it may precede the polyarthritis, but usually occurs in the first or second week of rheumatism and presents the usual characteristics, such as pain and tenderness over the muscles and nerve trunks, paralysis, muscle atrophy, reaction of degeneration, anæsthesia and lost reflexes. The prognosis is good and the neuritis reacts well to electrotherapy. Mononeuritis also occurs and usually regresses entirely.

The Eye.—Rheumatism may result in benign episcleritis, iritis, iridocyclitis, and possibly conjunctivitis. Optic neuritis, with or without atrophy, and retinal embolism or thrombosis occur exceptionally; their prognosis is doubtful.

Acute *thyroiditis* is most uncommon. Lymphadenitis is not uncommon, but is rarely marked or extreme. It occurs with arthritis, erythemata, endocarditis or pericarditis.

Prognosis.—The prognosis as to life is usually excellent and is generally proportionate to the severity and duration of the infection and to the number of joints involved. The percentage of deaths is 1.5 to 3 per cent. but these hospital figures are higher than in general practice. In statistics other arthritides are not carefully excluded. There are waves of increased mortality every three or four years.

In late years the death-rate has somewhat decreased. The *causes* of *death* are (1) hyperpyrexia, the most important; (2) cerebral rheumatism, chorea; (3) peri-, endo- and myocarditis; (4) pneumonia and lung infarcts. *Recurrence* occurs in 19 per cent. of cases or in a larger percentage in general practice. They are more dangerous in pregnancy or with complications such as influenza, etc. The prognosis is *less favorable in children* than in adults. The salicylate *therapy* influences the disease and also its relapses; but the heart complications are only slightly relieved. At the onset it is impossible to declare the case light or severe. According to Trousseau, the involvement of the small joints of the hands and feet predict a severe course.

Diagnosis.—1. Rheumatism in Childhood.—In the very young the disease is most rare (3 cases in 85,000 nurslings) but becomes more common after the fifth year. The course is more rapid and the urine is normal or less febrile. Generally speaking, the joints are less susceptible and the arthritis less intense, while the skin, nervous system, and heart, are more often involved. Heredity is most potent in the young, and the sexes are equally involved. The various phases of rheumatism occur separately,—first arthritis, then endocarditis, later chorea, subcutaneous nodules, pericarditis, and so on. The child rarely lies quietly, but tosses about; the tendons and fasciæ are more often involved than the joints. The pain is less and the arthritis may entirely escape recognition. The lower temperature is particularly remarkable at this epoch of life, when high fever is often excited by trivial lesions. There is much less sweating. The anæmia in childhood is especially rapid and marked. In children the differential possibilities are *acute osteomyelitis,* acute pyæmia, monarticular arthritis, especially of the knee and hip in nurslings, gonorrhœal arthritis, Barlow's disease, syphilis, and hæmophilic joints; if the rheumatism occurs in the spine, meningitis or torticollis are of differential importance. Endocarditis is usually not so clearly nor so immediately connected with the polyarthritis as in adults, but it develops more slowly, insidiously, later, and twice as frequently as in adults (80 per cent. Vernay). Thomas Watson knew but three instances of acute rheumatism, before puberty, which escaped severe heart disease. This is due to the greater functional demands on the child's heart and to the aorta being narrow at the isthmus. Endocarditis occurs relatively early in what Cheadle terms the "rheumatic progression," but pericarditis occurs later after the valvular lesion has developed. Pericarditis is almost always found in necropsies on children with valvular disease (Sturges). Rheumatic heart disease in general at this epoch, leads to greater hypertrophy because of greater nutrition, to more marked dilatation because of greater tissue elasticity, and to more frequent recurrence. The prognosis is therefore much more unfavorable than in adults. In the diagnosis, functional murmurs are not frequent in children. The pulse is often irregular, the precordium more readily bulges;

left-sided pleurisy is more frequent than in adults, and rheumatic nodes are more common. Oppression over the heart is infrequent, as are pain, cyanosis, dropsy, hepatic congestion, pulmonary infarction, dyspnœa and fever. Rheumatic children are more nervous. More marginate, papular and urticarious erythemata and more purpura develop in children. Large subcutaneous nodes (rheumatismus nodosus) are serious prognostically and always suggest danger from severe intercurrent valvulitis. Chorea is prone to develop with arthritis, heart disease, nodes, or with the erythemata.

2. Rheumatoides.—Rheumatoides (Gerhardt) or *pseudorheumatism* (Bouchard) are symptomatic arthritides resembling rheumatism, in that fever, arthritis, or endocarditis, may occasionally develop in their course. The nomenclature of Quincke, who speaks *polyarthritis gonorrhoica, scarlatinosa, etc.,* is the least objectionable. (*a*) Polyarthritis *gonorrhoica* resembles rheumatism most closely and most frequently. König holds that the most common of all joint diseases is the gonorrhœal (*v.* Gonorrhœal Infection). (*b*) Polyarthritis *scarlatinosa* often most closely resembles rheumatism, occurs most frequently in the third to fourth week, and varies with the epidemic. In 66 per cent. of patients it occurs in the hands, and lasts four days, frequently with endocarditis or pericarditis (32 per cent., Hodges), and sometimes with recurrence and serous or purulent exudation. With it have been observed nodes, erythema and chorea. (*c*) Polyarthritis may also complicate many other diseases, including most of the infections; *e. g.*, measles, smallpox, erysipelas, typhoid, angina, appendicitis, diphtheria, syphilis, pneumonia, etc., etc.

3. Severe Types.—In grave types the visceral findings are least confusing when secondary, both in development and degree, to the arthritis. Jaccoud speaks of the primary visceral type, wherein the findings (*e. g.*, cardiac) antedate the arthritis, but the diagnosis remains uncertain, until the joints are involved.

4. Gout (*q. v.*). 5. Arthritis Deformans (*q. v.*). 6. Tuberculous Polyarthritis (*q. v.*).

The *number of joints* involved is always highly suggestive, but not finally diagnostic. True rheumatism and gonorrhœal rheumatism are often polyarticular. Diagnosis of gonorrhœal rheumatism in women is often difficult for notable reasons. In women, puerperal infections, and an acute monarticular osteoarthritis, beginning acutely from exhaustion in pregnancy or lactation, may cause difficulty. In growing children, *acute osteomyelitis* may cause articular swelling, which, however, is monarticular.

Treatment.—1. Diet.—The diet should be limited to fluids, arrowroot, milk, gruels, etc. Biot's results, with a strict milk diet, were extremely good. Antecedent tonsillitis indicates tonsillectomy.

2. Clothing.—The patient should wear a flannel gown and lie between blankets, which are more comfortable than sheets on account of the sweating. Care must be taken lest the drenching sweats macerate the skin. This can be avoided by dusting talcum powder in the

axillæ, between the toes, etc., and by sponging the body with warm water and alcohol.

3. Local Treatment.—Fixation lessens pain, fever, pulse and respiration-rate. Cold applications, boric fomentations, and salicylic salve are useful (Acidi salicylici ʒj, adipis lanæ hydrosi ℥j). Davies' cantharides blister and the actual cautery are indicated for tardy involution.

4. Rest.—The patient should lie in the most comfortable position. Splints afford relief to the muscles and joints, but muscular atrophy may result from the use of plaster-of-Paris casts. The absolute dorsal decubitus, long into convalescence, or at least for two weeks, must be enforced because of the weak condition of the heart.

5. Salicylate Treatment.—Buss (1875) remarked the antipyretic action of the salicylates, and Strickler (1876) first discovered their remarkable action in rheumatism.—(*a*) *Effect on the Arthritis.*—The pain and swelling are usually relieved after three to five full doses or within twenty-four hours after taking the first dose. Salicylates are the best analgesic in rheumatism. Hypodermics of morphine are rarely necessary except, perhaps, in the first twelve hours. Salicylates are thought to cause articular hyperæmia.

(*b*) *Effect on the Temperature.*—The fever falls coincidently with relief of the articular symptoms, and reaches normal in two days. In the great majority of cases the acute stage is over within three days. In hyperpyrexia less influence is noted, especially if the salicylates are administered late.

c. Effect on the Endocarditis.—The weight of opinion is that endocarditis occurs as frequently with the use of salicylates,—*i. e.,* salicylates are analgesic and antipyretic but not strictly antirheumatic. Many, however, claim that the salicylates are in every sense antirheumatic. The writer believes that the salicylates are practically specific and that heart complications are lessened by shortening of the acute stage.

d. Relapses.—Relapses are said to be more frequent with the administration of the salicylates than with the alkaline treatment, and especially so when treatment is discontinued early. This claim seems unfounded. Relapses are apparently less frequent with the continued use of salicylates for two weeks after the initial symptoms are relieved.

Physiological Action of the Salicyl-preparations.—They are absorbed from local applications. The smallest doses produce symptoms of cinchonism, as fulness of the head or roaring in the head and ears. After larger doses dulness, headache, giddiness, a flushed dusky and moist skin, disturbed sight and hearing, and tremor, develop. Toxic doses produce the above symptoms, plus eye-muscle paralysis, pupil dilatation, deafness, increased reflexes, restlessness, delirium, dyspnœa, disturbed circulation, skin eruptions, sweating and also green urine (indican and pyocatechin). Post-mortem, visceral congestion and serous ecchymoses are seen.

Administration.—Sodium salicylate is the best preparation. Its action is most prompt and certain; it is soluble and least irritating. Where it fails, other preparations are usually unsuccessful. All salicyl-preparations are absorbed as sodium salicylate produced by the alkaline intestinal juices. Grains xv of the natural acid, which is better than the synthetic, or grains xx of the sodium salt, are given every two or three hours until *salicylism* appears, *i. e.*, until slight *dulness, dyspnœa* and *deafness* develop. This action is maintained for twelve hours, when the interval between doses is lengthened to four or six hours. The effects are better in proportion as the disease is more acute, the symptoms more pronounced, the inflammation greater, the number of involved joints larger, and the fever higher. It is well tolerated by children and usually can be given by mouth in fluid, if the taste be disguised by wintergreen oil, syrup of ginger, and syrup of orange. Even then, however, it is frequently nauseating.

℞ Sodii salicylatis ..... ʒvj.
Sodii bicarbonatis ..... ʒiv.
Syrupi zingiberis ..... ℥j.
Syrupi aurantii ..... ℥j.
Aquæ ..... q.s. ad ʒiv.
M. et S.—Shake. One teaspoonful every three hours.

Theoretically it should not be given in capsules, for in this form it may irritate the stomach, but it may be given in powders which are taken in starch wafers. As in cinchonism, bromide of potassium may be given to lessen the ringing in the ears. In case the stomach is intolerant, salicylates may be administered in pepsin, or given by rectum in peptonized milk. When given as an inunction, it is soon found in the urine, but absorption by the skin is uncertain. Aspirin (acidum acetylsalicylicum) is much less irritating to the stomach than salicylate of soda, less antipyretic and less analgesic but more sudorific. It is given in doses of ten to twenty grains at the same intervals. Salol (phenylis salicylas) is given in the same dose at the same interval, but the kidneys must be carefully watched; it is far less efficacious.

The *alkaline treatment* is said to decrease the heart complications and shorten the disease. Though these claims are not established, the alkaline treatment may be used in conjunction with the salicylates, using vichy salts with enough citrate of potassium or bicarbonate of soda to make and keep the urine alkaline. Long ago Fuller advised the following:—

℞ Sodii bicarbonatis ..... ʒss.
Potassii acetatis ..... ʒss.
Liquoris ammonii acetatis ..... ʒiij.
Aquæ ..... ad ʒij.
M. f. haust.
S.—To be taken in effervescence with—

℞ Acidi citrici ..... ʒss.
Aquæ ..... ℥ij.

Baginsky thinks the administration of gr. xv to xl of potassium iodide daily produces better results in children than do the salicylates.

Unusual Results.—Unusual results of salicylate treatment:

1. *Nervous.*—Salicylate delirium has been observed in anæmic girls, neurotics, and in alcoholics. It may be violent and protracted. Convulsions are quite infrequent; they are centric.

2. *Cardiac.*—Small doses stimulate the heart, and cardiac depression is observed only when the largest dosage is given, when the preparations are impure, or when the heart is already diseased. If the heart becomes weak or irregular, acute myocarditis is probably developing and salicylate treatment must be suspended, the dose must be decreased, or the heart muscle must be stimulated by coffee, strychnine or alcohol. Slight irregularity without weakness is not a contraindication. Cardiac depression may be noted in nervous subjects. Thorne has observed collapse from aspirin.

3. *Respiratory.*—With antecedent or concomitant bronchitis, the lungs should be explored for possible congestion which may result from the remedy. Cardiac depression, sudden respiratory failure and violent expiratory dyspnœa are observed in a few cases. Tuberculous patients should be carefully watched.

4. *Renal.*—Caution is necessary in old nephritics, because the salicylates are excreted by the kidneys as salicylic, or salicyluric acids; however infective albuminuria, directly due to the rheumatic virus, indicates, rather than contraindicates, the use of the salicylates. Binz believes that the salicylates may induce abortion.

5. *Alimentary.*—Vomiting (*v. s.*) and diarrhœa may result from salicylate treatment. Digestive disorders are partly due to inhibition of the digestive ferments. Constipation is said to favor salicylism.

Diagnostic Therapy.—Senator claimed that sodium salicylate is specific in true rheumatism and inert in pseudorheumatism. Though the general truth of this statement is acknowledged, its application is not absolute. Some rheumatic patients resist salicylates. Salicylates are of some value in the arthritic type of grippe. They usually are useless in other arthritides.

Convalescence.—Absolute rest must be enforced for two to four weeks to save the heart.

DENGUE.

Definition.—An acute specific infection, occurring chiefly in tropical and subtropical countries, extremely contagious, and characterized by fever, an initial prodromal eruption, violent articular and muscular pains, and a secondary polymorphous eruption.

Etiology and Epidemiology.—The actual cause is uncertain. A small diplococcus or bacillus resembling the Bacillus influenzæ has been described. Graham found, in the blood-cells, an organism resembling the Plasmodium malariæ, but small and unpigmented. For this reason infection has been connected with mosquito-bites. Eberle

describes a "plasmeba." The disease is very contagious and seems disseminated less by direct than indirect infection, *e. g.*, by fomites, bites, etc. It is carried by pilgrims, sailors, and emigrants. The disease spreads with remarkable rapidity and infects large numbers of people. Its home is in the tropical and subtropical zones and it prevails chiefly in the hot months. It has invaded Europe, Asiatic Turkey, South America and the United States. It is remarkable that the coast ports are alone invaded. It very seldom reaches inland, and even in regions where the disease prevails endemically, closely contiguous inland towns and higher places are exempt. Unhygienic conditions promote its development. In the first Philadelphia epidemic it was called "Front Street Fever," on account of the squalid condition of the houses along the river front. The first epidemic, in 1779, in Java, was described by Brylon, and Benjamin Rush wrote of the Philadelphia epidemic in 1780. The chief later epidemics or pandemics occurred in 1818, 1824 to 1828 (reaching Savannah and New Orleans), 1830 to 1870 (again reaching America and seen even as far north as Ohio), 1876 (Virginia), 1889, 1890, 1895 to 1897. The morbidity is enormous but the mortality is strikingly inconspicuous. Epidemics last two to five months. No immunity is conferred by one attack and two, three or even four, infections have been observed.

Symptoms.—After an incubation of one to two days, or sometimes a little longer, the disease *begins suddenly.*

1. Febrile Stage.—The fever rises quickly to 102°, 104° or even 107°, usually with a chill, severe frontal headache, and sudden intense pains in the back and limbs, which incapacitate the patient and involve especially the knees. The names "breakbone fever," first mentioned in Rush's description, and "dandy fever," given because of the mincing gait which results, bring out this salient feature. Many large and small *joints* are *swollen* (pseudorheumatism), one after another. The *muscles* are *painful* and the skin is sometimes hyperæsthetic. The pulse averages 100. The *face is suffused* and swollen, the conjunctivæ are injected, the flow of tears is increased, and in some cases there is coryza. Total anorexia, foul breath, coated tongue, and great thirst, develop; vomiting is occasional and constipation the rule. The *premonitory hyperæmic rash* may last but one to five hours, and is frequently absent. The urine is febrile. Prostration may be marked, and, in some cases, severe delirium, convulsions, and coma may develop. This stage lasts three days ("three-day fever") and ends with profuse sweating. Fever is absent in the lightest cases.

2. Exanthematous Stage.—The eruption begins on the fourth day chiefly on the face, hands, forearms and chest, and is decidedly polymorphous. It is scarlatiniform, morbilliform, urticarial, erysipelatoid, lichen-like, vesicular, or even pustular. It is present in 60 to 100 per cent. of the cases. It lasts two to three days, and is followed by fine desquamation, intense itching, sometimes by falling

of the hair, and by cutaneous hyperæsthesia. The throat is sometimes red or even inflamed, and occasionally lymph-gland enlargement, parotitis and orchitis occur. The fever in this stage may be elevated, may be absent, or subnormal temperature be noted. The pain often recurs. Herpes, splenic tumor, otitis, albuminuria and leukocytosis are absent.

3. Convalescence.—Convalescence begins in five to seven days, after a crisis or lysis, and is very tardy. There is great prostration, gastric uneasiness, insomnia, emaciation, and anæmia, all of which are remarkable after such a short illness. In no other disease of equal duration is such marked constitutional disturbance noted.

Complications and Sequels.—These are great rarities. Cutaneous or gastric hemorrhage, neuralgia, endocarditis, pericarditis, nephritis, pneumonia, pleurisy, œdema or ulcers, rarely develop.

Prognosis.—Death is rare, though the morbidity is enormous. The highest recorded death-rate was ½ of 1 per cent. One death occurred in 7,435 cases in the Anglo-Indian army (1872), and 10 deaths in 100,000 cases occurred in Smyrna. Relapse may occur in one to two weeks.

Diagnosis.—The geography and the presence of an epidemic are suggestive. The exanthemata, rheumatism, insolation, yellow fever (*q. v.*) and epidemic *erythema exudativum multiforme* may be suggested. *Influenza* is differentiated by its epidemic-pandemic incidence, its occurrence in all climates, seasons and locations (inland as well as on seaboard), its more frequent direct infection, the more general immunity conferred, the higher coincident mortality (pneumonia and tuberculosis), its more frequent respiratory catarrh and lung complications, its more severe nervous, intestinal and cardiac symptoms, and the greater frequency of herpes, splenic tumor, otitis media, and albuminuria. In the rare cases of *acrodynia,* observed in France, Belgium, Turkey, Persia, and Mexico, Scheube gives the following differential points: (1) Digestive disturbance; (2) facial swelling; (3) pains in the extremities, sometimes with anæsthesia of the soles; (4) spastic symptoms; (5) afebrile course; (6) erythema multiforme.

Treatment.—*Isolation* and quarantine are necessary. *Pain* is the prominent indication, for which acetphenetidinum and salicylates serve fairly well, though they are much inferior to opium.

CHOLERA ASIATICA.

Definition.—A specific infection caused by Koch's comma bacillus. It is chiefly disseminated by drinking-water, is characterized clinically by vomiting, severe purging, and by such symptoms as cardiac failure and suppression of urine which are due to loss of fluids and to toxæmia.

History.—The name has been used since the time of Hippocrates, Galen and Celsus, but apparently not for Asiatic cholera. Asiatic

cholera is said to have existed before Christ, and travelers in India in the sixteenth century spoke of it. Its first European invasion was in 1817, from which time our knowledge of cholera dates. Other epidemics have occurred in 1826, 1832, 1847–1855, 1865–1867, 1873, 1881–1884, and 1892–1894. Cholera visited America in 1832–1835, 1836, 1848, 1849, 1854, 1865, 1867, and 1873. This was the last epidemic, though isolated cases have occurred since.

Bacteriology.—Koch, while studying cholera in Egypt and India, in 1883–1884, discovered the specific organism in the fæces during life and in the bowel at autopsy. The *comma bacillus* is 1 to 1.5 μ long and half as wide; it is smaller than the tubercle bacillus, but more plump and curved and is motile and flagellated (Loeffler). Gaffky (1887) first obtained pure growths on various media. In cultures its form varies, being sometimes S-shaped, and thread- or spirillum-like. The comma bacillus is found in the stools and in the intestines, usually in pure cultures. After several days it may be found in the bowel wall, though rarely in the stomach or vomitus. It disappears from the movements after six to eleven days and rarely reaches the blood or viscera.

Etiology.—In its dissemination and contagion, cholera resembles typhoid rather than the exanthemata, *i. e.*, infection is less direct than indirect. Attendance on cholera patients is not dangerous if the hands are kept clean. The germ is very susceptible to boiling, acids, antiseptics, drying, and the presence of saprophytes in sewers and water. There is little risk of aërial dissemination, though flies may carry infection. Koch early maintained that cholera was conveyed by *water contamination.* He found the cholera vibrios in a water-tank in Calcutta from which infection was clearly traced. Fränkel, von Ermingen, Biernacki, and others, found it in flowing and stagnant water. It is well known that cholera vibrios may be found in the movements without cholera symptoms, just as diphtheria bacilli may be found in healthy throats. The normal acid of the stomach protects the individual, and animals are infected experimentally only when the gastric acidity is neutralized and peristalsis is arrested by opium. Pettenkofer vigorously objected to Koch's teaching and insisted that individual and local disposition was more essential. He even swallowed pure cultures, and luckily escaped infection. Nevertheless, the terrible Hamburg epidemic (1892–1894) amounted, as Koch said, to a demonstration of almost laboratory exactness. The disease developed "explosively." On August 16 there was one case; by August 27 there were 1,000 deaths, and September 3, there were 10,000 cases and 4,300 deaths. The total mortality was over 8,300. The Hamburg drinking water came unfiltered from the Elbe River, which was then at a low level, the weather being hot. The germ was found in this supply. Altona and Wandsbeck, which are parts of Hamburg, had a supply of good water, and practically escaped. That part of Hamburg which used Altona water was exempt. The cholera germ is difficult to find in water because it is so easily overgrown by saprophytes.

Infection by ice, by milk, by green lettuce, by flies, or in the laboratory, is less common. The washing of infected linen is particularly dangerous to washerwomen. In India, hundreds of thousands of pilgrims drink the "holy water" of the Ganges, in which they also bathe and defecate. Similar infections occur in the pilgrimages to Mecca. In former years the caravans carried infection from India through Persia and Afghanistan to Russia, which was also the route of dissemination in 1892. Ships from the Orient are extremely dangerous.

Individual Predisposition.—Decrease of physiological resistance increases the risk of infection. Alcoholism, strong purgatives, dyspepsia, overexertion, poor hygienic conditions, and debilitating diseases are predisposing factors. Counting the mortality in a cholera-stricken community for a few years, it is seen that the total death-rate is not increased, which means that weakly persons succumb to cholera and that later epidemics of other diseases have a diminished prey.

Epidemics involve a smaller percentage of the people in large than in small cities, but they last for months in large cities and usually only for weeks in small towns. Epidemics end with a lessening mortality, and both their ending and the lessened mortality are probably best explained by the decreasing virulence of the germ. Warm weather tends to increase epidemics and they are usually checked by frost. Nevertheless, Russia's severest epidemic occurred in the winter.

Symptoms.—After an *incubation of two or three days,* the disease develops with varying virulence. The bacilli are found in the stools of some healthy persons. Several stages are usually described, which also correspond to clinical types of different severity.

1. Prodromal Diarrhœa.—The stools contain the comma bacillus, are thin and normal in color, are voided without pain or tenesmus, and sometimes produce only moderate depression or malaise. The trouble may cease here, when we speak of cholera diarrhœa, or it may pass into the choleraic stage.

2. Choleraic Stage.—This may usher in the disease or follow the first stage. The stools, at first yellow and voided frequently, become colorless because the bile pigment is diluted and decreased, and become thin,—the ominous "rice-water" stools. Diarrhœa usually comes on first at night. The stools may number twenty or thirty and a pint to five quarts (500 to 5,000 c.c.) are voided daily. They have a fetid, not a fecal odor, and contain 98 to 99 per cent. water. In the evacuations are seen flocculi of comma bacilli, leukocytes, epithelial cells or shreds, detritus, and a few red cells. There is often gurgling, although little gas is voided. Much fluid is thus lost to the circulation, and this causes some of the following symptoms: The abdomen is flat, usually neither tender nor painful, and fluid may be percussed in the bowels. *Vomiting* is frequent, painless, although exhausting, and thin, and profuse, even to three quarts daily; sometimes Koch's bacillus is found. The *skin* early shows the lack of

fluid; it becomes *shrunken, lax,* wrinkled, and when pinched, its folds disappear but slowly. The nose becomes small and pointed, the eyes hollowed, and the malar bones jut out prominently; the extremities, nose, and ears become cold and cyanotic, and grayish-blue rings develop beneath the sunken eyes (*facies cholerica*). If pleurisy or dropsy are present they are rapidly absorbed. The saliva and urine diminish and disappear, though nursing mothers may continue to secrete milk. The loss of fluid occasions distressing *thirst* and *muscle cramps,* especially in the calves of the legs, but also in other muscles, which are found dry at autopsy. The immense transudation into the bowel has other effects; it *concentrates the blood,* whose cells and hæmoglobin rise; the red cells may total six to eight millions and the serum has a specific gravity of 1,040 or even 1,070 (1,028 being the normal). The *heart tones* become *weak* and the pulse rises to 100, because the body cannot lose 10 per cent. of its fluid without injury. The heart has not enough blood upon which to contract and the blood is viscid; the heart also suffers reflexly, as it does in intestinal incarceration, and degenerates from absorption of toxins (soluble substances from the bacilli). The difficulty in oxygenation increases the respiration to thirty or forty, and occasions precordial and epigastric anxiety. The *vox cholerica* is due to the dry larynx and weak muscles. The reflexes are decreased or absent, the pupils tardy; the sensorium is normal, although exhaustion, apathy, and vertigo are usual. The urine contains albumin and casts which are largely hyaline. While the external parts are colder by 5° to 10° or more, the patient feels hot, and the internal temperature may be 102° or higher. The loss of weight is great (even 0.5 to 1 per cent. hourly!). In some few cases the patient dies before diarrhœa develops (*cholera sicca*). Recovery is possible, even with these ominous symptoms; reaction may set in, the diarrhœa and algidity may lessen, the heart may improve, and erythematous, roseolous, urticarial and other rashes may develop. Indican, which was previously absent from the urine, may reappear; the urea is increased two- or threefold and the ethereal sulphates are increased. The symptoms are then chiefly toxic, and probably result from absorption of the cholera and other toxins from the intestine (the so-called *cholera typhoid,—i. e.,* cholera with a pronounced typhoid state).

3. Stage of Collapse.—This stage is characterized by asphyxia (literally meaning without pulse), paralysis or algidity. The heart tones are inaudible, the radial pulse imperceptible, the arteries empty, and the veins full. In the days of venesection the blood would not drain from the incised veins, and at autopsy little or no blood escapes from the vessels. The picture is that of collapse. The skin is lead-colored, cyanotic, even violet or purple, and will not vesicate after blistering. There is dyspnœa, the expired air is cool, the voice gone, the mouth dry, the conjunctivæ dry or opaque, and the lids are parted, yet the mind is clear. The evacuations cease. Pericardial or pleural rubs may be heard, due to the great viscidity found in all the serosæ

at necropsy. Very few cases with the symptoms of cholera-typhoid react and recover, but most of them die within a few hours, or a day or two. After death the muscles frequently contract, to the great terror of observers. The face is distorted, the fingers flexed, and the legs drawn up. Post-agonal rise in temperature may occur. At autopsy the small intestines are injected, filled with rice-water contents, their epithelium or villi are desquamated or necrosed, the lymphatic structures are sometimes swollen, reddened and reticulated. Comma bacilli are found in the stools and intestinal wall, and occasionally there is an agonal invagination of the gut. The muscles are dry, the spleen small, and the kidneys show a pale cortex with great cortical degeneration, especially in the convoluted tubules; the renal changes, once attributed to loss of water and disturbed circulation, are probably toxæmic.

Complications.—The so-called croupous or diphtheritic enteritis is due to the pyogenic cocci. Diphtheroid changes may also be found in the uterus, vagina and throat. Hemorrhages from mucous or serous surfaces, though usually small, may occur. Pneumonia (20 per cent. of fatal cases), pleurisy or lung infarct; skin, parotid or other suppurations; icterus, bronchitis, venous thrombosis or post-febrile psychoses, are uncommon. Griesinger's "cholera-typhoid" is the most frequent sequel (25 per cent. of cases); nervous complications and uræmia are far less common causes than the toxins absorbed late from the intestine and operating intensely on the already exhausted, slightly degenerated, and anæmic brain.

Diagnosis.—Diagnosis is usually easy in the presence of an epidemic. The first cases occasion the most difficulty, especially if they are light. As already stated, robust individuals may not react to the germs found in their stools; simple diarrhœa may result in other cases, and only a mild depression may mark some cases known as *cholerine*. On the other hand, aged, weakly, or tuberculous persons may succumb to cholera before its characteristic stages develop. In clinically suspicious cases, laboratory measures are necessary for diagnosis. The pseudocholera vibrios in the Elbe, Seine, or Danube Canal (Vienna) are differentiated with difficulty. The comma bacillus is obtained (*a*) in nearly *pure cultures* from the stools, especially from their shreds; (*b*) in peptone cultures (1 per cent., with 1 per cent. salt) in which the germs are found on the surface in six hours, because they rise to obtain oxygen; (*c*) in 10 per cent. gelatin plates, in which they develop in fifteen to twenty hours; (*d*) the nitrosoindol (cholera-red) reaction results on adding sulphuric or hydrochloric acid. It was thought that no other bacterium produced both nitrates and indol, but the pseudocomma organisms may produce the same reaction; (*e*) in animals, in which the cholera germ produces a sudden fall of temperature, but this is uncertain; (*f*) *Pfeiffer's test* is the sole absolute criterion, according to which the blood of animals immunized to cholera produces agglutination of the true cholera bacillus.

Cholera nostras produces a similar picture. It is usually less severe. In some few cases the Finckler-Prior bacillus is found, but in the majority of cases other microbes are present, such as the colon bacillus, and less often the streptococcus, staphylococcus, and a bacillus like the Bacillus subtilis; vibrios, proteus and pyocyaneus forms; an encapsulated bacillus; Gärtner's Bacillus enteritidis; in meat, anaërobic forms; and in mollusks, Zieber's Bacillus piscicidus agilis. It is possible that many of our domestic intestinal organisms may become virulent. Liebermeister said cholera nostras bears the same relation to cholera Asiatica as varicella does to variola, German measles to measles, or epidemic icterus to yellow fever. It occurs at the same season. Its symptoms differ chiefly in degree, and occasionally, a large percentage (even 40) of deaths occur, chiefly in the extremes of life.

Arsenical poisoning, peritonitis, and intussusception without bacteriological confirmation may closely resemble cholera.

Prognosis.—The prognosis is always grave. Cholera ranks next after the plague in death-rate, and averages over 50 to 60 per cent., and sometimes 80 per cent.; 100,000 to 700,000 die yearly in India. In 1883, 28,000 died in Egypt, 8,605 died in Hamburg, and 800,000 in Russia (1892–1894). About 45 per cent. of the deaths in Hamburg occurred in the first twenty-four hours and 17 per cent. in forty-eight hours, whence the prognosis is more favorable after the second day; 85 per cent. die when the stage of collapse is reached. Light cases may become severe or the worst cases may recover. Unfavorable factors are youth and old age; pregnancy; colorless stools, early frequent movements, early vomiting, anuria and low peripheral temperature.

Treatment.—1. Prophylaxis.—Quarantine of ships, isolation of early cases, thorough disinfection of the stools, soiled linen, etc., and special attention to light cases and to any suspicious diarrhœa, are most important. In convalescence the stools may remain infective for seven weeks (Kolle). Despite the sudden outbreaks in Germany in 1892–1894, sanitary measures kept the total of deaths at 10,000, while in Russia 800,000 died in the same period. Individual prophylaxis concerns the maintenance of physiological resistance; moderation in eating and drinking; boiling of water for drinking and washing; boiling of milk; care in regard to ice; avoidance of green fruits or vegetables, and contamination of food by flies. In one series of cases the use of the prophylactic serum of Ferrán and Haffkine reduced the morbidity two-thirds and the mortality one-half.

2. Diarrhœa.—This should be treated by absolute rest in bed, and by initial cleansing of the bowels with calomel and castor oil; thin gruels should be given as a diet; red wine and aromatics, tea, and full doses of bismuth and opium should be given. Intestinal antiseptics are inert. The diarrhœa, except the early movements, is not easily stopped, because absorption is arrested. Cantani advised that 1 or 2 quarts of 1 per cent. tannic acid solution be given slowly by rectum

in the hope that it would reach the small intestine, and Griesinger advised that larger quantities be given, and that expulsion by the rectum be forcibly prevented. The fluid was given until it was vomited, apparently with some benefit.

3. Vomiting.—Vomiting is intractable. Fluids should not be withheld. The stomach may be washed with normal salt solution. Hot wine, champagne, and hypodermics of morphia are also indicated.

4. Heart.—Failing heart action and renal secretion are sometimes helped by a warm bath. Hypodermatoclysis was recommended by Cantani (1865) and intravenous transfusions were employed by Hermann and Latta (1830). Their use was studied during the Hamburg epidemic and their employment often increased the heart's force and benefited the general condition greatly, at least for a time. The results are somewhat better than the mortality (65 to 77 per cent.) would indicate, for they were used in the stage of profound collapse (where the death-rate is as high as 85 per cent.). If used earlier the results might have been better. The kidney secretion is apparently little influenced by this treatment.

5. Muscles.—Muscular cramps are helped by warm baths, sinapisms, friction, local heat, saline infusions, and morphine, which should be given hypodermatically.

6. Convalescence.—In convalescence, great care is necessary lest the diarrhœa return.

Other symptoms, complications and sequels are treated expectantly.

YELLOW FEVER.

Definition.—A specific infection of unknown etiology, which is transmitted by the mosquito, is endemic in the American tropics and subtropics, and is characterized pathologically by moderate degeneration, especially of the liver and kidneys, and clinically by a rapidly developing fever, by icterus, albuminuria, adynamia, hæmatemesis, and by other hemorrhages.

Etiology.—The cause is unknown, although two microörganisms have been especially described: (1) Sanarelli's Bacillus icteroides, which is said to be present in the blood, and viscera, but not in the intestine; it is a facultative anaërobe, with rounded ends, 2 to 4 μ long and half as wide; positive agglutination tests are said to have been made. Sanarelli's organism is generally considered as a contamination. (2) Sternberg's bacillus, resembling the influenza germ. While the cause is not known, it is certain that the disease is transmitted by the mosquito (*Stegomyia fasciata*). This was first suspected by Carlos Finlay, in 1881, and the mosquito was demonstrated to be the intermediate host by the investigating board composed of Walter Reed, James Carroll, A. Argamonte and J. W. Lazear. Dr. Carroll recovered from a severe attack, but Dr. Lazear died from inoculation. Dr. Myers, of the English Commission, also succumbed. The Stegomyia breeds not only in stagnant street pools, but also in

cesspools and sewers, whence its special danger in unhygienic tropical towns. Voluntary inoculations by mosquitos, which twelve days previously had bitten yellow-fever patients in the first three days, produced yellow fever in non-immunes. Finlay failed to prove his correct suspicion because he allowed the mosquitos only three to six days in which to develop the germs which they had absorbed, while twelve days are necessary. The mosquito carries infection only when it bites the patient in the first three days of the disease. As in malaria, the tropical foci, the hot summer and autumn seasons, the invasion of low altitudes, river courses and coast towns, humidity, and exposure to night air, are only conditions which are suitable to the mosquito. Frost stops infection because it kills the mosquito. Direct contagion does not occur. Nurses and physicians are not infected from contact with yellow-fever patients. Persons going from the City of Mexico to Vera Cruz may acquire the disease, but on coming home with it they do not cause its dissemination, because the mosquito is not present. Postmortem wounds and direct voluntary inoculations, however, have directly communicated the disease. The yellow-fever virus is the only virus, infecting man, which passes through fine filters. Reed's committee experimentally exploded the theory that fomites, clothing, etc., were infective. Volunteers remained in rooms occupied by yellow-fever patients and slept in their soiled linen without contracting the disease.

Predisposition.—The lighter races are more prone to infection. It is thought that immunes are merely those who have had light and unrecognized infections. Most cases occur in males between twenty and forty years of age. Yellow fever is kept alive in endemic foci by mild cases, especially among the Creole children.

Foci and Epidemiology.—The two prominent homes of yellow fever are (1) Mexico, the Antilles, Mexican Gulf and Caribbean Sea; and (2) Africa, from Senegal, through Sierra Leone, to the Gold Coast. The first clear description of yellow fever, according to Sodré and Couto, was in 1686, but certainly yellow fever was known before the discovery of America. It reached this country possibly in the sixteenth century and has extended as far north on the coast as Boston and on the Mississippi River as far as St. Louis. It has reached Quebec and Chicago. In Philadelphia (1793, 1797–1798), 4,041 died in a year; in New York, 2,080; and in New Orleans, in 1853, 8,000 died. Epidemics occurred also in 1802, 1805, 1853, 1867, 1873, 1878, and to a slight degree in 1897–1899; New Orleans was visited severely in 1905; there were about 9,000 cases and 1,000 deaths. Europe is seldom, but South America is frequently invaded.

General Clinical Description.—Incubation.—The incubation averages three or four days. Prodromal symptoms last less than a day; they are malaise, fatigue, headache, dyspepsia, colic, or other indistinctive symptoms. The disease is often divided into stages, not all of which are always present:

Stage I.—The *stage of congestion* begins (*a*) with some headache, frontal, supraorbital or retroöcular, the earliest and most prominent symptom; (*b*) sometimes with a chill; (*c*) with flushed face; the conjunctivæ are injected and sometimes icteric even at this stage; the eyes are sensitive to light, the lids somewhat puffed and the expression is a mixture of pain, restlessness and confusion, comparable to the alcoholic facies; (*d*) with backache, and pains radiating into the calves of the legs; (*e*) with fever rising to 104° or more; and (*f*) with a full, tense pulse of 100 to 110.

The tongue is usually clean or slightly coated dorsally, and its tip and edges are red. The urine is febrile and very early albuminous. The skin is intensely red and dry. The epigastrium is tender early in the disease and there is marked gastric irritability and intolerance of food, water and medicine. This stage averages two days.

Stage II.—The *remission stage,* or deceptive stage, occurs in some cases and is so-called because of the subsequent severe symptoms. The fever, pulse and the pain remit. Slight jaundice may appear.

Stage III.—*The Reaction Stage.*—Slight temperature, the "black vomit" (*vomito negro*) of blood, hemorrhage into the nose, intestines, uterus or skin, hiccough, epigastric pain and tenderness, liver enlargement, and icterus mark this period. The urine becomes scant and laden with bile pigment, or its secretion is suppressed; the pulse becomes weaker, and insomnia, prostration and restlessness may appear.

Various *issues* are possible: (*a*) Gradual amelioration; (*b*) fatal anuria; (*c*) profuse hemorrhages, which end in collapse, and rarely in recovery; (*d*) profound icterus, which results in adynamic phenomena and usually in death, and (*e*) death, from cardiac insufficiency.

Special forms of yellow fever are described by Soudé and Couto:—(*A*). *Variations in intensity* include (1) light, abortive, ambulatory, atypical forms, especially in children; (2) the usual severe forms, and (3) the foudroyant type, which is rapidly lethal in one to two days. (*B*). *Visceral forms:* (1) The hepatic type, with two weeks of the status typhosus, often with normal temperature and sometimes with hemorrhages; (2) the uræmic type, often with early death; (3) the hemorrhagic; (4) the cardiac; and (5) nervous or atactic type.

Special Symptomatology.—1. Fever.—The fever is neither cyclic nor diagnostic. Sternberg describes a single febrile paroxysm of two to seven days. It is usually highest in the first stage, and reaches an average of 104°. It may fall on the second or third day to nearly normal in benign cases or in the ominous uræmic type. High fever on or after the second day is a poor prognostic, and is sometimes due to secondary infection with the pyogenic cocci, or the typhoid or colon bacillus. In the second stage fever is usually between 99° and 100°, and may rise in the third stage. A high postmortem rise is frequent. In convalescence, the temperature is subnormal.

2. Nervous Symptoms.—Nervous symptoms are often absent in both benign and malignant cases. Insomnia and headache are common in the congestive period. Delirium is infrequent, and in 80 per cent. of the cases, is uræmic rather than purely toxæmic. Atactic symptoms are most common in children, alcoholics and in the debilitated. In children, the status typhosus or meningo-encephalitic symptoms (convulsions, unequal pupils, Cheyne-Stokes' breathing, and coma) are possible. Pathologically, few changes are found except the slight degenerations common to the febrile state, punctate hemorrhage (mostly agonal), or bile-tinging.

3. Circulatory Symptoms.—(*a*) The pulse in the congestive stage is usually 100 to 130 and is tense from increased blood pressure; on the third day, 90 to 100 (even 70 to 80); in the second stage it is still lower in benign and more rapid in severe cases (110 to 140), is small, and the manometer shows lowered arterial tension; in convalescence it is usually slow (50 to 60, or even 30). Faget, of New Orleans, considers the slowing of the pulse with maintenance of the fever, almost pathognomonic. (*b*) In severe forms the first tone of the heart becomes weak and is finally lost. The same is true of the second tone. The heart action is rarely irregular or intermittent. Embryocardia is found in severe cardiac types. In the second or third stage, cardiac dilatation is common (in 70 per cent. of autopsies). Cardiac insufficiency is due to myocardial degeneration; pericardial and endocardial ecchymoses are frequent. Cardiac action is exaggerated when uræmia is present. (*c*) Often there is no anæmia. The white cells are decreased in proportion to the severity of infection. The polynuclears are relatively increased.

4. Respiratory Symptoms.—Respiratory symptoms may be due to intercurrent, or more often to cardiac or renal complications.

5. Digestive Tract.—Severe hiccough is attributed to irritation of the phrenic nerve. Epigastric pain, tenderness and anxiety may be symptomatic of cardiac distress, premonitory of the black vomit, or indicative of disturbed circulation in the medulla.

6. Icterus (Yellow Fever, Typhus Icteroides).—Jaundice usually develops on the third to the fifth day. It is frequently absent in children, or in cases of early death from uræmia. It is absent in 50 per cent. of cases which recover. The explanation of the icterus is disputed:—Féraud distinguishes (*a*) the early, lighter jaundice, due to blood pigment, and (*b*) the later and more severe type due to bile pigments.

7. Liver.—The liver is slightly enlarged and is tender. Pathologically, its color is light, and has a cooked appearance. The left lobe may reach the spleen. On section, it is oily, fatty, and anæmic.

8. Urine.—The urine is early febrile. *Albuminuria* occurs in 88 per cent. of cases in the first three days. Leucin, casts and the bile pigments are common. Indicanuria is very infrequent. The kidneys, anatomically are normal in size, rather pale or yellowish, and, microscopically, show great fatty degeneration of the tubules,

which in severe cases are actually choked by desquamated epithelium and casts. The glomeruli show remarkably few changes. *Anuria* is due to these changes and is fatal in 97 per cent. of cases. *Uræmia* causes 33 per cent. of the fatalities; it is marked by delirium, nervousness, twitching or convulsions, decreased or suppressed urine, by rapid, forced, irregular, labored or Cheyne-Stokes' breathing, and by a tense pulse. Death in coma may occur on the third day. Uræmia is rare in children.

9. Hemorrhages.—They are most frequent in the alimentary tract, nose, and uterus. They result from fatty degeneration, or actual rupture of the bloodvessels. (*a*) *Alimentary tract.* Hemorrhages may occur from the mouth, tongue, lips, stomach or intestine. The *black vomit* is gradually transuded blood altered by the gastric acid; it most often occurs on the fourth to the sixth day; it is the ordinary "coffee grounds" vomit or is ink-like; the stomach, at autopsy, shows the black contents, congestion, or even dark discoloration, epithelial desquamation and fatty change in the muscularis. Hemorrhage from the small intestine is equally frequent. (*b*) *Epistaxis* is often profuse, and sometimes fatal. (*c*) *Uterine hemorrhages* are severe, especially at the menses, and in pregnant women cause abortion or premature delivery. Less frequent are (*d*) *skin petechiæ,* (*e*) *muscular hemorrhages* and (*f*) hemorrhage in other mucosæ, serosæ or viscera.

Complications and Sequels.—These include (1) secondary infections such as endocarditis, pericarditis, pneumonia, erysipelas, parotitis, arthritis, suppuration, and gangrene. (2) Coincident typhoid, malaria, influenza, and insolation. (3) Post-febrile psychoses.

Diagnosis.—The diagnosis is usually easy from (1) the flushed, semi-intoxicated facies; (2) icterus; (3) the almost invariable and early albuminuria—the most valuable finding in doubtful cases; (4) black vomit, gastric intolerance, and marked epigastric tenderness; (5) hemorrhages, and (6) the slow pulse, which is absent in some severe, but therefore clear, cases. *Dengue* lacks these features. Pernicious *malaria,* with icterus, is distinguished by the blood findings, enlarged spleen (always absent in yellow fever), the infrequency of early icterus, albuminuria and hemorrhages (except hæmaturia). This form of malaria is rare in the cities, whence the constant suggestion of yellow fever, when more than sporadic cases are reported. *Weil's disease* (infectious jaundice), and acute yellow atrophy of the liver are usually readily distinguished.

Prognosis.—The mortality of epidemics varies from 10 to 85 per cent. The average death-rate is about 30 per cent. The mean death-rate, according to *age,* is 33 per cent. (one to three years), 14 per cent. (four to eighteen years), and 37 per cent. (nineteen to forty years). Unfavorable events are hepatic, renal, or cardiac insufficiency, the typhoid state, diffuse hemorrhages, combination of special types (cardiac plus renal, or hepatic plus renal), high fever with hepatic symptoms, and hemorrhages.

Treatment.—1. PROPHYLAXIS.—Disinfection of the fomites is now less important than the question of the mosquito. Probably no greater triumph of hygiene exists than the stamping out of yellow fever since the American occupation of Havana. It illustrates not only the efficiency of the investigating scientific commission, but also the possibilities of medicine in administrative capacity with unlimited discretionary power. In Havana the average death-rate was 500 yearly, until Gorgas cut it down to *nil.* The patient should be isolated, not to prevent direct personal contagion but to keep mosquitos from the infected person. Healthy individuals should be kept from infection by the mosquito by the same measures that are employed in malaria, *e. g.,* screens, and extermination of breeding foci. The Stegomyia is found outside the habitual yellow-fever foci, and after biting patients from abroad, may inoculate other persons. Infected insects, too, may be carried by ships.

2. SYMPTOMATIC TREATMENT.—(*a*) In the *first stage,* fluidextractum aconiti, ♏ iij, and sodium salicylat., gr. xx, may be given every two to four hours for a few doses, until the congestion and pains are relieved. Coal-tar products tend to depress. Fractional doses of calomel, gr. $\frac{1}{4}$ every hour for five doses, should be given and followed by two ounces of castor oil. Overmedication is particularly to be avoided because of the gastric irritability, and feeding by rectum is often indicated. Early or violent cerebral, uræmic, or pulmonary symptoms are sometimes treated by phlebotomy. (*b*) In the *last stage,* the black vomit contraindicates any fluid, food, or medication by mouth; warm peptonized milk and normal salt solution should be given by rectum; morphine by mouth is dangerous. (*c*) In *cardiac insufficiency,* digitalis is especially good, and is beneficial even when cardiac failure is absent; saline solution, by rectum or subcutaneously, is better than intravenous transfusions. The patient must be kept absolutely quiet. (*d*) *Uræmic* symptoms are treated in much the same way. Much water should be given by the bowel. (*e*) *Nervous* manifestations necessitate cool spongings, bromides, morphine and hyoscine, as in Typhoid. (*f*) Vomiting is relieved most efficaciously by chopped ice (see TYPHOID).

THE PLAGUE.

Definition.—The pest, or bubonic plague, is a specific acute infection caused by the Bacillus pestis and characterized by a pronounced typhoid state, fever, buboes and carbuncles, very frequently by septicæmia (pesticæmia), and by extremely high mortality.

History.—The first clear accounts date from the times of Trajan, in the second century, and of Justinian, in the year 542. The plague was important among the pestilences of the middle ages. In the fourteenth century the "black death" swept over Europe with a mortality of 25,000,000 (one-quarter of the world's population). It reappeared in the sixteenth and seventeenth centuries, but in the

eighteenth century it withdrew southeast, and disappeared from Europe until the small Russian epidemic in 1878–1879. Egypt was the early focus for African epidemics, and European epidemics started from Turkey. Syria was the leading Asiatic focus for thirteen epidemics (1773 and 1843). The present Oriental focus is Thibet, whence India and China have been invaded thirty times between 1823 and 1897. Uganda is considered a permanent plague home by Koch; plague is endemic in Asia near Mecca, in Mesopotamia and in Siberia. In 1899–1900, New York, San Francisco, Cape Town, South America and Australia were visited. Epidemics appear, reach their maximum, and decline at nearly the same time each year.

Etiology.—The *Bacillus pestis* was found by Yersin, in 1894, and at the same time Kitasato found a possibly identical bacillus. Its ends are rounded and stain deeply; its sides are somewhat convex; it measures $1\frac{1}{2}$ to $1\frac{3}{4}$ by $\frac{1}{2}$ to $\frac{3}{4}$ μ and is encapsulated. It is polymorphous, and appears as rods or diplo-bacilli. Atypical forms are oval, ovoid, annular, chain-, thread-, or branch-like; degenerative or involution forms are the pyriform, biscuit, comma-shaped, or spermatozoid. It is most often outside of the cells. It contains no spores or flagella. It is found in all organs, in the sputum, the urine and fæces, and in the bubo and carbuncles. Its cultures are typical, and superficial, and have bulging, wall-like edges. It stains with ordinary stains, but not by Gram's method. It is readily killed by sunlight, desiccation, antiseptics, and is often overgrown by secondary infection. It lives three to four weeks in buried bodies, but persists longer (*v. i.*) in excreta of convalescents.

Atrium.—It may enter (1) by the *skin,* through invisible wounds, scratches, or the intact integument (Albrecht and Ghon). Post-mortem and laboratory inoculation is possible, as in the unfortunate case of Müller, of Vienna, who was writing his well known monograph. Flies, *fleas,* ants, bed-bugs, etc., may inoculate the disease. (2) The *mucosæ,* conjunctivæ, nose, tonsils, anus and external genitalia, may be primarily infected. The *nose and throat* are especially frequent atria in children, for obvious reasons. (3) The alimentary tract affords a possible atrium. (4) The bacillus may cause primary plague pneumonia.

Contagion may be direct or indirect. (*a*) *Direct.*—Dissemination of plague pneumonia by the sputum is most dangerous, for the bacilli are scattered by coughing, as shown in Flügge's experiments in tuberculosis (*q. v.*). The pest spreads rapidly only when it is of the pneumonic type. The bacilli may penetrate from unruptured carbuncles or buboes or scatter from them after their rupture. The urine, the fæces, and the milk of nursing women are also dangerous. Direct contagion may result from autopsies, from operations, or from nasal or urinary hemorrhages. (*b*) *Indirect.*—This may result from fomites, or bites of animals or insects containing the germ. Epidemics among rats precede human epidemics. In Canton, 35,-

000 dead rats were found in one district. The disease also exists among apes, cats, mice, and ground-squirrels. Infection by rat-fleas is clearly proven. Flies may convey infection from the urine or fæces of infected rats. The bacillus produces *virulent toxins.*

Symptoms.—There are two factors in the clinical picture: (*a*) The local changes, as those in the primary localization (bubo or pneumonia), and (*b*) the successive septicæmia (pesticæmia) and toxæmia. The average *incubation is five days.* Occasionally there is prodromal depression, or glandular pain, but usually the *onset is very acute,* with marked headache, intense vertigo, severe rigor, sudden fever, and, in children, convulsions. Early vomiting is quite frequent. There is *early glandular pain,* the prostration is profound, and an early typhoid state develops. The *fever* reaches 104° on the second day, is either continuous or remittent, in convalescing cases falls by lysis (fifth to seventh day), or may remain high until death. Crisis is seen in light cases only. The *skin* is *hot* and dry or may be covered with sweat as death approaches. Herpes never develops. Skin *hemorrhages* are frequent, due to embolism by the Bacillus pestis; they occur shortly before death, and are seen mostly in the upper trunk, arms, head and neck. Abscesses, gangrene or bedsores, are not common.

1. Characteristic Local Finding.—This is the *bubo, for the lymph glands are more important than in any other infection.* The *several clinical forms* of the plague usually described are (*a*) the *bubonic* type (77.7 per cent.); (*b*) the *septicæmic* type (14 per cent.); (*c*) the *pulmonary* type (4.3 per cent.); (*d*) the carbuncle type (2.5 per cent.); (*e*) mixed infection (1 per cent.), and (*f*) the ambulatory type (pestis minor, 0.5 per cent.), which is considered by Scheube as not plague but "climatic bubo." The figures are from Choksey, of Bombay. In probably all except the pulmonary type, *the bubo is found* very early (90 to 98 per cent.) and local pain, tenderness and swelling are present. The primary bubo occurs in the external lymph glands in the region of the atrium; it is large, very painful and tender, and is attended by great periadenitis, œdema, hemorrhage and vesicle formation in the skin. Its size and rapid growth are due in part to medullary swelling of the lymph glands, but chiefly to hemorrhage, which may spread along the nerves, arteries or veins, into which it may break, thus disseminating the bacilli. The glands may fuse, and a brawny mass, as large as an egg, or a man's fist, results. The glands and surrounding tissues contain enormous numbers of bacilli. The location is inguinal (70 per cent.), axillary (20 per cent.), and cervical (10 per cent.). The inguinal and axillary adenitis is more common in males; cervical adenitis results in children from infection of nose and throat by dirty fingers; the popliteal and epitrochlear glands, curiously, are involved in but 1 or 2 per cent. Strangely, too, the lymph-vessels are usually unaffected, although they are sometimes inflamed around the buboes, carbuncles, or atrium of infection. From the primary bubo the con-

tiguous glands are involved; *e. g.*, after the infection of the inguinal glands, the iliac, aortic, mediastinal, subpectoral, infra- and supraclavicular or cervical glands may be involved; *i. e.*, infection usually occurs in the direction of the lymph-current. On the fourth to the sixth day necrosis occurs, and on the eighth or ninth day suppuration develops, resulting more often (66 per cent.) from the Bacillus pestis alone than from mixed infection (33 per cent.). Some glands may resolve without suppuration. The primary bubo is often inconspicuous and its size has no bearing on the intensity of the disease, although cases with marked periadenitis rarely recover. Besides the primary bubo, Albrecht and Ghon described the *secondary bubo* which is infected by the bacilli-laden blood current. The bacilli have a remarkable affinity for lymphatic structures and settle in them largely. These *hæmatogenous buboes* are generally smaller and less extensive, tender, painful, œdematous, and hemorrhagic, than the primary bubo. They contain bacilli in large numbers, as do the mesenteric glands, the pharyngeal follicles, stomach, intestine, and lung. *Carbuncles* result chiefly from pesticæmia, although apparently in some cases they are the atrium. They occur in 10 to 15 per cent. of the cases, often near the primary bubo. The crowning serous vesicle is ruptured, discharges bacilli, and leaves an ulcer, whose floor is bluish-red from hemorrhage, or yellow from swarms of bacilli. A dark crust then forms. There is great peripheral infiltration, and the whole limb may become œdematous. These lesions indicate that the pesticæmic type is less a distinct type than an almost regular part of the disease. Cases rarely recover which show bacillæmia. The blood shows some anæmia and moderate polymorphonuclear leukocytosis. The spleen is increased three to four times, is reddish, and is somewhat less soft than in other infections. Its cut surface is finely granular and is slightly glistening. It is infiltrated with white cells and bacilli, which are found in the innumerable foci of minute necrosis. It is usually palpable on the first or second day.

2. Nervous System.—The nervous system suffers few anatomical changes except meningeal hemorrhages and brain œdema. Clinically the toxæmic "typhoid" symptoms are prominent. There is early headache and marked prostration. Vertigo is especially notable, causing a drunken, cerebellar gait, and vomiting. The delirium is more often dull, dreamy and docile, than active or maniacal. Twitching, spasms, rigidity of the neck and even bulbar symptoms, such as increased pulse and respiration, polyphagia or polydipsia, may suggest meningitis. Paralyses are uncommon. Ear symptoms are unusual. Conjunctival hyperæmia, sometimes with ecchymoses, is so frequent that it is, together with the fever and buboes, made one of the cardinal symptoms by Yamagiwa.

3. Circulation.—The circulatory and the nervous changes measure the intensity of infection and forecast the prognosis. Possibly there is no other cardiac poison as intense as the plague toxin which

operates on the medulla and its vagus nucleus. Early in the attack the pulse is full and dicrotic, with a rate of 80 to 90. Later, the pulse increases to 120, 150, or even 200; the heart tones become muffled; tachypnœa, great dyspnœa, and all the signs of acute cardiac insufficiency develop. In some virulent infections the pulse may remain slow. At autopsy the heart is lax, the right ventricle dilated, the myocardium cloudy, and the serous investment ecchymotic. Endocarditis and pericarditis almost never occur.

4. Respiratory Tract.—The lymph structures in the larynx, trachea, or bronchi are infected by aspiration from tonsillar foci or through the blood. Most important is the *plague pneumonia,* of which there are two varieties, (*a*) the *primary,* in which the lung is the atrium (or analogue of the primary bubo). It was clearly the "black death" of the middle ages (Forbes). It was seen in India, in 1838, and became well known in the Indo-Chinese epidemic of 1896–1897, when Childe first found the Bacillus pestis in the lung. The bacillus, if inhaled, causes typical confluent lobar pneumonia and salient lymphatic involvement. The bacilli exist in hordes in the lung and sputum, and may cause hæmatogenous buboes and carbuncles. The diagnosis is positive only from the sputum, cyanosis, dyspnœa, hæmoptysis, pleuritic pains, signs of consolidation, adynamia, splenic tumor, and collapse. Death occurs in 97 per cent. of cases. (*b*) *Secondary* pneumonia is (i) embolic, as multiple small foci, difficult to diagnosticate, though bacilli in the sputum, dyspnœa, and pleuritic pain are suggestive. (ii) Aspiration pneumonia usually occurs in the lower lobes, its diagnosis is difficult, it follows bubonic disease of the tonsil, pharynx or bronchi, and is almost invariably fatal.

5. Digestive Tract.—A moist, then dry, tongue, hemorrhage from the mouth, and involvement of the tonsils or pharynx, are common. There may be ecchymoses and erosions in the stomach and intestines. Bubonic changes in the lymph structures, generally considered rare, were found by Wilm in 20 per cent. of his cases, and mesenteric adenitis in 60 per cent. Vomiting is frequent, meteorism and diarrhœa occasional. The liver is swollen from degeneration or stasis.

6. The Genito-urinary Tract.—The urine is decreased, and moderate albuminuria is very common. Nephritis is quite frequent. Anatomically, degeneration, minute areas of focal necrosis, and glomerular hemorrhage from capillary embolism of the bacilli, are common. Bacilluria occurs in about 30 per cent. of the cases. The bacilli have been found in the aborted fœtus. Genital complications, except uterine bleeding, are rare.

Diagnosis.—The *cardinal signs* are fever, buboes, pesticæmia, and the results of examinations by cultures, agglutination, and inoculation. In the blood, the cultural results are reliable. The agglutination reaction is not well marked until the second week of infection, but is best developed in convalescence. A positive test is final, though negative results do not exclude plague.

Prognosis.—A patient, stricken in the morning, may die before night. The average duration of the disease is about a week. If the patient outlives this time, the chances of recovery are good. Recurrences are not frequent, and the immunity conferred is only relative. Ominous symptoms are marked nervous and cardiac toxæmia, digestive disorder, hemorrhages, cervical buboes, tonsillar involvement, and pneumonia. The death-rate is often 80 and sometimes 90 or 95 per cent. It is highest between the fiftieth and sixtieth years. In Toulon (1721), 77 per cent. of the population had the plague and 62 per cent. died—higher percentages than in any other epidemic disease. 430,500 died in Bombay, from 1896 to 1899.

Treatment.—1. Prophylaxis.—The plague patient should be isolated and carefully covered with netting. Infected houses should be isolated, unroofed to admit the sun, fumigated to kill insects and rats, and the walls should be washed with antiseptics. Vigorous attacks should be made on rats. Ships should be held in quarantine until the rats in their holds are killed, the bilge-water disinfected, and all clothing fumigated. The sputum may be infective for three months and the urine for one and a half months. Carbuncles and buboes should be washed with bichloride of mercury and dressed antiseptically. Prophylactic inoculation with dead cultures and with serum from immunized animals and from convalescent patients, is a measure assured of success (Haffkine, Yersin, Roux and Lustig).

2. Symptomatic Treatment.—Buboes and carbuncles are most efficaciously treated with weak sublimate dressings. Surgical extirpation disseminates infection, and injection, into the buboes, of carbolic acid, does not stay the septicæmia. Digitalis, alcohol, and hydrotherapy are employed as in other toxæmias. Bichloride of mercury is well tolerated and was suggested by the observation that syphilitics under mercurial treatment withstood infection.

DYSENTERY.

Definition.—An acute contagious disease, (*a*) with varying etiology, but caused chiefly by the Amœba dysenteriæ and Shiga's Bacillus dysenteriæ; (*b*) attended anatomically by bowel inflammation (catarrhal, necrotic, or ulcerative) and (*c*) characterized clinically by colic, pain, tenesmus, and frequent muco-bloody passages.

History.—The name dysentery dates from Hippocrates (480 B. C.), and the affection is clearly mentioned in the oldest medical script, the *Papyrus Ebers* (1550 B. C.), and in the *Veda*. It has been the scourge of armies from the times of the Greco-Persian wars. The most severe and wide-spread epidemic occurred in 1847–1856. In our civil war there were 287,522 cases among the Northern troops. Among the English soldiers in India 30 per cent. of the deaths were due to dysentery. It prevailed in the recent South African and Spanish-American wars, but was practically absent among the Jap-

anese forces (1904–1905). Though essentially a disease of the tropics and subtropics, it is frequently observed in temperate zones, especially in prisons and in asylums. Lack of hygiene, overcrowding, eating of spoiled food or fruits, impure water, and alcoholic or other excesses, are predisposing causes. For several decades the disease has decreased in Europe and America.

Classification.—A classification and description of dysentery is most difficult, because our knowledge of all its causes is as yet imperfect. Two well known forms will be described first, and various indeterminate allied forms afterward.

Shiga's Bacillary Form of Dysentery.—In 1897 Shiga, of Japan, described the *Bacillus dysenteriæ* as the sole cause of the Japanese form. Shiga demonstrated that: (*a*) It is always found; (*b*) it occurs in no other affection; (*c*) its numbers correspond to the intensity of infection; (*d*) it is found in the intestinal wall; (*e*) the bacillus and its toxins, when inoculated, produce intestinal hemorrhage; (*f*) there is a specific agglutination, increasing in intensity under observation and most marked in convalescents; (*g*) the serum of the immunized cases is of prophylactic and therapeutic value; (*h*) actual inoculations of man produce dysentery, as in Strong's inoculation of a criminal under sentence of death, and in Flexner's assistant, as well as in accidental laboratory infections. The *bacillus* resembles the colon and typhoid bacilli in form. It usually occurs singly. It is less motile than the Bacillus typhosus but is flagellated; it is a facultative anaërobe, contains no spores, stains readily but not by Gram's method, and grows on all media. In gelatin it grows with deep, pin-point cultures, is brownish-yellow without liquefaction, and has well marked borders; it develops best in glucose agar (grayish growth along the puncture, without fermentation), or in litmus milk (some slight acidification, followed by alkalinization and a brownish color). Inoculations by mouth succeed, even without first disordering digestion. Inoculation intraperitoneally or subcutaneously is usually fatal, but sometimes causes colitis. Almost pure cultures are obtained from the movements. The bacilli are most abundant when the lower intestine is greatly involved, and are less in the early stages from diarrhœal stools than in the later stages from muco-bloody stools. When the dejecta become purulent (latest stages) the bacillus disappears. It is found in the mesenteric glands, but not in the blood, spleen, or liver. Infection is probably carried by contaminated drinking water. Flexner, Barker, Strong, Musgrave and Craig found this type in the Philippines; Kruse found it in Germany, Flexner, Goodliffe and others in America (1901). It has been found in Porto Rico. Duval, Bassett, Wollstein and other clinicians have found it in infantile types. There are *three strains* of the bacillus: (1) The Shiga type, which attacks glucose but not other sugars; (2) Flexner-Harris type, which attacks glucose, mannite and dextrine but not lactose, and forms more acid; (3) Hiss-Russell type, which attacks glucose and mannite but not lactose or dextrine. The

blood of a dysentery subject will agglutinate all types, but particularly the second. The bacillary form constituted 54 per cent. of Strong's and Musgrave's series of 1,328 cases in the Philippines. Epidemics have existed for centuries in Japan, chiefly between the months of June and October. In the Philippines dysentery prevails at the end of the rainy season.

Symptoms.—After an *incubation* of two to eight days, there is a *sudden onset* with fever, reaching 100° to 102° (even 104°), colic, and diarrhœa. Pain becomes marked and the simple diarrhœal stools soon show mucus, and then *bloody mucus* or pure blood, which is voided with great frequency and severe rectal tenesmus. The passages are small, numbering twenty to sixty or even one hundred a day, and contain Shiga's bacillus. The rectum may prolapse. The urine is scanty. In mild cases the symptoms abate in two to three weeks, and the stools become purulent and contain fewer bacilli.

The symptoms arise from *local inflammation* and from *systemic reaction,* not to toxins, according to Shiga, but to solution of the bacilli themselves in the tissues. In the more severe cases the systemic reaction is profound, especially when lesions occur in the lower small intestine, the absorptive power of which is great. A *typhoidal* condition results, with coated tongue, nervous toxæmia, and weak pulse, and death frequently occurs as early as the fourth or seventh day. Shiga names this form entero-dysentery, as distinguished from the ordinary type, colo-dysentery. If the stools become putrid or gangrenous, the outlook is hopeless. Ecchymoses on the chest, epigastrium, and inner surface of the limbs are quite frequent. Some choleriform cases occur. The disease sometimes lasts for weeks or months. Emaciation is marked in acute and chronic forms, which is due to diarrhœa, inanition, pain, and intoxication.

The symptoms are largely explained by the *anatomical findings.* The bacilli lodge more in the small folds and large flexures of the colon and sigmoid than in the small intestine. Their action is furthered by constipation. The mucosa is intensely hyperæmic and shows various grades of inflammation, according to the intensity and duration of the infection. It becomes catarrhal, hemorrhagic, diphtheritic, and finally necrotic with ulceration. The rugæ are wide and swollen; the entire mucosa is swollen, œdematous, thickened, and shows superficial coagulation necrosis, fibrinous exudation, and cellular infiltration; the submucosa is infiltrated and vascular; the muscularis is sometimes infiltrated and the serosa is ecchymotic and freshly fibrinous. In marked cases the necrotic areas become ulcers, in or between the follicles; they have a flat base, occupy chiefly the folds, usually reach only the muscularis mucosæ, and in intense cases impart a sieve-like aspect. The swelling may obliterate the lumen of the intestine. Cocci and colon bacilli are in the upper layers but never in the lower ones, where we find the Bacillus dysenteriæ in and around the glands. In the virulent cases the gut becomes gangrenous. Stenosis may follow cicatrization of the ulcers.

Diagnosis.—*Local rectal disease* (syphilis, cancer, or papilloma) may possibly be confusing. In contradistinction to *amœbic dysentery,* it is generally acute, with a course of two or three weeks; toxæmic symptoms, such as fever, headache and delirium, and hemorrhages, are common, because involvement of the small intestine is common; anatomically the ulcers are flat, are on the folds of the intestine and are never undermined; liver abscess never occurs. Amœbic dysentery is characteristically subacute or chronic. The small intestine less frequently suffers; liver abscess is common (20 to 33 per cent.), and anatomically necrosis begins in the submucosa and produces ulcers with undermined edges. *Agglutination,* similar to the Widal reaction in typhoid, occurs in dilutions of 1 to 20 (even 1 to 50, 100, or 1,000). Shiga observed that it gradually increased as the disease progressed, as Förster noted in typhoid fever. It is usually seen in the second week and most clearly in convalescence, and therefore late for diagnostic purposes. If it increases while under observation and is marked, it is a good prognostic; the converse is true.

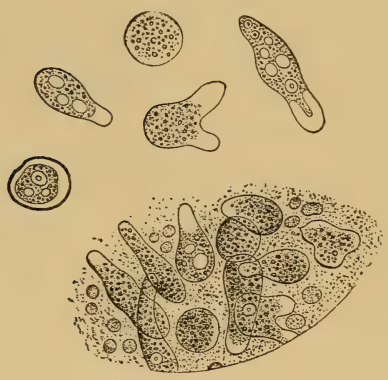
Prognosis.—The prognosis is more favorable in *involvement* of the *rectum* and sigmoid flexure than when the upper colon or cæcum is affected. The outlook is grave in patients under 5, or over 50 years of age. *Intoxication* is a bad prognostic. The effect of *serotherapy* (*v. i.*) is another factor. The mortality is high, particularly in Japan, ranging from 20 to 25 per cent. *No immunity* is conferred. A second attack was observed in 2.7 per cent. and a third in 0.3 per cent. in Ogata's collection of 25,279 cases.

Amœbic Dysentery.—Lambl (1859) and Lösch (1876) discovered an amœba in the bowel movements, but Kartulis, in Egypt, found the amœba in over 500 cases and found the secondary liver abscess. Kartulis established his claims for the Amœba dysenteriæ on the grounds that (*a*) it was constant in certain forms of dysentery, in the ulcers, fæces and liver abscesses; (*b*) it was inoculable in cats; (*c*) negative results were obtained with other organisms and with the amœba which is found in normal fæces. This was confirmed by Koch, Hlava, Fenoglio, Quincke, Kovâcs, and others in Europe, and in America first by Osler (1890); the important monographs of Councilman and Lafleur, Dock, Simon, Musser, Stengel, and Harris soon followed. The home of amœbic dysentery is in the tropics, especially in Egypt, India, Formosa, China, Arabia and in Italy, where it is endemic and sometimes epidemic. Sporadic cases occur everywhere in the temperate zone, especially in Germany and in America, where it is the most frequent form. The author has seen six cases which were clearly acquired in Chicago. In the Philippines it constituted 67 to 80 per cent. of Strong's series. The amœba is found in the pus, glairy mucus, or blood-tinged mucus of the evacuations. It is round in the quiescent stage or irregular from protrusion of pseudopodia, particularly when the slide is slightly warmed. It is unicellular, measures 10 to 50 μ

and consists of a hyaline ectoplasm and a finely granular endoplasm. For diagnosis, it must be motile. The minutely granular nucleus measures 5 to 7 μ. Besides the nucleus one or several vacuoles may be seen in the cell. Its glistening appearance, size, movement, swarming granules, vacuoles, and frequent red blood-cell inclusions, readily differentiate it from swollen epithelia or leukocytes. It stains readily by the Wright-Romanowsky method and in tissues best with thionin. It has been cultivated in pure cultures (Wooley and Musgrave). The amœba probably enters in the food or drink. Unhygienic conditions favor infection. Intestinal amœbiasis (Musgrave) occurs largely in males in the third or fourth decade.

Symptoms.—The symptoms of *acute amœbic dysentery* begin rather abruptly with much the same picture as in Shiga's type, although the fever is generally lower and shorter, rarely lasts more than two or three days, and is sometimes absent. The movements are alkaline, pultaceous, fluid, and yellow, at first, and later, mucous and bloody. Sometimes in one movement fæces are voided first, followed by mucus and blood, or blood-tinged fluid containing amœbæ, slightly yellow, glairy clumps like swollen sago or frog spawn, pus cells, detritus, and sometimes the Charcot-Leyden crystals. The stools may be chocolate-colored. Sometimes large masses of necrotic tissue are voided. The stools number 5 to 20 or more daily; their odor is fetid when fæces are present; they are odorless when only blood or mucus is present; and are stinking when gangrene occurs. The repeated and painful movements cause straining, sweating, and sometimes syncope. *Colic* and borborygmus are nearly always present; pain is referred to the navel; tenderness is usually marked over the sigmoid, colon and frequently over the cæcum, and tenesmus and a burning anal pain mark involvement of the lower bowel, though less than in Shiga's type. The rectum may prolapse and vesical strangury appear. The abdomen is sometimes distended. *Very light cases* show only catarrhal movements, but blood, pus, membrane, or shreds, indicate necrosis and ulceration. Strong men may be ill for months or even die before the malady is suspected. *Severe cases* may begin with chills, fever of 103° or 104°, vomiting, herpes, and symptoms like those of cholera; *e. g.*, 20 to 100 movements daily, leg cramps, scanty urine, failing pulse, rapid emaciation from loss of rest and fluid, and from pain. Early death may result. *Peracute cases*, with chill, high fever, great abdominal pain, collapse, and even sudden death, exceptionally result from rapid necrosis or gangrene.

Fig. 18.



Amœba (Hallopeau).

In the average acute case, the course of one and a half to three weeks, varies with the individual resistance and the treatment. Perforative peritonitis or profuse exhausting hemorrhages sometimes occur. Light cases, if neglected, easily become severe or chronic; spontaneous recovery is quite uncommon.

Chronic amœbic dysentery results from the acute form or is subchronic from the beginning. The symptoms are essentially the same but less intense. There is marked, and sometimes extreme emaciation, particularly in the tropics. A secondary anæmia develops. On the other hand, some patients maintain their weight and suffer only from recurrent diarrhœa every few weeks or months, or from irregular moderate intestinal derangement. In the average case the tongue is lacquered and red, the abdomen is somewhat distended and tender, the lower colon and sigmoid are palpable as a thick cord, and sometimes mental depression or hypochondriasis is observed. The *outcome* is (1) recovery after two to four months; severe infections generally leave intestinal cicatrices or chronic catarrh; recovery is seldom complete after a year's illness; (2) recurrent attacks for months or years, constipation alternating with diarrhœa; (3) complications, such as liver abscess (20 to 33 per cent.), especially in the tropics (peritonitis, empyema, or lung abscess). Strong reports 10 cases of profuse fatal enterorrhagia, coincident with liver abscess. Perforative peritonitis, brain or splenic abscess, pseudorheumatism, myelitis, neuritis, and less frequently, endocarditis, or chronic nephritis, are other complications; (4) gradually increasing anæmia, emaciation and death from exhaustion.

Pathology.—The early changes in the mucosa are catarrh, inflammatory injection, swelling, and hemorrhage, mostly in the large intestine. The essential change is not superficial in the mucosa, but in the *submucosa,* as John Hunter first noted, though Councilman and Lafleur first brought out this point clearly. The amœbæ enter by the tubular glands and reach the submucosa where, by their action alone, without bacterial coöperation, they cause cellular accumulations which are not leukocytes, but chiefly fibrinous exudation and connective-tissue cells. Sometimes the amœbæ enter the blood-vessels early and pass through them to the submucosa. These foci undergo coagulation necrosis and slightly elevate the mucosa which at first covers the small submucous cavities, but afterward sloughs, and exposes the primary grayish-yellow necrosis in the submucosa. When the necrotic tissue sloughs out, the dysenteric ulcers are more apparent. They lie according to Kartulis, in the whole of the large intestine (50 per cent. of cases); in the colon descendens and sigmoid (25 per cent.); in the cæcum, with ascending or descending colon, or in the rectum (25 per cent.); in the rectum alone (20 per cent.); in the cæcum alone (5 per cent.), and only rarely in the small bowel or in the appendix; Musgrave found the appendix involved in 7 per cent., the ileum in 3.5 per cent., and the entire bowel in 80 per cent., of his series of 200 cases. The deep ulceration generally

exceeds the superficial ulceration, and the ulcers in the early stage may communicate with the intestinal lumen only by fistulous tracts which undermine the apparently healthy mucous membrane. Even when the ulcer's opening is large, its edges are generally undermined. Ulceration of the solitary follicles is less frequent than submucous necrosis. The ulcers may be round, or serpiginous and are usually discrete. They may penetrate to the muscularis or to the serosa, which is sometimes perforated, thus causing diffuse or localized peritonitis. The lymphatic structures of the bowel are less affected than in Shiga's type. Secondary changes include membranous diphtheroid deposits (usually streptococcic), diffuse sloughing of entire blocks of the intestinal wall, gangrene, and great hyperplasia of the mucosa. In chronic cases the muscularis frequently thickens, which may greatly narrow the lumen of the intestine. The small intestine may atrophy or hypertrophy. Healing ulcers produce pigmented stellate scars which sometimes cause stenosis. Amœbæ have been seen in the mesenteric bloodvessels, by which they reach the portal circulation (pylephlebitis; liver abscess). The *liver shows* focal necroses which are probably toxæmic, and, in a large percentage, it shows *solitary or multiple abscesses.* The single, large abscess generally develops in the right lobe near the diaphragm or colon. The multiple small abscesses are also superficial. In both types there is central necrosis and softening, the contents are yellow, green or chocolate-colored, contain fat droplets, liver detritus, and the amœbæ lie on the abscess walls. In the older cases there is a firm connective tissue capsule but round cell infiltration is as infrequent as it is in the intestine. Amœbæ may be found in the sputum when the liver abscess ruptures into the lung, or in the pus of secondary empyema. The pseudorheumatism and neuritis are probably toxæmic. The mortality averages 6 per cent.; 7 per cent. of the deaths in Cairo are dysenteric.

Indeterminate Dysenteries.—The classification and cause of these forms are not yet defined, but some are apparently primary and others are secondary infections. The colon bacillus, proteus, pyocyaneus, pneumococcus, streptococcus, and staphylococcus have been found, but their action is still undetermined. Varieties: (*a*) *Catarrhal* dysentery, in which the mucous membrane is injected, cloudy, and infiltrated; the solitary follicles are swollen and often ulcerated,—the " acute follicular colitis " seen especially in children; the ulcers are round, deep, and small. In some instances there is also inflammation in the small intestine. The symptoms are usually mild pain, and muco-bloody evacuations. (*b*) The *croupous* or diphtheroid dysentery represents a severe stage or degree. The epithelium necroses, the submucosa is considerably infiltrated, and there are membranous deposits on the rugæ over slight superficial ulcerations, or there may be deeper, wider ulcers with great thickening of all the intestinal layers. This may occur (i) as a primary infection, with dysenteric symptoms, marked typhoidal symptoms, fever and chill,

and frequently with a fatal outcome; or (ii) as a secondary incident in various infections, e. g., in pneumonia particularly, in typhoid, scarlatina, diphtheria, cholera, syphilis, cardiac affections, cachectic conditions, mercurial or other poisonings and in uræmia. This secondary form may produce marked symptoms or clinically may be latent. (*c*) The *gangrenous* dysentery is attended by the same changes, but results in enormous swelling, softening, and sloughing of dark, gangrenous shreds, and sometimes in tubular sloughs of the larger part of the intestinal circumference. Perforation sometimes occurs. In the Cochin China endemic form, enlarged glands, splenic tumor and intumescence of the liver are found. *Coprostasis, foreign bodies and parasites* are promoting factors in all these types. In some cases the Cercomonas, Trichocephalus dispar, or Anguillula stercoralis may be found. The Distoma hæmatobium (Bilharz) may cause symptoms closely resembling those of dysentery; bloody urine, and strangury result from invasion of the vesical venous plexus by the Distoma; anatomically, hypertrophy of the mucosa, even to the extent of small or large polypi, may result, as well as superficial ulceration and infiltration of the submucosa.

Treatment of all Forms.—1. Specific Treatment.—This is limited to the bacillary type. Shiga's bactericidal and antitoxic serum reduces the mortality at least one-half and shortens the course; in mild cases two and a half drams (10 c.c.) are injected once; in medium cases two and a half drams (10 c.c.) twice, and in severe cases 20 c.c. daily for two or three days. The best results are obtained by early injection.

2. Prophylaxis.—Drinking water should be boiled, and fruits and vegetables should be cooked. In the tropics, sudden variations of temperature are to be carefully avoided.

3. Diet.—Acute and chronic cases must be carefully dieted and the patient must be kept in bed. Boiled milk, raw or slightly cooked eggs, or the whites of eggs beaten with water should be given to relieve colic and tenesmus. The nearest allowable approach to solid food is well-cooked rice or sago. Broths and cold drinks irritate the stomach and excite peristalsis. Some lemonade is permissible. In convalescent acute or in chronic cases, solids should be given late, commencing with well-cooked tender meat and starches, and avoiding vegetables and fruit until convalescence is absolutely established.

4. Eliminative Treatment.—Sodium or magnesium sulphate is given in early cases to cleanse the bowel and to minimize the danger of ascent of the virus from the lower intestine (℥j every two hours until the bowel is thoroughly emptied). Aromatic sulphuric acid, mxx p.c., is sometimes given after the saline treatment. Castor oil is a mild laxative but is of little use except in the early stages. Calomel is considered beneficial by some writers, by whom it is given in frequent and small doses until slight mercurialization is noted.

5. Antisepsis.—Betanaphthol and salol (phenylis salicylas), āā gr. x, t. i. d. or q. i. d., are sometimes helpful, although probably but

small parts of the drugs reach the larger intestine. Bichloride of mercury, gr. $\frac{1}{30}$ to $\frac{1}{20}$, is efficient in some cases.

6. Ipecac.—This was introduced from Brazil, by Piso, in 1648. It is considered almost specific in India and Brazil, but, like the drugs above named, has little effect in the amœbic type. Frayer held that it reduced the mortality from 11 per cent. to 5 per cent. in his series. In India it is given as follows; food is withheld for four or five hours, and then a hypodermic of morphine, or opium by mouth, is administered to quiet the stomach and lessen nausea; 15 grains of powdered ipecac are administered every hour for two, three, or possibly four, doses. If the first dose is vomited, the opium and ipecac are given again. A mustard plaster should be applied to the epigastrium or a turpentine stupe to the abdomen. In Brazil, a decoction is preferred. The *pulv. ipecac. et opii* and salol may be given, āā gr. x, for a few doses.

7. Pain.—Pain necessitates the use of opium, which also quiets the patient, enforces rest and decreases tenesmus and peristalsis. It is given most efficaciously by rectum in a suppository, or, if poorly retained, by enema, mixing Tr. opii deodorat. ♏ xx with an ounce of starch water.

8. Local Measures.—Under this caption the use of *bismuth* by mouth may be mentioned, because its action is for the most part local; doses of ʒj to ij are necessary, and even two ounces daily are given. Musgrave dislikes the use of bismuth because it seems to cake in the ulcers; if employed, an occasional enema should be given to cleanse the bowel. *Lavage* is difficult because of the tenesmus, and is not without danger, for perforation has resulted from copious irrigations. The rectal tube must be introduced carefully as the rectum is very sensitive. Tenesmus may be avoided by injection of a cocaine or laudanum solution, or a cocaine, opium, or iodoform (five-grain) suppository.

℞ Ac. tannici ..... gr. xv.
Extr. opii ..... gr. iv.
Extr. belladonnæ ..... gr. j.
Olei theobromatis ..... q.s.
M. et ft. suppos. no. iv.

Carbolic acid and bichloride of mercury solutions are seldom employed; Bellei employs as an antiseptic and anæsthetic douche, carbolic acid ♏xx, tannin ʒj, marshmallow root ℥ij to a quart of warm water. Among the other solutions of value are quinine, of which Strong advises two quarts of a 1 to 3000 solution, the bag being three to four feet higher than the bed and the flow being slow. A weak peroxide solution for the amœbic variety is recommended by Harris; all enemata should be warm. Strong silver solutions (1 to 500 or less) cause great pain, and weak dilutions are inert; silver, however, is most suitable for chronic cases. In the author's experience, Kartulis' method is the most valuable;—e. g., $\frac{1}{2}$ per cent. solution of tannic acid, injecting as much as the bowel will tolerate

and retain. It kills the amœbæ and two or three injections are usually sufficient. Kartulis has "never been disappointed in hundreds of cases." It may be given in the genupectoral position as an enteroclysis, as advocated by Cantani in cholera.

9. Turpentine Emulsion.—This may be used for tympanites, and the ice-bag, or Leiter's coil with opium, may be used for hemorrhage or peritonitis.

10. Colostomy.—In chronic cases, colostomy has been advocated.

MALTA FEVER.

Definition.—A specific infection, which is endemic on the coast and in the islands of the Mediterranean Sea, is caused by the *Micrococcus melitensis,* somewhat resembles typhoid, but is characterized by a much longer course, by recurrent, irregular, "undulant" fever, severe sweats, splenic tumor, constipation, orchitis, and rheumatoid or neuralgic pains.

History.—Observed about a century ago, it was first described by Marston (1858) as a separate disease. Marston, Bruce and Hughes, English army surgeons, have been the largest contributors to our knowledge of Malta fever. Its distribution is fairly well covered by its names,—Mediterranean, Rock or Gibraltar fever, Malta fever, Neapolitan or Levant fever. It has also been seen on the Danube, Red Sea, Bosphorus; in China, India, the Philippines; in the Canaries, Azores, and Antilles; in Venezuela (Caracas fever), Brazil and South Africa. Less than a dozen cases have come to this country. Musser and Sailer published an account of the first one.

Etiology.—The *Micrococcus melitensis,* which was discovered by Bruce (1887), is round or oval, measures 0.3 μ, often occurs in chains, is non-motile, and stains with methylene blue, or gentian violet, but not by Gram's method. It grows best on slightly acid 1.5 per cent. beef-water, with peptone and agar; cultures grow characteristically in the form of small pearl-like spots; these after a few weeks become yellowish-brown. The bacillus is found in the blood, in the milk, fæces and urine (even two years after recovery), and in the liver and spleen. The apes inoculated by Bruce and Hughes showed a typical clinical course and postmortem findings. The disease prevails chiefly between the sixth and thirtieth years, in the hot, dry weather from May to mid-October, and in old unhygienic buildings. It is not directly contagious. The *atrium* is the lungs (by dust, Hughes), or the alimentary tract (by food or drinking-water, Bruce). In Strong's laboratory infection, the conjunctiva was the point of entrance. It has been recently suggested that mosquitos and goats convey infection; the organism is found in the blood of goats in 50 per cent. and in their milk in 10 per cent.

Symptoms.—Symptoms appear after an *incubation of six to ten days.* Some symptoms so resemble those of typhoid and malaria that the three diseases were formerly confounded. The *fever* rises grad-

ually, reaching 104° to 105° after a few days. The face is red, there are chilly sensations, frontal headache, insomnia, depression, and pains in the back and legs. The tongue is coated, is at first moist, then dry and fissured, and sometimes bleeds. The tonsils are frequently swollen, and the pharynx is red or at times ulcerated. Anorexia, nausea, epigastric pain and tenderness, and constipation, are the rule, but vomiting, icterus, meteorism and diarrhœa,with dark, malodorous, mucous, and even bloody stools, are sometimes seen. Bruce denies that there are any anatomical findings in the intestines. There is considerable bronchitis and the sputum is often streaked with blood. Dyspnœa is quite frequent. The pulse-rate is at first between 80 and 90; later it becomes faster, and is frequently attended by palpitation. Severe sweats occur, usually after midnight, and are accompanied by sudamina (*febris sudoralis*). An acute splenic tumor and hepatic intumescence develop, which are due to degeneration and infiltration by round-cells.

After one to three weeks, the *fever,* which at first is continuous, remits, other symptoms abate, and convalescence is apparently established; but the symptoms again return, frequently several times. The fever curve shows waves accompanying each relapse (literally " undulant fever "). The patient becomes weak, emaciated and pallid. There is no leukocytosis and the micrococci are found in the blood in 60 per cent. of cases.

Complications.—Complications, which are uncommon in typhoid, are frequent. *Pseudorheumatism* occurs in 50 per cent. of cases and is accompanied by redness, swelling, and pain in the shoulders, knees, hips, and sacroiliac joints, and sometimes also in the bursæ, periosteum and fibrous tissues. *Neuralgias* are common. *Orchitis* and epididymitis are usually unilateral and ephemeral. Occasionally *polyneuritis* develops from intoxication and exhaustion and is evidenced by partial anæsthesia, hyperæsthesia, especially of the soles of the feet, paræsthesia and rarely paralysis. Psychoses may follow Malta fever.

Erythematous or hemorrhagic *skin eruptions* occur in the relapses, sometimes accompanied by hemorrhages from the nose, tongue, gums or lungs; desquamation and falling of the hair are common. In convalescence, tuberculosis not infrequently develops.

Course and Prognosis.—In the majority of cases relapses occur after two or three weeks. The average duration of the disease is 120 days, but Bruce records a case which lasted over two years. Hughes described three types: (*a*) The *undulant,* the most common type; (*b*) the *malignant,* which ends in death in from one to three weeks from weak heart, hyperpyrexia (110° or 111°), or possibly lobular pneumonia; and (*c*) the *intermittent,* which lasts for months. The severity of the infection varies; Melland found that 50 per cent. of cases in the Canary Islands lasted from two to three weeks, and in only 10 per cent. did the protracted undulating type occur. The death-rate is constantly decreasing. In the last century it was over

3 per cent. but it is now under 1 per cent. *Immunity* after one attack is usual.

Diagnosis.—The differentiation from typhoid is often difficult. In Malta fever the roseolæ, diazo reaction, Widal, and the bacillæmia are absent. The course is longer, the sweats more severe, and the joints inflamed; orchitis, and neuralgias develop. A positive diagnosis can be made by the use of Wright's *serum reaction* (1897). The blood responds to this test after the fifth day. Agglutination is obtained equally well with the living or dead cultures; the dilution of the blood is 1 to 50, and the time for the test is limited to one hour. The severe bronchitis, fever, and sweats may cause confusion with tuberculosis. Indeed, the older writers spoke of Malta fever as "Mediterranean phthisis." The bronchi in Malta fever are intensely hyperæmic and the sputum is frequently blood-tinged. Malaria, ulcerative endocarditis, febrile pseudoleukæmia, liver abscess, etc., are differentiated by the agglutination test.

Treatment.—There is no specific treatment, though serum therapy is likely to prove valuable (Aldridge, Fitzgerald, Ewart). Quinine and arsenic are ineffectual. Headache and backache are relieved by acetanilide and morphine; insomnia by bromides, chloral and hyoscine; diarrhœa by tr. ferri chlor.; pseudorheumatism by heat, wool investment, tr. iodi, locally, and by salicylates, internally; orchitis, by local heat and suspensory elevation. The general therapy is the same as in typhoid. Milk diet lessens the chance of intestinal complications, though Dalton advocates a solid diet. The milk of goats should be discontinued. In convalescence a change of climate is necessary.

BERIBERI.

Definition.—Beriberi, or *polyneuritis endemica* (Bälz, Scheube), is an endemic and epidemic affection of the tropics and subtropics, of unknown etiology, characterized anatomically by degenerative inflammation of the peripheral nerves, and clinically by motor and sensory disturbances, anasarca, cardiac disturbance and by a subacute or chronic course. *Beri* is said to signify a sheep's gait, and the term *kakke* also means disturbance of the gait.

History.—Beriberi is mentioned in the oldest Chinese writings and was observed in the Roman legions, 24 B. C., by Strabo and Cassius. In the seventeenth century, beriberi was noted in Brazil and in the Malay archipelago; in recent times (1835) it has been described by Malcolmson in India; by Bälz, Scheube, Grimm, and Miura in Japan; by the physicians in the Dutch Oriental colonies; and by Birge, Putnam, and Bonduret in this country.

Distribution.—The main foci of beriberi are (1) the Malay archipelago, Sumatra, Borneo, and Java; also China, India (where it is decreasing), the Philippines, Japan, and even Australia. The mortality has been very high among the Chinese coolies in the Dutch possessions. (2) Africa, including the mainland and adjacent

islands. (3) America and the West Indies, Brazil, Honduras, and Cuba. Scheube doubts that the cases which developed in the United States, in the epidemic at Tuscaloosa, Alabama, were beriberi, but they are apparently identical with oriental beriberi. Cases have appeared in Dublin (1894–1898, with forty-two deaths), in England and in Paris.

Etiology.—The etiology is greatly disputed and there are two distinct views: (1) That it is an *acute infection* (Bälz, Scheube), because it occurs in certain localities as epidemics and endemics, it occurs in the summer months, and in moist, alluvial land near the sea, and near the river mouths,—*i. e.*, the same telluric conditions promote berberi, which promote malaria. It is considered by some persons to be contagious, though no constant microörganism has been found. The fact that many cases develop in houses, barracks, and ships favors the theory of contagion. In the 80.000 cases which developed among the Japanese troops in the Russo-Japanese War, Kokubo frequently found a coccus. (2) The second view is that beriberi results from *food*. Shelled rice is said to have caused it in the Dutch East Indies, and raw fish is considered the cause of the disease among the Japanese (Miura). The use of fish in the navy was forbidden by Takaki, and the mortality fell from 25 per cent. to almost nothing. Natives are more often affected than Europeans, and males more frequently than females. Improper diet may be a cause of beriberi or an indirect factor. In 1879 there were 8,197 cases in Japan and in 1891 this number decreased to one. Poor food, overcrowding and lack of hygiene are important factors.

Symptoms.—The incubation is probably several months, and the disease is due to repeated infections. The *symptoms* are threefold: (1) those of neuritis, (2) dropsy, and (3) cardiac insufficiency.

1. Neuritic Symptoms.—A more detailed description of these symptoms will be given under Multiple Neuritis. Anatomically there is the same symmetrical degeneration (or inflammation) in the distal parts of the peripheral nerves to which, however, it is not limited; the phrenic and pneumogastric nerves and the cardiac, renal, and solar plexuses, are often involved; the nuclei of the sheath multiply; the axis cylinders degenerate; there is a lumpy degeneration in the medullary sheath; and later, there is connective-tissue hypertrophy. In eight cases there were degenerative changes in the cord, anterior horns, and posterior ganglia. Clinically, the onset is gradual and the course chronic.

(*a*) *Motor Symptoms.*—The muscles of the calves, the knee extensors, and the abductors and flexors of the thigh are weakened, flaccid, swollen and atrophied. The muscles are more frequently tender than the nerve-trunks. In severe cases the trunk, arms and even the face, are involved. The muscles are anatomically much degenerated. Phrenic weakness and vagus symptoms, such as rapid heart, vomiting, epigastric oppression, or digestive disorder are not infrequent; the lungs are sometimes acutely emphysematous, from involvement of

the pulmonary plexus. Symptoms of involvement of the larynx, of glossopharyngeal, hypoglossal or ocular participation are more rare. Cramps in the calves and fibrillary contractions are frequent, but ataxia is uncommon, although the name beriberi refers specifically to disturbed locomotion. The gait is described as like that of one walking in wet clothes or wading in water.

b. Sensory Symptoms.—Paræsthesia is common. Hypæsthesia is most marked in the distal parts, though the sole of the foot is exempt; it is more frequent than anæsthesia, which is rarely complete. Hyperæsthesia may occasionally occur over the spine and abdomen. Pains over the joints and intercostal spaces are quite common.

c. Trophic Symptoms.—These are uncommon. They include joint relaxation, herpes or skin atrophy.

d. Reflexes.—The skin reflexes are seldom abolished. Reduction of the tendon reflexes develops equally with the paralysis.

e. Degeneration.—Slight reaction of degeneration is common and may be marked in severe cases. It occurs particularly in the leg.

(2). Dropsy.—Dropsy to some degree occurs in 97 per cent. of cases; it may be marked in the legs, and face, and in all the serous chambers. It is most severe in the pericardium, quite severe in the pleuræ, and least severe in the peritoneum. It is caused by cardiac and perhaps also by vasomotor involvement. Dropsy invariably occurs in fatal cases.

(3). Cardiac Insufficiency.—In the acute cases, death may result in a day, and in 65 per cent. of cases acute forms terminate fatally within two weeks. There is palpitation, cardiac or epigastric oppression, or actual pain; tachycardia, dyspnœa, and cyanosis develop; the right ventricle is hypertrophied and dilated, the first apical tone is weak; there is splitting of the heart tones, gallop-rhythm, and systolic or even diastolic functional bruits; the urine is decreased in amount; the liver is turgid. Anæmia is marked and the leukocytes may be increased. Acute pulmonary œdema is usually fatal. The heart muscle is pale, friable, colloid, or the seat of acute myocarditis.

The *types* of beriberi only accentuate the above-named symptoms. (1) The *rudimentary form* may begin with fever, and catarrh of the air-passages, and alimentary tract. There is a slight œdema of the ankles; muscular weakness, especially in the legs, indicates neuritis; muscular tenderness, paræsthesia, and palpitation are also symptoms. (2) The *atrophic form* is marked by atrophy and paralysis. If œdema does not develop, these types are called "dry forms." (3) The *dropsical form* is characterized by much transudation and pronounced insufficiency of the heart. (4) The *acute cardiac or pernicious form* (*v. s.*).

Diagnosis.—Diagnosis of beriberi is difficult when it occurs in the temperate countries. The leading points are (*a*) the multiple neuritis, which is most marked in the legs; (*b*) vagus neuritis and cardiac insufficiency; and (*c*) dropsy.

Prognosis.—In Japan and the Dutch Indies the death-rate ranges between 3 and 4 per cent.; in Brazil it formerly varied from 50 to 75 per cent.; and in the Malay archipelago it reached 40 to 50 per cent. Death results primarily from heart failure, secondarily from paralysis of respiration, less frequently from uræmia, embolism of the lungs, aspiration pneumonia, dysentery, tuberculosis or marasmus. Even in recovery, permanent residua are common, such as weakness, obstinate contractures of the legs, absence of knee-jerk, and cardiac instability and hypertrophy. In 43 per cent. of the fatalities, there is a sudden death. *Recurrences* are common.

Treatment.—In the early stages of the disease purgation and salicylates are beneficial. In the later stages, electrotherapy and other measures employed in multiple neuritis (*q. v.*) are indicated. The heart should receive stimulation. Venesection is advisable to relieve the overladen right heart and dyspnœa, and promote action of the kidneys. Digitalis is strongly recommended by some, whereas others fail to find any benefit from its administration because the vagus is degenerated. In dropsical forms it is helpful, as is caffeine. The ice-bag over the heart, bromides and belladonna, may mitigate cardiac unrest and palpitation. (See TREATMENT OF VALVULAR DISEASE OF THE HEART.)

ANTHRAX.

Anthrax is one of the *zoönoses* (diseases acquired from animals). Glanders, foot-and-mouth disease, vaccinia and hydrophobia constitute the remainder of this group. Actinomycosis is less a zoönosis than a disease acquired from a common cause by man and beast. Anthrax is also called splenic fever, pustula maligna, and carbunculus contagiosus.

Definition.—It is an acute infection caused by the Bacillus anthracis, and is usually communicated to man from the herbivora.

History.—Over a century ago Morand and Fournier distinguished between the simple carbuncle and that of anthrax, which could be inoculated. The bacilli were seen by Pollander (1855) and Branell (1858), but Davaine (1864–1873) noted that the organisms were bacteria. It remained for Koch with his epoch-making technique to find the spores and to absolutely differentiate the disease, which is now one of the best known maladies.

Bacteriology.—The anthrax bacillus is the largest pathogenic microbe, and measures 3 to 10 by 1 to 1.5 μ. In growths it occurs singly or in chains. The ends are slightly rounded, and the chains resemble the phalanges of the finger, for the organisms lie end to end. They are non-motile, and transparent, and may be stained by Gram's method, and also with various dyes, especially Bismarck brown. At high temperatures they grow in long, thread-like, winding, segmented filaments. Spores are absent in the cadaver and in the living tissue; in the living tissues the bacilli multiply by fission. In attenuated cultures, one spore develops for each bacillus; these become

bacilli which are extremely resistant to drying and to high temperature.

Cultures.—The bacillus is an obligate aërobe which requires oxygen and carbon-dioxide, and grows best on neutral or weakly alkaline media and at a temperature between 95° and 102°. Stab cultures of agar are characteristic. A white, milky yet transparent and band-like growth appears in one or two days, which develops horizontally in blood serum, and grows downward at an acute angle, so that in three days it resembles a quill with a feathered upper end. Growths on gelatin have a thick, wooly covering. The gelatin liquefies after a few days, and a white, granular sediment falls to the bottom of the tube. The bacillus loses its virulence in blood which has been rapidly dried, but in blood which has been slowly dried, it develops spores.

Anthrax is *endemic* and *epidemic* in Europe and Asia. There are persistent foci in Germany, especially in Saxony, parts of Bavaria, and in Frankfort; in France, in the provinces of Burgundy, and Auvergne; in Hungary; in Russia, principally in Siberia (the "Siberian plague"); in China, India, and in South America. Anthrax is rare in America, though small epidemics have appeared in Delaware and Pennsylvania. From Russia, infection may be disseminated by horse's hair and from Asia by mohair (Angora goats). Among animals, the herbivora are most often affected, especially sheep and cattle, and in Russia, horses. Omnivora are more susceptible than carnivora. Mice, guinea-pigs, rabbits, dogs, and fowl rarely acquire the disease. Pasteur thought that earth-worms carried the bacilli from buried bodies to the surface. Transmission of the spores by means of snails, flies and fleas is possible. Spores reaching water may be widely disseminated. The germ may have a facultative saprophytic existence on vegetable media.

Infection may be carried to man from animals, and, on rare occasions, from other human beings. Of Koranyi's cases, 65 per cent. were attributed to sheep and 35 per cent. to cattle; Bourgois considers that the greatest percentage comes from cattle, the second largest from sheep, and the third largest from horses.

External Anthrax.—External anthrax is by far the most common type. The atrium is an abrasion of the skin, possibly through intact hair-follicles. Koch observed that sheep were infected by fly-bites on the exposed skin of the neck. Shepherds, coachmen, and all those who attend diseased animals; butchers, tanners, wool-sorters and persons who handle diseased hides or meat; and saddlers, tanners, rag-pickers and gardeners are most likely to acquire the disease. Jacobi records infection from a hypodermic needle, first used on a patient with anthrax. The spores are more virulent than the bacilli.

Site of the Pustula Maligna.—In Koch's collection of 1,077 cases, the exact distribution is noted in 966; 51 per cent. developed on the head and face, 38 per cent. on the upper extremities, 2 per cent. on the lower, 5 per cent. on the neck, and 4 per cent. on the trunk,

that is, the exposed parts are the usual seat of primary infection. The lesion is usually single, but several pustules or carbuncles may develop, possibly from inoculation by the finger-nails in scratching.

Symptoms.—The incubation lasts two or three days. A small red prominence is first seen, which resembles an insect-bite. It itches, burns, and in twelve hours develops a small vesicle. This is filled with yellow or blood-stained serum, which becomes purulent if ruptured or scratched, and dries with scab formation. It develops a dark, dry, central, necrotic, leathery slough, which is caused by streptococcic infection, and is surrounded by dense round-cell, fibrinous, and often hemorrhagic, infiltration. This in turn is surrounded by œdema of the connective tissue, in the deeper lymph-vessels of which the Bacillus anthracis abounds. If the carbuncle extends, a circle or even concentric circles of new vesicles appear, which fuse and increase the infiltration and œdema. The eyes may be closed by swelling of the lids, or the lips may be so swollen that the jaws cannot be opened. Demarcation now occurs, followed by sloughing; granulation tissue and cicatrization or *general infection* develop, and the adjacent lymph-vessels and glands become infected, soft, and hemorrhagic. This usually occurs on the fourth to sixth day, or earlier in violent infection or reduced physiological resistance. It is marked by depression, headache, backache, pain in the limbs, chilliness, continuous or remittent fever, soft pulse, rapid breathing, vomiting of mucus or blood, vertigo, leukocytosis, colic, diarrhœa, and collapse. Hemorrhages or even gangrene may develop around the carbuncle. Anthrax bacillæmia is sometimes found. The mind is usually clear, although delirium, convulsions or coma may develop, and death may occur on the seventh to ninth day with an antemortem fall of temperature. Toxæmia and septicæmia cause the symptoms given above. In fatal cases there is only slight rigor mortis but rapid decomposition, dark fluid blood, hemorrhages into various tissues, swelling of the lymph glands, and lymphadenoid tissue of the intestine and sometimes of the spleen. In the liver, kidneys, spleen, heart, lungs, brain and meninges, deposition of bacilli and hemorrhages and swelling may be found; the bacilli are seen less in the large vessels than in the capillaries. Bourgois describes another cutaneous form of the disease in which there is great œdema (*charbon blanc,* anthrax œdema), but no pustule or carbuncle. He considers that it occurs more often on the body than does the carbuncle.

Diagnosis.—In most cases this is not difficult. The *simple carbuncle* resembles that of anthrax, in that it is also hard, has a prominent central necrosis, is surrounded by œdema and sometimes is vesiculated. It usually causes great pain, has numerous openings which discharge ordinary pus, and is often malodorous. The *anthrax carbuncle,* however, may be differentiated by the presence of bacilli, depression of the necrotic centre, the corona of vesicles, more rapid evolution, greater œdema, remarkable freedom from pain, and lack of odor. *Glanders* may be distinguished by involvement of the mucosæ,

painful nodes without eschars, and ulcerations which discharge the Bacillus mallei. It is often difficult to recognize Bourgois' *charbon blanc.*

Prognosis.—The prognosis is more favorable in children than in adults, in cases with local than in those with general symptoms, and in those with simple carbuncle than in those with œdema. In Koch's collection of cases, 68 per cent. recovered. Nasarow's figures show 26 per cent. mortality when anthrax occurs in the head and face, 23 per cent. when it occurs in the trunk, 19 per cent. in the neck, 14 per cent. in the upper, and 5 per cent. in lower extremity.

Treatment.—Prophylaxis.—Infected animals should be cremated entire and their hides and wool destroyed. Toussaint (1880) and later, Pasteur, Chauveau and Colbert, attenuated the virulence of anthrax cultures by exposing them to sunlight or increased atmospheric pressure, and used them to immunize animals. In France (1882–1893), 1,788,677 sheep were thus inoculated, with a resulting mortality of 0.9 per cent. and 200,962 cattle were also inoculated, and a 0.3-per-cent. mortality occurred.

Active Treatment.—Wounds in those who have been exposed to infection should be promptly washed with strong sublimate solution. Cauterization with the live cautery, carbolic or nitric acids or caustic potash; surgical excision through sound tissue; and injections of iodine or corrosive sublimate have been recommended, but it is also maintained that equally good results may be obtained by elevation, the use of mercurial paste, and expectant treatment, which at least avoid dissemination of the parasite through sound tissues. Erysipelas serum has apparently been useful in anthrax infections. Sclavo (1903) reported 164 cases which were treated by serotherapy; the mortality was but 6 per cent. In 1,073 cases treated with Mendez's serum, the mortality was but 4 per cent.

II. Internal Anthrax.—Internal anthrax is less frequent than the external type. It occurs in the alimentary and respiratory tracts.

1. Alimentary Form.—The alimentary form (mycosis intestinalis) was first described by Wahl and Recklinghausen, and was more fully discussed by Waldeyer and Münch. Bacilli may enter the system through cuts in the mouth and throat, but usually directly invade the intestines and stomach, which are more frequently infected by spores in milk or water than by diseased meat. The gastric juice kills the bacilli but not the spores. Peasants often eat the pickled meat of diseased animals without being infected. In Wurzen, Saxony, there were 206 cases of intestinal anthrax in 1877.

Symptoms.—There may be prodromal chills, fever, epigastric or lumbar pains, or depression. The onset is, however, usually abrupt, with fever, coated tongue, thirst, and vomiting of mucus or of blood. This is followed by sensitiveness or great pain over the bowels, by serous and afterward bloody movements, meteorism, dyspnœa, weak pulse, and by symptoms of collapse which resemble those seen in the generalization of infection in the cutaneous form. Secondary

carbuncles, or more often hemorrhages into the skin of the neck and of the abdomen, may develop. The course is usually severe and, in some instances, is peracute; death usually results in five or six days in well marked cases, although recovery is possible.

Diagnosis.—The history of the case, the finding of bacilli in the stools and in the blood, or the findings at autopsy, determine the diagnosis. In the small intestine there are changes like those of the cutaneous carbuncle. These consist of localized induration with central sloughing which usually occurs opposite the mesenteric attachment and is accompanied by surrounding hemorrhage and œdema in the mucosa, and of swelling of the solitary follicles, of Peyer's patches, of the mesenteric glands and of the omental and retroperitoneal cellular tissue. The bacillus is found in these lymphadenoid structures in large numbers and sometimes also in the chylevessels and portal bloodvessels. The greatest changes most frequently occur in the upper small intestine, sometimes in the stomach or ileum, seldom in the colon and most rarely in the rectum. More than thirty or forty foci may be seen in the small gut, but the lesions in the stomach or rectum are fewer in number and more discrete. Secondary embolism of the intestinal arteries may result from anthrax of the skin, in which event hemorrhagic stools are uncommon. Serohemorrhagic fluid may be found in the peritoneal cavity. Treatment is without effect, though ipecac, calomel and bichloride of mercury are often recommended.

2. Respiratory Form.—The respiratory form was described by Bell (1879), and Spear and Carpenter (1881) in England, as the "wool-sorters' disease"; and by Eppinger (1894) in Steiermark, Austria, as the "rag-sorters' disease." Infection may, on rare occasions, occur in the nose, larynx, tonsils or bronchi. Particles of inhaled chalk are thought to facilitate infection by producing minute traumata, but the bacilli are apparently capable of entering the uninjured bronchioles and alveoli. Wool-sorters' disease results especially from infection by hides imported from Russia and Brazil, and, in 1897, anthrax developed in this country from hides of Chinese origin.

Symptoms.—Prodromes are uncommon. The symptoms begin suddenly with chill and high fever, tachypnœa and dyspnœa; there are bronchitic and irregular pneumonic findings; the sputum, which in some instances contains the pathognomonic bacilli, is viscid or hemorrhagic. The later symptoms are cyanosis caused by diffuse mediastinal infiltration; serous pleurisy, weak heart, vomiting and the terminal collapse and antemortem fall of temperature. Icterus, hæmaturia, glycosuria and anthrax endocarditis have been observed. The sensorium is clear to the end in most of the fatal cases, though convulsions and coma occur in some cases, due to meningeal hemorrhage. The patient may die on the second or third day. The mortality in rag-sorters' disease is 50 to 87 per cent., and is rather lower in wool-sorters' disease. At the autopsy the special findings are as follows: The bacilli enter the alveoli, wherein they produce alveolar

desquamation and cellulo-fibrinous exudation—the analogue of the carbuncle—and much sero-hemorrhagic exudate,—comparable to the skin œdema. Where there is much cellular exudate, the bacilli decrease and recovery may result from demarcation; where there is much serous infiltration the bacilli are abundant and extension of the infection is likely along the lymphvessels in the direction of the mediastinal cellular tissue, peribronchial lymph glands and pleuræ, which may hold as much as four quarts of anthrax-infected serous blood-stained exudate.

Treatment.—The disease may, to a great extent, be prevented by sterilization of the germ-laden hair, hides or hair used in the manufacture of paper. The actual therapy is wholly symptomatic and supportive.

3. Anthrax Septicæmia.—Anthrax septicæmia is a rare form in which the blood is charged with bacilli, though no point of entrance can be found. It is almost always fatal.

GLANDERS.

Definition.—An acute or more often a chronic contagion, which is caused by the *Bacillus mallei.* It is characterized by the formation of nodules and ulcers, which occur chiefly in the nose and skin and less often in other structures. It is observed largely in the horse, which is the main source of human infection.

Etiology.—Glanders and farcy were known to Aristotle and Hippocrates. The Bacillus mallei was discovered by Loeffler and Schuetz (1882), and independently by Bouchard, Capitau and Charrin. By its discovery, glanders and farcy were identified as one disease. Weichselbaum (1885) first found the bacillus in human infection. It closely resembles the tubercle bacillus morphologically, but is shorter and thicker and measures 2 to 5 by 0.4 to 1.5 μ; it is straight or slightly bent; its ends are somewhat rounded; it is usually single, but may lie in pairs or groups; and is not motile. It stains with the alkaline anilines, easily in cultures but with more difficulty in sections, and the bacillus tinges unevenly, showing lighter areas, which are thought by some to be spores. It is a facultative aërobe and its most characteristic culture is on potato, in which a thin light yellow film develops in two days, soon becomes amber-colored and in eight days assumes a weakly red tinge with somewhat greenish-blue borders. The *atrium* is usually the skin, through which the bacillus enters by some small, perhaps microscopic lesion. Babes considers that infection may take place through the intact mucosa of the nose and air-passages. Horse-bites may cause inoculation. Infection may also occur through the conjunctiva, nose, respiratory tract or very rarely, the digestive tract (Nocard). Man is usually infected by chronically diseased horses; man-to-man infection is far less frequent. The bacillus is found in the nasal nodes (*glanders*); in the skin nodes (*farcy*); in the lymphvessels and glands (in which its

virulence is greatest); in the saliva, urine, sweat, semen and in the blood-current, where it occurs more frequently in man than in animals, in which it is found only in the most acute cases.

The vitality of the bacillus is not great. It is soon destroyed in pus, but may endure for three or four weeks in the viscera of cadavers or three or four months in cultures; it is rapidly killed by sunlight, but persists two or more weeks in damp protected places. It is killed in two minutes at 212°, in fifteen minutes by 1 to 1,000 sublimate solution and in one hour by 5 per cent. carbolic solution. Babes (1890) and Kalming (1891) isolated a toxin, and Kalming, during his researches, succumbed to acute glanders. Hellmann (1891) isolated mallein and Babes (1892) morvine, both of which are successfully injected for diagnosis and for immunization. The horse, ass and mule are the most susceptible animals and from them, goats, sheep, cats, lions, rabbits and guinea-pigs may be inoculated. Babes found that in some localities half the horses have latent glanders. Cattle, mice, rats and fowl are practically immune. Infection is carried by the nasal or cutaneous discharge, whence glanders and farcy are chiefly observed in stable-boys, coachmen, farmers and veterinary surgeons.

Symptoms, Pathology and Course.—These are considered under two topics: (*A*) Farcy, the cutaneous type; (1) acute or (2) chronic; and (*B*) glanders, the nasal type; (1) acute or (2) chronic. In most cases the types are not wholly distinct, though symptoms of one localization may prevail.

(*A*) Farcy.—1. *Acute Form.*—The incubation is from three to five days and is attended by depression, nausea, headache and leg pains. The infected skin shows infiltration, which may heal only to break out anew. Lymphangitis and lymphadenitis are less marked than in the horse and result in lymphangitic farcy-buds. Subcutaneous and cutaneous nodes or areas of diffuse infiltration develop. These swellings are classed with the infective granulomata, are composed of epithelioid, lymphoid and white blood cells, and contain the bacilli, which are free, rarely intracellular and most abundant in the centre of the node. Early sections are succulent and show central necrosis, which is followed by suppuration and ulceration. The ulcers are painful, deep and crater-like, with sharply cut, everted borders and with speckled bases; they have a thin, puriform, hemorrhagic or ichorous discharge and they often fuse. Thrombophlebitis, diffuse phlegmons or gangrene may develop. The rise of the fever may closely resemble the ladder-like ascent of the temperature in typhoid. Chills are common. Polyarthritis and large muscular abscesses are frequent. In some cases a skin eruption occurs which is successively macular, papular and pustular and resembles that of smallpox except that it is rarely umbilicated; it may be scant and scattered, or very diffuse and even confluent; and may affect the mouth, throat and conjunctivæ. The patient is often delirious and has an offensive diarrhœa; the pulse becomes rapid and fresh skin

nodes develop, which are accompanied by ulceration or perhaps gangrene. The pulse is fast, small and irregular; the urine contains albumin or casts, or shows the diazo reaction, and death occurs in about two weeks from exhaustion or from lung inflammation. In the horse, acute farcy occurs with acute nasal localization.

2. *Chronic Form.*—Chronic farcy shows the same skin changes, but they are gradual in onset, slow in progression and unaccompanied by essential inflammatory reaction or by lymphatic invasion. For the first month or two there are pains in the limbs and joints, and after a few weeks, indolent swellings appear in the extremities and in the periarticular tissues. These are followed by ulceration. The ulcers may cicatrize, but break out again after months and therefore somewhat resemble lupus, which heals on one side and develops on another. Muscular abscesses may appear and are very often located in the pectoralis, biceps, brachialis and gastrocnemius. The course lasts for months or years (two to eleven). Recovery occurs in 50 per cent. of the cases (Bollinger), or death results from exhaustion and acute dissemination. In horses the lymphvessels and glands are greatly swollen, and appear as sausage-shaped masses, on account of which fact the term "farcy" was used by Vegetius. In horses the skin eruptions and orchitis are more frequent than in man.

(*B*) GLANDERS.—1. *Acute Nasal Form.*—The conjunctivæ are sometimes swollen, but the process generally begins in the nose after an incubation of three or four days. Miliary nodules develop. These are elevated, are yellowish-white in the centre, grow to the size of a pea, are surrounded by a pale red zone and become ulcerated. New nodes form on the edges of the ulcers, which break down, fuse with them, and discharge a thin, puriform, dirty, sanguinolent fluid. Diphtheroid deposits or crusts form; desiccation may also occur. Ulceration may erode the bone or cartilage and cause perforation of the septum. The nose is swollen and eroded externally, and the lesions may resemble those of erysipelas. The process reaches the mouth by the lymph channels or by thrombophlebitis. Perforation of the palate may occur; the tongue, gums or tonsils are invaded; the angular lymphatic, parotid and submaxillary glands are swollen, and the larynx, and finally the lungs are infected. Beregin found that microscopic nodes may develop in nearly all the parenchymatous organs, the liver, kidney, spleen and brain. In the lungs the nodes may resemble miliary tubercles, or actual hepatization (pneumonia malleosa) may develop and is frequently accompanied by hemorrhage, abscess formation, gangrene, a distressing cough and raspberry, mucopurulent or sometimes stinking sputum. Secondary suppuration in the bones, muscles, joints or skin may develop. The course is that of acute farcy, though perhaps more rapid, and is attended by drenching sweats, diarrhœa, clouded sensorium and convulsions; death occurs in one or two weeks. The leukocytes are increased.

2. *Chronic Nasal Form.*—This is similar to acute glanders except

that it is chronic. It is less frequent, and in forms which are limited to the nose, confusion with other diseases is common. When nasal involvement is slight it may resemble pulmonary tuberculosis, chronic coryza, pyæmia or osteomyelitis. Healing by cicatrization is infrequent. According to Babes, recovery may take place or, at least, the malady may "slumber," possibly to be aroused later by trauma or some other infection, notably symbiosis with the staphylococcus. Death occurs from exhaustion or from generalization by lymphangitis or thrombophlebitis.

Diagnosis of Glanders and Farcy.—Typhoid, pyæmia, rheumatism, erysipelas, variola, syphilis, lupus, tuberculosis and anthrax may cause confusion, but the surroundings, the history and the bacteriology of the case determine the diagnosis. The cultural findings are proven by intraperitoneal inoculation of a guinea-pig, in which orchitis should develop rapidly. Injections of mallein differentiate the disease in 90 to 95 per cent. of the cases for they produce a rise of temperature of three degrees or more. The bacilli are agglutinated by the serum of normal horses in a dilution of 1 to 200, and of those subject to glanders in a dilution of 1 to 1,000.

Treatment.—The prophylaxis of glanders is like that of anthrax. The principal measures indicated are isolation of diseased, and cremation of dead animals, and the use of strong antiseptics in wounds of persons exposed to infection. Skin ulcers should be curetted and bandaged antiseptically; if they are obstinate, the live cautery should be applied. Infection of the nose should be treated by carbolic douches, zinc chloride paste, iodol or iodoform. Arsenic, mercurial inunctions, iodides, quinine and alcohol for the maintenance of strength are recommended. Injections of mallein may prove of therapeutic as well as of diagnostic value (Babes, Pilavios). Large doses of potassium iodide have been recently recommended.

TETANUS.

Definition.—Tetanus, or lock-jaw, is an acute infection caused by the tetanus bacillus and is characterized clinically by greatly increased reflexes, muscular rigidity and tonic spasms such as trismus and opisthotonos. Tetanus is literally tension or stretching. The disease is described in Hippocrates' aphorisms.

Etiology.—In 1884, Carle, Rosenbach and Rattone produced the disease by inoculations of pus, and in the same year, Nicholaier discovered the *tetanus bacillus,* of which Kitasato (1889) first obtained pure cultures from wounds and from earth, and found it an obligate anaërobe. The Bacillus tetani measures 3 to 5 by $\frac{1}{3}$ to $\frac{1}{2}$ μ and is enlarged at one end, in which there is a spore, thus having a pin-head or drum-stick shape. It becomes thread-like in cultures. It stains readily by Gram's method, and is delicately flagellated and motile. It is anaërobic and cultures emit an onion-like smell. It is very difficult to isolate but grows well in sugar solutions. The bacillus is

very enduring and lives even if dried or frozen; the spores may live eight years. It always exists in the primary wound and is thought to exist only on rare occasions in the tissues of the brain and cord, but recent figures show that it is present in 44 per cent. of the severest infections, so that tetanus no longer seems to be a simple toxæmia. The bacillus has been found in the blood and sputum. Tetanus is usually classified as traumatic or idiopathic (rheumatic); the so-called idiopathic form is similar to idiopathic erysipelas, for the *atrium* is either unseen, is microscopic, or is apparently through the pharynx. The bacillus produces *toxins* which are 100 to 400 times as toxic as strychnine and which in turn produce the symptoms. They are tetano-spasmin and -lysin. The bacillus does not produce inflammation, though mixed infections are usual. Tetanus symptoms result from the tetanus toxin alone, but both infection and virulence are enhanced by association with various putrefactive microorganisms, and innocuous solutions of tetanus poison become lethal if combined with sterilized cultures of these putrefactive bacteria. Mice, guinea-pigs and rabbits are inoculable. In *nature* the bacillus is found in the alimentary tract of herbivora, whence it occurs in manure, garden earth, street or house dust, hay, putrefying fluids and in splinters. Though it is spread over the world, the disease is ten to twenty times as frequent in the tropics as in temperate zones, because the bacillus thrives in heat. More cases exist in Prague than in Berlin. It may lurk in certain districts, as it did formerly in the eastern end of Long Island, or as it does in the West Indies where negroes are more often and more seriously affected than whites.

Mode of Infection.—Incised wounds are obviously less dangerous than punctured, gunshot or contused wounds or hæmatomata. Tetanus occurred less frequently in our Civil War than it did in the Spanish-American conflict, in which many cases developed among the Spaniards. It may result from minor operations, as circumcision, hypodermic injections, the administration of antitoxin, tooth-extraction and vaccination (95 cases), and also from major operations. Parturition, lack of attention to the navels of the new-born, the use of imperfectly sterilized cat-gut, and leech-bites or pin-pricks may also cause its development. Chauffard (1903) collected 18 cases in which infection occurred from injections of gelatin for various acute hemorrhages, and Dieulafoy reports 11 cases after injections for aneurysm. Preventive inoculations against the plague caused 19 deaths. The germ is very frequently found in gelatin, which should therefore be sterilized three times in very small amounts, each time for thirty to forty-five minutes at 212°. H. G. Wells has placed emphasis on the danger resulting from the use of toy pistols and blank cartridges for Fourth-of-July celebrations. He noted that 39 per cent. of tetanus mortality of 1903 occurred near that date; 415 deaths from tetanus are listed in the United States for that year as against 89 in 1906; 75 per cent. of cases occur between the tenth and fortieth year of life.

Incubation.—This lasts from six to twelve hours in severe experimental inoculations, from two to nine days in less intense inoculations, and in man, the incubation in 66 per cent. of cases lasts from five to fourteen days; Rose observed that in 33 per cent. of his cases the symptoms appeared in one, and in 45 per cent. in two, weeks.

Symptoms.—These appear suddenly, without any or with only indeterminate prodromes, and are due to toxæmia. This is proven by the fact that autopsies show no constant gross or minute alterations except those common to all convulsive conditions—*e. g.,* points of congestion or punctate hemorrhage. Ehrlich's side-chain theory is a fairly satisfactory explanation, for the anterior cells of the cord and medulla combine with the toxins. Babes believed that some third substance formed and produced the symptoms. These are, at first, *rigidity of the jaw* ("lock-jaw," trismus) and *stiffness of the neck.* In most animals the muscles become rigid near the seat of inoculation first, and sometimes, in injuries received in war, the muscles of a limb may be first involved. The probable explanation of this is that the toxin travels along the nerve trunks (Marie, Morais, Ransom, Meyer). The horse, like man, develops trismus first. The poison incites the anterior cells of the cord and medulla to tonic contraction.

The *facies tetanica* results from a spasm of the zygomatic muscles, which produces the *risus sardonicus,* and imparts a smiling expression to the upper and a sad expression to the lower part of the face (König); there are folds in the forehead; the eyebrows are raised and approximated, the eyeballs directed forward, the masseters prominent, the teeth set and the nares dilated. The spinal extensors are tonically contracted, arching the head backward and the trunk forward (*opisthotonos*); the abdominal muscles are hard and retracted and are occasionally even ruptured by their violent contraction; in rare cases the contracture is lateral (pleurothotonos), or forward (emprosthotonos), or the body is straight and rigid (orthotonos). The limbs are extended, the legs more so than the arms and the feet and hands least. The spasms are chiefly *tonic,* but clonic exacerbations for a few seconds may result from peripheral stimulation of the sensory nerves or those of special sense, by the least touch, jar, breath of air, bright light or sound. The *reflexes* are *enormously increased,* particularly those of the skin. The spasms occasion violent muscular pain, oppression over the lower chest and epigastrium, speechlessness, insomnia and, from participation of the medulla, profuse sweats and increased and irregular heart action. Fever may be present (even 105° to 111°) or the temperature may be normal or subnormal. After death a temperature of 114° has been observed. The *sensorium* is usually clear. Evacuation of the bladder and rectum is difficult, and erection or ejaculation may occur. The urine is decreased and sometimes contains albumin, sugar, acetone or indican; but increased excretion of urea and creatin is not noted. In some cases the lymph glands adjacent to the seat of infection are enlarged. In very rare cases the toxins cause death without muscular spasm; *i. e.,* they combine with other than the nervous tissues.

Death.—Sixty-six per cent. of the cases die in a paroxysm. The usual fatal factor is a respiratory spasm, an œdema or spasm of the larynx, hyperpyrexia, exhaustion or an increasing heart weakness.

Diagnosis.—The diagnosis is based on the nature of the wound, the presence of the Bacillus tetani, trismus (which is necessary for a diagnosis), the facies, substernal oppression and opisthotonos. Serum from cases of tetanus obtained by blistering, is fatal to white mice in twelve to twenty-four hours. In *strychnine poisoning* the history, tests for the drug in the stomach washings and urine, the more abrupt onset of the spasm and reflex excitability, the more diffuse and violent clonic intermittence of the convulsions, their greater involvement of the hands and feet, and the later appearance of trismus and its interparoxysmal relaxation, are entirely distinctive. *Hysteria,* hydrophobia and tetany (*q. v.*) are wholly different from tetanus. In *meningitis* the sensorium is affected. Trismus may be due to other causes as dental caries or parotitis. Inflammation of the temporo-maxillary joint usually results in a tender area in front of the tragus, produces unilateral masticatory cramp and is easily distinguished by the clinical evolution alone. Escherich's *pseudotetanus* is characterized by tonic contractures of the jaw, back and limbs, which occur oftener after an acute infection than as an independent disease, and by convalescence after a few weeks.

Prognosis.—The death-rate averages between 80 and 90 per cent., and is modified by the following factors:

1. Incubation.—The later after inoculation tetanus develops, the better is the outlook. The mortality is 91 per cent. if it appears in the first week, 81 per cent. if in the second, and 53 per cent. if later (Rose).

2. Site of Infection.—Infection in the arms results in a 70 per cent. mortality; in the legs, 90 per cent. (Norris); in cephalic tetanus (the *tetanus facialis* or *kopftetanus* of Rose, first described by Charles Bell, in 1830), the mortality is almost 100 per cent.; Ross (1906) collected 129 cases of this type; J. H. Lloyd finds the prognosis better than in the ordinary tetanus; Lloyd found seven cases of facial diplegia. In this type there is facial paralysis and involvement of the pontine and medullary nuclei, which sometimes causes dysphagia (hydrophobic type) or paralysis of the eye muscles. The author observed these symptoms in a boy who had been kicked over the ear by a horse. *Tetanus* or *trismus neonatorum,* especially described by Beumer (1887), is an infection of the navel which occurs in the second week of life. It prevents nursing and is fatal on the third or fourth day in 83 per cent. of the cases. In the West Indies 50 per cent. of the children died of tetanus at one time, in the Western Hebrides, 67 per cent. and in Iceland, 100 per cent. Anders and Morgan collected 1307 cases (1850 to 1905). *Tetanus puerperalis,* particularly described by Heyse (1893), is a uterine infection, usually follows difficult labor, abortions or ministration by midwives and is generally fatal. In three years there were 232 cases in Bombay (Gowers).

3. Course.—Hippocrates observed that patients who survive the fourth day may recover; 68 per cent. die in the first four days. According to Willard 88 per cent. of acute and 75 per cent. of chronic cases die.

4. Trauma.—The death-rate in traumatic cases is over 80 per cent.; in the so-called idiopathic cases it is 50 per cent. or lower.

5. Sex.—More women recover than men.

6. Symptoms.—Slight trismus and response to antispasmodics are favorable; high fever and involvement of the pons or medulla, respiratory spasm, dysphagia and ocular paralysis are unfavorable.

Treatment.—1. Prophylaxis.—This consists of general surgical antisepsis and special care of *punctured wounds, hæmatomata* and injuries caused by blank cartridges. Iodine and silver nitrate are very helpful, but wide incision to open the punctured wound, for the bacillus is anaërobic, thorough lavage and packing are most essential. Antiseptic conduct of labor, and care of the navel in the new-born, need but little emphasis at the present day.

2. Antitoxin Therapy.—Behring and Kitasato (1880) discovered an antitoxin and were able to immunize animals by cultures which were heated from 122° to 140°, or treated with Lugol's solution, which changes the toxins to toxoids (Ehrlich). The antitoxin was found in animals naturally immune to tetanus, such as crocodiles and chickens, and it was found in the fluids (the blood, milk, and bile) but not in the tissues. Tetanus antitoxin is not bactericidal,—*i. e.*, it does not act on the bacilli. It neutralizes the tetanus toxin in the blood, but *not after the toxin has become fixed in nervous tissue,* from which the antitoxin cannot dislodge it. Behring and Ehrlich think that the antitoxin neutralizes the toxin chemically, while Babes and others consider that cell metabolism is the most important factor. From the above it is obvious that (*a*) serum prophylaxis may be successful in suspected cases of earth infection or in dirty abortions, although the term "successful" is presumptive, as it implies that tetanus would certainly have developed; it has been successful in cases of wounds received on the Fourth of July, and in the Prague obstetrical clinic, in which latter instance every other measure failed; (*b*) if used early it may neutralize circulating toxins which have not yet become attached to nervous structures; and (*c*) when the toxins have combined with the motor cells, it must be ineffectual. Babes estimates the mortality at 70 per cent. even when the serum is used, which confutes the alleged lowering of the death-rate (34 per cent., Engleman; 43 per cent., Köhler; 16 per cent., Behring). The first dose should be 20 to 30 c.c., and later 15 to 20 c.c. should be given two to four times daily. It should be injected near the site of inoculation, and especially into the nerve trunks near the injury, in order to reach the toxins more readily. Subdural inoculations saved a severe case of cephalic tetanus (Holub). The technique is the same as in lumbar puncture (see page 169).

3. Clonic Convulsions.—These may be minimized by absolute

quiet and seclusion, by darkening the room and by careful handling of the patient. *Morphine* may be given hypodermatically to relieve pain, insomnia and cutaneous reflex irritability; one-quarter grain may be given every four to six hours for a day or two, but the pupils and respiration rate must be carefully watched. Occasionally a corrective hypodermic of atropine should be given. *Chloral hydrate* depresses the motor elements of the cord; twenty grains may be given every two hours for three doses, but even this amount may cause dangerous cardiac symptoms in alcoholics or in cases of arteriosclerosis. Digitalis may be given with chloral hydrate to steady the heart, and morphine and potassium bromide to enhance its depressomotor action and to lessen the cutaneous irritability. The bromides should be given, ʒiss every three hours, for six doses, after which the interval should be lengthened. Inhalations of chloroform and amyl nitrite may inhibit the onset of severe attacks.

4. Nutrition.—This must be carefully maintained by the nasal catheter (see Typhoid) or by the rectum. In this way, the difficulties offered by trismus or dysphagia are overcome.

5. Other Measures.—(*a*) J. B. Murphy performs lumbar puncture and injects a weak solution of eucaine and morphine, with apparent results; (*b*) Baccelli, Ascoli and Babes secure better results with hypodermics consisting of 10 drops of 3 per cent. carbolic solution, than they do with serotherapy; no ill effects were observed from long continued daily use; (*c*) Wassermann's proposal to inject subdurally an emulsion of sheep brains is endorsed by Babes and others. (*d*) Meltzer has recently been successful with intraspinal injections of a dram of a 25 per cent. solution of magnesium sulphate.

HYDROPHOBIA (LYSSA; RABIES).

Definition.—An acute specific infection, of unknown causation, peculiar to carnivora, the bites of which convey infection to other animals and to man. The disease is characterized by tonic and clonic spasms of the œsophagus, respiratory and other muscles and by an inevitably fatal evolution. It was described in ancient Jewish, Egyptian and Indian writings and was discussed by Democritus in the fourth and fifth centuries, B. C. The first adequate account was by Youatt (1828, London).

Etiology.—In Germany it is infrequent, because of the strict laws; from 1887 to 1894 only 16 cases occurred. In England, France and America it is far more common and in Russia many cases develop. Australia alone is totally exempt. Dogs are most susceptible, are most frequently affected and are the chief cause of dissemination (90 per cent.); next come wolves, cats and cattle; and finally skunks, horses, deer, pigs, rabbits and even fowl may be inoculated. The character of the *virus* is unknown. It is found *in the nervous tissues most abundantly, in the saliva* and also in small quantities, in tears, sputum, semen, urine and milk and in the adrenals. Its propaga-

tion has been held to occur along the nerve trunk but it must also reach the blood current. It resists putrefaction for fourteen to twenty-four days but, on drying, becomes attenuated in ten to fifteen days. The disease prevails chiefly in warm weather, particularly in the "dog days" and most laws are based on this assumption; but it is also fairly common in cool weather. In Paris most cases occur in March and May. Sixty per cent. of cases occur in males and 40 per cent. under fifteen years of age. Inoculation occurs through the skin and nearly always results from bites of infected animals, which may transmit the disease even during the incubation period. Punctured or deep wounds and those in the nerves or muscles are most dangerous; 17 per cent. of those bitten by mad dogs, 62 to 90 per cent. of those bitten by wolves and 60 per cent. of those bitten by cats die. In exceptional cases, rabies results from being licked by an infected dog, from kissing an infected person or from dissecting an infected body.

The *incubation* period varies greatly, averaging 40 days. It varies with (*a*) the age, being shorter in children than in adults; and (*b*) the site of the wound. If this is on the face or head, the incubation is very short and the symptoms are very severe. The incubation is longer and the symptoms less intense when the point of infection is on the body, because the clothing tends to clean the virus off the teeth.

Symptoms.—Three somewhat arbitrary stages are described.

First or Prodromal Stage.—The scar may become tender or painful; the adjacent glands sometimes swell. Distinct psychical alteration is usual, such as depression, irritability or extreme anxiety. Headache, backache, insomnia, hyperæsthesia to light, sound and touch, anorexia and some elevation of the temperature and pulse rate are usual. Sometimes speech is slightly disturbed, the voice is hoarse, the pupils are somewhat dilated and premonitions of dysphagia are experienced. This stage lasts from one to two days.

Second or Excitation Stage.—(*a*) The *excitement* is more *motor* than psychical. The cutaneous and deep reflexes are increased. There is such hyperæsthesia that the least sound or light, the slightest breath of air, or food and water in the throat, induce violent reflex spasms, whence the patient's dread of these excitants (photophobia, aërophobia, hydrophobia). Tonic spasms, following attempts at swallowing affect chiefly the muscles of the mouth, the hyoid elevators and the laryngeal and respiratory muscles. Even without involvement of the larynx, the respiration is labored, with irregular jerky and often deep or sighing inspirations, during which the shoulders are lifted and the epigastrium is protruded. The spasms may extend to the trunk and limbs. (*b*) The excitement is also *psychical* and is marked at the time of the spasm by restlessness and often late in the course by delirium, horror or even by violent mania. Delirium tremens may be simulated. The mind is usually clear between the spasms. The patient rarely attempts to injure attendants, although such an impression may be made by the patient's

restlessness, the peculiar growling sounds occasioned by pharyngeal and laryngeal spasm and the "frothing at the mouth," which is due to inability to swallow the increased flow of saliva. Other less constant symptoms are elevation of temperature from 100° to 103° or 105°; salivation and lachrymation; thirst; tremors, erections and ejaculations; increased pulse rate; scanty, albuminous urine (25 per cent.), often with casts. The excitation stage lasts one and a half to three days.

Third, Paralytic or Terminal Stage.—This stage is less common in human than in animal rabies. In animals it may be marked from the beginning and the other stages may be lacking or rudimentary—the "dumb" rabies as contrasted with the furibund type. The spasms and excitement cease, the pulse becomes fast, small and irregular, the pupils are wide and irregular, sweating is profuse, swallowing becomes impossible and paralyses develop, mono-, para-, or hemiplegic, or of the acute ascending type of Landry. The sphincters are sometimes paralyzed. The patient dies, unconscious, of cardiac collapse, rarely of asphyxia or convulsions. The third stage seldom lasts more than eighteen hours.

Autopsy Findings.—Pathologists are not unanimous in regard to the specific autopsy findings. Minute hemorrhages and special congestion of the medulla and cord mark hydrophobia as they do other convulsive states. Babes' *rabic tubercles* are aggregations of leukocytes around the vessels and nerve cells. The latter may degenerate. Rapid diagnosis of rabies may be made according to von Gehuchten and Nélis, by examination of the sympathetic and cerebro-spinal ganglia, under the capsules of which are found *accumulations of epithelioid and lymphoid cells*, in animals, only, which died of the disease. The bodies described by Negri and Pavia (1903) in the cell processes may be protozoal parasites or artefacts.

Course and Prognosis.—In twelve hours to ten days—on the average two to four days—all untreated cases die, 10 per cent. in the first day and 60 per cent. in the first three days. Högyès' is the only recovery verified by inoculations of the saliva.

Diagnosis.—The diagnosis is usually made with ease. After bites from supposedly rabid animals, microscopic examination of the plexiform ganglion of the vagus can be made and also subdural inoculations in rabbits, which die in two or three weeks with paralytic symptoms. *Tetanus* of the cephalic and hydrophobic type is easily distinguished by the invariable trismus. *Hysteria* (pseudohydrophobia, lyssophobia) in one bitten by a dog may at first cause suspicion but rarely any protracted doubt. The initial spasmodic dysphagia and irritability are not followed by the other symptoms and stages of hydrophobia, and the clinical evolution alone is distinctive.

Treatment.—Prophylactic.—In Australia, where laws concerning dogs are not only passed but are enforced, hydrophobia is extinct, while in Roumania over 100 cases occur yearly. In human rabies it must be remembered that the saliva is infective.

Preventive Inoculation.—Pasteur, in 1881, found the rabic virus

in the nervous tissues. "The virus of the street," as Pasteur called it, varies in virulence and subdural inoculations from the brains of rabid dogs, into rabbits, killed them in periods varying from fourteen to twenty days. By inoculating a series of rabbits, the incubation period is progressively shortened to a fixed period of seven days, this virus being called the "fixed virus." Pasteur attenuated this virus by suspending the spinal cord of the rabbit in a clean jar and desiccating it with caustic potash. Animals were inoculated with virus which had been desiccated for two weeks, then with virus a little stronger,—*i. e.,* with virus desiccated for ten or twelve days, until finally immunization was secured against inoculations which previously would have been fatal. In 1885, human beings bitten by rabid beasts were thus treated by two inoculations daily, for fifteen days and immunization was secured, for the slow rabic incubation gives ample time for treatment. Early treatment secures the best results. Statistics show that the former mortality of 80 per cent. in untreated bites in the head or face has been reduced to 1.25 per cent.; the mortality of 60 per cent. in those bitten in the hands is reduced to 0.75 per cent.; the mortality of 80 per cent. in those bitten in the covered parts of the arm and trunk, and of 15 per cent. in those bitten in the leg, is reduced to 0.25 per cent. *The total mortality has been reduced* to 0.5 *per cent.* As sequences of Pasteur's treatment, neuritis, bulbar or spinal and Landry's paralysis are very rarely found (Babes, 1905).

2. Primary Wound Treatment.—The wound may be sucked by the victim, but by no other person. It should be washed with strong antiseptics, burned with caustic potash, pure carbolic acid or nitrate of silver, and as soon as possible should be exposed surgically, treated with the live cautery and packed with gauze, so that healing may be delayed for over a month.

3. Late Treatment.—In developed cases the mortality is 100 per cent., whence curative therapy is out of the question. The treatment of the *convulsions* is similar to that of tetanus, and nutrition is maintained by nutritive rectal enemata. Morphine and inhalation of chloroform are better than chloral and bromides and should be given until they show their full physiological effects. Cocainization of the mouth and pharynx may overcome the pharyngeal spasm incident to swallowing milk and water and to medication. Tracheotomy is useless for the respiratory spasm, which reaches further than the larynx and involves the muscles of the chest and diaphragm; and besides, it is maintained by many that the larynx itself is not closed.

ACTINOMYCOSIS.

Definition.—An infection caused by the actinomyces (literally "ray-fungus"); it is mostly sporadic in cattle and less frequently so in swine, horses, deer or elephants; it occurs occasionally in man; it is rarely endemic or epidemic; it is marked by chronic inflammation

(*a*) in the mouth, head or neck, (*b*) in the lungs or (*c*) in the intestines or abdomen; and it usually runs a chronic course.

History.—Von Langenbeck (1845), Davaine (1850), Laboulbêne and Robin (1853) and Lebert (1857), observed the peculiar sulphur granules in the discharges of pus. Rivolta (1868) and Perroncito (1875) described the fungus, Bollinger (1877) first scientifically established its constant occurrence in cattle and Hartz named it *Actinomycosis bovis.* J. Israel (1878) first found the parasite in human cases, but to Ponfick (1879) is due the credit of identifying the bovine and the human types. William T. Belfield, of Chicago, first found actinomycosis in this country. The monographs by Ponfick, Israel, Bostroem and Illich are the most important.

Etiology.—Its *distribution* varies. Statistics in Berlin, Breslau and Vienna show large numbers of cases. There are more cases in Scotland, Denmark, Italy and Russia than in America, where it is less frequent in the United States than in Canada. Up to 1902, 100 cases were observed in America (Erving). Seventy per cent. of the cases occur in males and most of them are in subjects between twenty and forty years of age (33 per cent. between twenty and thirty; 82 per cent. between ten and fifty). The fungus enters the system chiefly by the mouth, in which ragged teeth, abrasions, etc., favor its access. Though almost nothing is known of it outside of the body, it probably exists in the intestines of herbivora. Most cases occur in young cattle at dentition; 89 per cent. in the last half of the year, when the hair and the grain which are ingested are dry. The drinking water may be the source of infection.

Bacteriology.—The *parasite* of actinomycosis is classed as a streptothrix and is found in small granules which measure 0.15 to 0.75 millimeters, are usually ovoid in shape and show a "radiate" form when squeezed under the cover glass. The younger forms are transparent, sago-like, white-gray bodies which may be easily overlooked and the older forms are more yellow. These *sulphur-like granules,* on minute examination, consist of threads and coccoid bodies which some consider spores. The older yellow forms become clubbed, which is considered a degenerative change. The threads divide and branch. The fungus is therefore phleomorphic. It stains fairly well with the usual stains and by Gram's method. Cultures are successfully grown in 50 per cent. of the cases, and are best obtained by mixing the granules thoroughly with softened gelatin and bouillon and then by spreading or inoculating them as usual. The colonies are thick, folded, membranous, resistant and at first have a mucoid or crystalline appearance. Experimental inoculations may succeed but infection is very rarely direct either from animals to man or from one person to another. The virulence is, therefore, of low grade.

Forms.—Incubation averages four weeks. Clinically there are *three main forms:* (1) That of the head and neck; (2) that of the lower digestive tract; and (3) that of the respiratory tract.

1. Head and Neck.—The forms localized in the head and neck

constitute 50 per cent. of actinomycosis. The atrium is in the soft tissues of the mouth and throat. Entering these, the fungus is surrounded by a massive accumulation of round cells which soon degenerate centrally. The reaction of the connective tissue is marked and the proliferation shows numerous round or polygonal cells with large nuclei,—the epithelioid cells. Giant cells also form. The process is designated by Bostroem as a chronic inflammation, and by Israel as an *infective granuloma,* resembling the tuberculous, syphilitic or leprous process. The further destiny of these granulations in the tissue is modified by the resistance of the tissues and by the animal diseased. (*a*) *In man* the granulation wall usually melts so that the fungus is surrounded by a mucoid, creamy, cloudy fluid, in which there are oil, fibrin, blood, pigment and actinomycotic cocci and threads; the "cold" abscess is formed by the fungus itself which, unaided by pyogenic organisms, burrows and reaches the surface by circuitous *fistulæ;* it discharges a thin, puriform secretion like that of scrofulous glands but contains the *sulphur-like grains.* (*b*) *In cattle and horses* the granulation tissue is more effectually obliterative and limitative and hard, board-like or ligneous tumors develop ("lumpy jaw," "wooden tongue"); suppuration and miliary dissemination are less frequent, and calcification is more frequent than in man. In human subjects the infiltration may involve the neck, face, tongue, pharynx, tonsils, larynx and thyroid gland. In general, the most frequent involvements are inflammation of the soft tissues about the jaw, especially those under the lower jaw, submaxillary or submental infiltration and superficial invasion of the bones. In the tongue, actinomycosis may resemble gummata or cancer but in cancer there is more pain and glandular invasion. In some few cases the maxillæ may be primarily invaded (Israel, Murphy). *Sarcoma* and *caries* may be simulated. The process sometimes *spreads regionally* along the bones to the base of the brain and results in meningitis; along the spine and causes vertebral erosion; and behind the sternum with mediastinal infiltration.

Symptoms.—These vary with the part invaded. The fever is irregular, rarely continuous. Pain and tenderness are not marked, for the process is indolent or chronic. Dysphagia accompanies retropharyngeal involvement, œdema occurs in laryngeal localization and other local interference such as venous obstruction is frequent. The writer observed trismus and severe unilateral trifacial neuralgia from a primary focus at the base of the skull; recovery followed operation. Besides the regional invasion by contiguity, above named, *hæmatogenous extension* by metastasis may occur in this type as well as in the others to be described later. Unlike tuberculosis, extension to the lymphatics, or general dissemination by them, is unusual. Subacute hæmatogenous, *pyæmic deposits* may occur with a very wide distribution. In the head-and-neck form the mortality is 10 per cent.

2. Digestive Tract.—The lower digestive tract ranks next in frequency of infection (20 to 30 per cent.). Any tissue between the œsophagus and rectum may be diseased but 50 per cent. of this group occurs in the region of the ileum, cæcum and appendix (*perityphlitis actinomycotica*). Of 200 instances of inflamed cæcum, 4 per cent. were actinomycotic (Friedrich). Local pain, tenderness, induration and adhesions frequently follow the primary lesion of the intestine. In rare instances this lesion is superficial (enteritis actinomycotica superficialis, Chiari), but small nodes usually develop in the submucosa, followed by central softening and the development of small ulcers, the undermined edges and uneven bases of which resemble tuberculous ulcers. Secondary pyogenic infections are frequent. The ulcers may cicatrize or fuse into larger ulcerations.

Symptoms.—The signs are often those of appendicitis; symptoms may be absent, but colic and vomiting are often present and constipation is more frequent than diarrhœa. *Extension* of the process is usually downward; it involves the iliac bone and occasionally the thigh, hip, bladder, ovaries, vagina or rectum; the ray fungus is voided in the urine and fæces; and anteriorly, preperitoneal, suprapubic or muscular infiltrations occur, with external fistulæ. The process then extends backward into the retro-peritoneal cellular tissue, in which it may burrow around the kidneys or even into the chest. These varied localizations may simulate perinephritis, peripsoitis or parametritis. In rare cases, freely movable, and therefore operable tumors may develop. Vascular metastases may occur but suppuration in the liver is the most frequent development, and is secondary clinically and pathologically, though it is sometimes apparently primary (forty cases are reported). In 71 per cent. death results from exhaustion, mixed infection or amyloidosis. Diagnosis must be made from the yellow granules or microscopic recognition of the fungus.

3. Respiratory Tract.—Respiratory involvement is still less frequent. It is (*a*) secondary to extension by contiguity from the neck or abdomen, or to miliary or larger infarcts, or (*b*) primary in the lungs, which is a less frequent form (14 per cent.). Primary pulmonary actinomycosis is rarely of the superficial type (bronchitis actinomycotica superficialis). Canali described a case of seven years duration. This form may easily escape recognition. A case of actinomycotic fibrinous bronchitis is reported. More often it is deeper and invades the alveoli. The lower lobes, its usual seat, show a gray hepatization with the ordinary actinomycotic changes. Fusion of the usually small foci may involve large portions of tissue, but demarcation is usual in the lung. The clinical picture varies. (i) In some instances *cavity formation* is obviously present. There are fever of an hectic type, an harassing cough and malnutrition; the sputum is puriform, sometimes fœtid and stained with altered blood like raspberry jelly and often reveals the actinomycosis rays. (ii) *Induration* may occur from cicatricial development. (iii) *Pleurisy;* (iv) *peri-*

pleuritis; (v) pericarditis; or (vi) mediastinitis may be the dominant finding. There may be much luxation of the intrathoracic contents, and involvement of the heart is characterized by great dyspnœa, dilatation and hydrops. Penetration may occur through the chest wall with characteristic discharge from the sinuses which run along the spine and iliopsoas muscle, or into the abdomen. Hæmatogenous generalization occurs as in other forms. The examination of the sputum is essential for diagnosis unless fistulæ develop. In 25 per cent. of cases the ray fungus is seen and *tuberculosis* is excluded by absence of tubercle bacilli and elastic fibers. Otherwise the resemblance may be very close or differentiation impossible. The *course* is usually subchronic (averaging under a year), less often chronic (two to three years) and very rarely acute. Only 6 recoveries are reported.

4. Other Localizations.—Other primary localizations are rare; 23 primary skin cases are reported. They may resemble lupus or tubercle. Primary brain lesions are infrequent; they may simulate brain tumor or cause cortical epilepsy. Primary genito-urinary infection is possible, though very few cases are recorded (Stanton). The lachrymal duct or middle ear is very rarely the primary focus.

Diagnosis.—Similar fungi may cause difficulty in diagnosis, but their classification is still a mooted question. Eppinger's cladothrix asteroides may cause deposits in the cervical glands or brain, and miliary nodes in the lung. Rosenbach noted a similar growth in erysipeloid, and Vincent in Madura foot, which produces a red pigment. The leptothrix of the mouth is a possible cause of confusion.

Treatment.—1. Prophylaxis.—Besides general measures employed in any contagion, care is necessary in regard to the holding of straws in the mouth or picking the teeth with them. Thorough cooking of cereals before eating is essential.

2. Surgical Extirpation.—This is indicated, if possible; or a sharp spoon should be used to clean accessible foci and fistulæ, which should then be packed with iodoform or sublimate gauze. Injections of bichloride of mercury ($\frac{1}{10}$ to 1 per cent.) are used especially by Illich and Hochenegg. Recurrence is frequent in abdominal types.

3. Potassium Iodide.—In cases wholly inaccessible to the knife, potassium iodide is excellent or even specific, according to some writers. It is not parasiticidal, but softens the tissues, prepares operable cases for surgical measures, inhibits growth and minimizes metastasis; 53 per cent. of bovine cases recover under its use. Thomassen and Netter use six grams daily for a month and then decrease the dosage. Rydygier injects large quantities of a 1 per cent. solution into the affected tissues.

4. Tuberculin Injections.—These were advantageous when used by Billroth, Kahler and Socin. Bevan has seen benefit result from the internal use of copper sulphate, $\frac{1}{4}$ to 1 grain.

GONORRHŒAL INFECTION.

Lightly spoken of as "the badge of virility," gonorrhœa is often untreated and neglected. It frequently becomes chronic, and in its so-called latent form may be conveyed after marriage to the wife, on whom it usually entails suffering, frequently sterility, and sometimes permanent invalidism. Its other dangers are:

I. Regional Invasion.—The regional invasion of the genital tracts (*a*) in the male, of the prostate, seminal vesicles and testes; (*b*) in the female, of the vagina, cervix, uterus, tubes, ovaries and peritoneum; in institutions, repeated epidemics of vaginitis in children have occurred; (*c*) in both sexes, of the rectum, bladder, pelvis and kidneys, in which extension, mixed infection is an important factor.

II. Gonococcal Septicopyæmia.—Hæmatogenous generalization has already been discussed under septic infections. The fever is toxæmic and septicæmic. The fever is intermittent and is often marked by great variations. Injection of sterilized cultures produces violent reaction, which hitherto has baffled attempts at animal immunization. Among the gonorrhœal invasions of tissues other than the urethra, Wertheim first found the organism in the bladder wall. The gonococcus has been found in the blood 29 times (first by Thayer and Blumer, 1895); in the myocardium (Councilman); in the pleura (15 cases collected by Lemoine and Gallis, 1905); and in the joints (v. i.), kidneys, spleen, muscular abscesses, lymph-glands and skin. It has also been found in meningo-myelitic foci; in myositis (7 cases collected by Harris and Haskell, 1904); in arteritis and phlebitis (25 cases collected by Heller, 1904; the author saw one in 1905 with Dr. Cuthbertson); in felons, osteomyelitis and periostitis. 49 cases of ulcerative gonorrhœal endocarditis were collected by Kulbs in 1904. Septicopyæmic lesions may be due to Neisser's diplococcus alone, or to mixed infections.

Most cases of *gonorrhœal arthritis* must be regarded as a form of mild septicæmia. Occasionally it is part of a severe septicopyæmia. It was described by Selle and Swediaur in 1781, but first clearly by Brande, 1854; the gonococcus was first found by Petrone (1883) and Kammerer (1884) and cultivated by Höch (1893). According to Damasch it develops in 2 to 3 per cent. of gonorrhœal infections. König asserts that it is the most common disease of the joints. Eighty per cent. show an acute urethritis; in the balance the urethritis has subsided or become chronic. It has followed experimental gonorrhœa. In 18 cases reported by Lucas, the infection was primarily gonorrhœal conjunctivitis, especially in infantile cases; the coccus reaches the blood by way of the nose. Northrup's series of 252 cases of gonorrhœal arthritis gives 91 per cent. in males; Brun and Bennecke find 62 per cent. in women, which is contrary to common experience. About 50 per cent. of the cases occur between twenty and fifty years of age. Too active treatment and sexual indulgence during the florid stage of gonorrhœa are held to be predisposing causes.

Symptoms.—The symptoms are extremely variable. Fever rarely exceeds 102°. The joints alone may be involved, or there may be more severe evidences of blood invasion, such as endocarditis. While the synovial membrane is most often inflamed, gonorrhœal "rheumatism" is more than synovitis. The accessory bursæ, contiguous tendon sheaths and the extracapsular structures are often involved, whence the name of *gonorrhœal arthritis.* It is often held that it affects one joint (monarticular) or at least few joints (oligarticular). Though fewer joints are usually involved than in genuine rheumatism, in Jullien's collection the affection was polyarticular in 60 per cent.; in Northrup's series more than one joint was affected in 78 per cent. of the cases, and three or more joints in 69 per cent.; and in Gerhardt's series two to nine or more joints were involved in 87 per cent., and one joint only in 13 per cent. Gonorrhœal arthritis frequently involves joints which are usually unaffected in acute articular rheumatism, *e. g.*, the sacro-iliac synchondrosis, sternoclavicular, temporo-maxillary and intervertebral joints. The knee is affected in 70 per cent., the ankle in 27 per cent., fingers and toes in 19 per cent., hip in 13 per cent., wrist in 12 per cent., shoulder in 10 per cent. and the elbow in 9 per cent., of the cases. Within the joint a greenish serofibrin exudes, and without the joint there is œdema and infiltration. Suppuration is not frequent but may occur with or without coincident pyogenic infection, especially in the joints and tendons of the hand. The gonococci are found with variable frequency, the highest reported percentage being 75.

König's *pathological classification* includes (1) *simple hydrops* of the joint; (2) *catarrhal* or serofibrinous *hydrops;* (3) *pyarthrosis,* and (4) *gonorrhœal phlegmon,* affecting the soft parts. A *clinical classification* may be made as follows: (1) *Arthritic form,* pain with exudation; (2) acute or subacute *polyarthritis,* by itself, or with septicæmia; (3) polytendosynovitis and polybursitis, often with periostitis,— this is the *periarthritic form;* (4) *acute monarticular form,* with local reaction, sometimes with suppuration; and (5) the *chronic monarticular hydrarthrosis* especially of the knee, a far less frequent form. We might add (6) the forms in which simple arthralgia is present or the process localizes in the heel.

Course.—The course is essentially chronic and is sometimes relapsing, or marked by complications; iritis is fairly frequent; atrophy in the contiguous muscles may occur as in rheumatism; ankylosis may result, which is far oftener plastic than osseous. In exceptional instances, sciatica, permanent arthritic changes resembling arthritis deformans (*q. v.*), exhaustion psychoses or multiple neuritis develop.

Treatment.—Recovery is a matter of time and nature, rather than of therapy. The salicylates are almost wholly inert and the iodides highly unsatisfactory. In the acute stage, immobilization by plaster casts or splints, and in the chronic stage, blisters, Pacquelin cautery, massage, passive movements and extension, to overcome residual exudation and contractures, are useful. Some cases are helped by

Roentgen rays and by superheated air (See Arthritis Deformans). Injections into the joint of 1 per cent. formalin are advantageous. Bier's passive congestion is frequently of great benefit. Surgical incision and simple irrigation are indicated after a reasonable lapse of time and after the failure of expectant treatment. In some intractable cases, however, there is no fluid to drain. The vaccine therapy promises results.

SYPHILIS (POX, VARIOLA MAGNA, LUES VENEREA).

Syphilis is evidently an ancient disease as is indicated by probably syphilitic bones belonging to the stone age found in Japan. Nusi King described syphilis in China 2600 B.C. The first clear outbreak occurred in 1494 among the troops of Charles VIII, who was then investing Naples. A full account of this disease will be given because of its colossal importance.

Definition.—A specific chronic infection, found in man only, either hereditary or acquired by inoculation, in the seat of which a primary lesion, hard chancre, develops. This is followed in two or three months by secondary lesions in the skin and mucosæ. Later, after months or years, tertiary lesions or gummata develop in the viscera, bones and skin, and these are finally followed in some instances by meta- or parasyphilitic manifestations, as tabes dorsalis, dementia and aneurysm. This division of Ricord is most convenient but does not invariably hold,—*e. g.,* in hereditary syphilis and sometimes in the malignant, acquired variety, where the stages overlap. Virchow finds a very close histological resemblance between the primary, secondary and tertiary lesions.

Stages According to Ricord.—The Primary Stage.—(1) Incubation lasts until the *chancre* appears, when syphilis is already a general disease; (2) the chancre develops, the lymph-vessels and adjacent glands enlarge, and there is a period of latency, sometimes called the "second incubation."

The Secondary Stage.—(1) Symptoms prodromal to the eruption, as fever or anæmia, develop and then (2) the *eruption* appears on the skin and mucosæ, with headache, "rheumatism," iritis, or sometimes jaundice, albuminuria and splenic tumor.

The Tertiary Stage.—This may follow very closely upon the secondaries or appear with them (syphilis maligna), but usually is not manifest for three or four years (even fifty or sixty). Tumor-like gummata occur in the skin and viscera, especially in the liver, scrotum, brain and cord. Unlike secondary manifestations, they are asymmetrical, persistent, recurrent, less contagious, and respond to different therapeutic agents.

The Quaternary or Parasyphilitic Stage of Fournier, Sigmund, Lancereaux and Baümler is not included in Ricord's division.

I. Acquired Syphilis.—Etiology.—1. Transmission by Sexual Intercourse.—This causes the great majority of cases, but the term

lues venerea is often incorrect and unjust since syphilis may occur in the innocent (syphilis insontium); in certain districts in Russia 70 per cent. of syphilis is innocent. In 1905 Schaudinn and Hoffmann described a *spirochæte pallida,* so named because of the difficulty in staining it: It is long, delicate, thread-like, refractile, actively motile, pointed at its ends and twisted like a corkscrew; it measures 4 to 20μ in length; it lies free and not in the cells; it resembles the spirochæte of relapsing fever but is a flagellated protozoön. It is present in all cases, stages, types and tissues, and probably is the cause of syphilis. It is found in anthropoid apes which were successfully inoculated by Metschnikoff and Nicolle. With Giemsa's stain[1] the spirochætes stain a delicate violet and the leucocyte nuclei deep red. The spirochæte refrigens, found in the mouth, in ulcers, smegma and venereal warts, is differentiated by its being more quickly stained, its greater size and, particularly, by its broader and more wave-like undulations. (See Plate VI.)

The sound skin and mucosæ resist infection, and hence a raw surface, erosion, wound or hyperæmia is necessary for infection. Syphilis not only has a certain *choice of place in its inoculation,* but *persists with peculiar obstinacy in certain localities,* notably in the genitalia, under the breasts, in the axilla, in the navel, between the toes, in the angles of the mouth, on the tongue, iris, palate or tonsils, because of irritation by secretions, pressure by clothing, trauma or irritation from smoking, etc. These foci are of great moment in the awakening or reproduction of the disease. Syphilis is sometimes called a disease of cities; though more common in cities, syphilis very frequently escapes recognition in the country.

2. Accidental Infection.—Accidental infection occurs in surgical or obstetrical practice, in skin-grafting, by postmortem infection (two cases of personal observation), in ritual circumcision, in nursing, vaccination, kissing, from use of pipes, in shaving, by scratches, from instruments used in glass-blowing, or from catheters.

Symptoms.—1. Primary Stage.—After an incubation of two to three weeks, the *initial lesion* occurs (the *ulcus durum,* Hunterian chancre or initial sclerosis). Multiple lesions occur in 25 per cent. (Fournier records 26 simultaneous chancres.) The *induration* is usually oval, is always sharply localized, of variable or even cartilaginous hardness, freely movable, and usually papular and copper-colored. *Hardness* is the most constant characteristic of the chancre; it may last a long time, and heals only when the hardness disap-

[1] Azure II (azure plus methylene blue āā) 3.0
Eosin (B. A.) 0.8
Glycerin (U. S. P.) 250.0
Methyl alcohol (Kahlbaum I) 250.0

It is prepared by grinding up the dyes in the absolute alcohol and then adding the glycerin. The blood films are fixed for a minute in absolute methyl alcohol and then stained for five minutes in a mixture of 14 drops of the dye to 10 c.c. of distilled water, which is freshly prepared; a trace of sodium carbonate may be added to the water to intensify the basic colors. After washing in water the films are blotted.

PLATE VI

Spirochætes of Syphilis. (Simon.)

a, S. refringens; *b*, S. pallida. (Stained with Giemsa's stain.)

pears. It cannot be diagnosticated before the sixteenth day, and is most characteristic in the third or fourth week, when the adjacent glands enlarge. Retrogressive changes as *ulceration* or necrosis are frequent. The *ulcer* varies in size, is round, elliptical, slit-like or irregular; it is hard, painless, with even surface, seldom with elevations or excavations; it has no areola, but a smooth, glistening base with thin secretion. On the skin the chancre may appear as an erosion. Contact ulcers around the corona are hard. The sclerosis may resemble herpes, but there is seldom itching. An herpetiform chancre becomes indurated in about sixteen days, while simple herpes lasts but three or four days; herpes may be the atrium for syphilitic inoculation. If the sclerosis occur on a mucous surface there is little elevation; maceration and bleeding readily occur. (*a*) The *genital sclerosis in men* occurs on the urethral orifice; in the fossa navicularis, possibly producing some stenosis or ulceration, with a thin, "meat juice" secretion; on the glans, often as an erosion, usually not extensive, and difficult to diagnosticate because of the hardness of the glans; on the corona, circular and hard; on the (broken) frenulum or in the sulcus coronarius, usually with ulceration and much induration, giving the "split pea" appearance, one-half lying on the glans and the other half on the prepuce; on the prepuce, often with phimosis; or on the dorsum. (*b*) The *genital sclerosis in women* occurs mostly on the labia; on the labia majora, as large, readily ulcerating contact ulcers with inflammatory œdema; on the labia minora as small, hard and circumscribed nodes; on the prepuce as hard fissures; in the urethral orifice with widening and induration which is felt from the vagina; on the posterior commissure, as erosions, which are especially prone to infect during coitus; on the hymen relics, as fissures, or large hard ulcers reaching into the vagina; rarely in the vagina, because of its paucity in glands and its thick epithelium, although chancre may develop on gonorrhœal erosions; in the vaginal portion of the cervix, usually in women who have been pregnant, in which location it produces chancres on the male meatus and is difficult to differentiate from simple and gonorrhœal erosions. (*c*) The *perigenital sclerosis* occurs on the scrotum, mons veneris or inner thigh. (*d*) *Extragenital scleroses* (3 to 6 per cent. of chancres) are especially dangerous in midwives, physicians and wet-nurses. They occur on the scalp, conjunctiva, nose, eyelids, lips, tongue and tonsils (from sexual perversion), cheeks, fingers, arms and mammæ. In rare cases of accidental syphilis the chancre may not be found. Among 9,058 extragenital chancres, 1,863 occurred from vaccination, 745 from cupping or phlebotomy, 179 from circumcision and 82 from tattooing (Bulkley). Fenger demonstrated that sweat and tears are infectious, if enough is inoculated.

The primary sclerosis is always inoculated from an individual with a chancre or secondary syphilis, and, in very rare cases, from tertiary syphilis; Williams (1906) reports 41 infections from gummata. *When the sclerosis is recognized, the blood is already infected.*

Histology of the Sclerosis.—The round cells increase the vessel-walls ten- or twentyfold, and the vessel lumen is narrowed from external pressure and endothelial proliferation, even to obliteration, when retrogressive ulceration ensues. The lymph vessels and glands are involved later. Epithelioid and giant cells, and proliferation of the connective tissue in and about the vessels, are noted. The spirochæte pallida is invariably present.

Course.—Retrogression usually occurs and induration disappears in thirty to ninety days, generally with scar formation. In exceptional cases (1) the sclerosis lasts longer, especially on the corona or prepuce, and relics may be found after years (even 50) due to permanent vascular injury; (2) acute or (3) phagedenic inflammation sometimes intervenes. Chancre and chancroid are separate affections, according to the *dualistic doctrine* developed by Bassereau and Layroyenne in 1852 and Ricord in 1860.

Diagnosis (According to Neumann).

Chancre or Sclerosis—*vs.*—Venereal Ulcer or Chancroid.

Incubation; two to three weeks.	Three days.
Form; indurated erosion, ulcer, nodule.	Pustule, ulcer, etc., without induration.
Number; single in 75 per cent., rarely successively inoculated.	Often multiple at beginning of successively inoculated.
Depth; erosion is superficial, flat, with little elevation, rarely deep excavations.	Through entire cutis or mucosa; "punched-out" appearance.
Border; gradual descent.	Abrupt, sharp, indented, undermined.
Surface; red, livid, sometimes pseudomembranous, thick, yellow-white covering or crusts.	White, gray.
Induration; marked, cartilaginous, sharply outlined, movable, sometimes thin, lasting weeks, perhaps months.	No basal induration; not sharply outlined; merges gradually into adjacent tissue; short duration.
Secretion: scanty, serous, autoinoculation difficult; *spirochætes.*	Rich, purulent, autoinoculation easy.
Pain; little.	Much.
Destruction; rarely phagedenic (and then circumscribed).	More often phagedenic; diffuse.
Re-infection; extremely rare.	Frequent.
Lymph-vessels; inflamed frequently, hard and wide.	Painful; cylindrical.
Lymph-glands; regional, indolent, swelling hard, painless, movable; rarely pus formation.	Often absent or acute inflammatory swelling; often pus formation, capable of autoinoculation.
Nature of the disease: constitutional symptoms follow in eight weeks.	A *local infection*, or involving at most contiguous lymph-glands.

Mixed infection may occur, whence the now abandoned idea of the unity of all venereal ulcers. The real practical lesson from this doctrine is *diagnostic reservation* for some time in doubtful cases.

Lymphangitis syphilitica is marked in 70 per cent. of cases. The lymph vessels may become as thick as a lead pencil, bulging at the site of the valves ("bubonulus"); they become hard, are usually painless, reddened, and are narrowed in their lumen from endothelial proliferation and from pressure inward of their infiltrated walls. The inflammation is best seen on the dorsum penis, extending toward the symphysis pubis; it is usually not over 6 to 8 centimeters in length, but may reach the glands. Resorption occurs in three to eight months. Lymphangitic œdema is particularly suggestive in women when the initial lesion cannot be found. *Local lymphadenitis* (indolent buboes) is present in 97.3 per cent. of cases, and appears in three weeks if there be much, or in four weeks if little secretion from the chancre; and in six weeks reaches the contralateral inguinal, the iliac and lumbar groups, and in seven weeks, the cubital glands. Just before the secondary eruption, the other glands are involved.

2. Secondary Stage.—Secondary symptoms usually appear toward the end of the eighth week (sixth to twelfth week). (*a*) *General infection* occurs. *Fever* is spoken of by the oldest writers, but is less frequent at the present time (20 to 33 per cent.). It is far less common in primary or tertiary than in secondary syphilis ("fever of syphilitic invasion"). It often just precedes the eruption, and mostly in the pustular variety. It may be continuous, remittent or intermittent. Solution by crisis has been observed by Gunz. Fournier described a typhoidal type. Janeway reports instances resembling tuberculosis pulmonalis. It may occur with chills, suggesting malaria. Other toxæmic symptoms are *anæmia, headache, nocturnal bone-aches,* vasomotor symptoms, psychical depression, vertigo, neuralgias, palpitation, night-sweats, polydipsia, digestive disorders, synovitis, increased reflexes and icterus; swollen tonsils, spleen or liver, and albuminuria may also occur as prodromata to the exanthem.

(*b*) The *exanthem* is *polymorphous,* sharply marked, frequently grouped in a circular arrangement and is without pain or itching; it is highly infective, is not reddish as in other eruptions but *copper-colored,* a fact recognized in the fifth century; it appears where other eruptions are rare, as on the forehead or nasolabial fold; crusts may develop on an infiltrated basis. It is recurrent and responds to antisyphilitic treatment. The most frequent form is the *macule* or roseola, next the papular syphilide, and then forms resembling pustules, varicella, pemphigus or impetigo. In the folds or damp parts of the skin the so-called *mucous patches* appear, as at the angles of the mouth, the groin or perineum.

(*c*) The *enanthem* on the mucous membranes is observed on the tongue, lips and cheeks (*mucous patches*), and is often accompanied by pharyngitis.

(*d*) The *lymphatic enlargement* becomes general, although it may

remain localized. Syphilitic "rheumatism," bone-aches, iritis, etc., are treated under special symptomatology.

Late Secondary Symptoms occurred after two to thirty years in 5.8 per cent. of Fournier's 19,000 cases; they are best designated "recurrent syphilis."

3. The Tertiary Stage.—This is characterized by the *gumma* which occurs in many tissues. It is not known what determines tertiary development; in 2,396 tertiary lesions, 78 per cent. of the cases were not treated at all, 19 per cent. moderately and only 3 per cent. well treated (Fournier). The specific symptomatology of gumma will be described below. The gumma may be microscopic, or an inch or more in diameter. On section it is usually hard, grayish-yellow, homogeneous, centrally caseous and peripherally fibrous. Baümler first remarked that tertiary syphilis, particularly of the viscera, may be attended by fever. In 20 to 40 per cent. of cases of tertiary syphilis, no evidence of earlier lesions can be found, which, the author believes, explains many instances of the so-called *mendacia syphilitica.*

Special Symptomatology of Syphilis.—1. The Skin.—The *macular* (*roseolous*) *syphilide* is *the most frequent secondary eruption.* It is hyperæmic at first and later the spots show some infiltration; it develops rapidly, lasts eight to ten days, varies in size from that of a lentil to that of a dime, is at first red, then livid, and pressure leaves it yellow. It comes out more clearly by chilling the skin, and sometimes after administration of mercury. It occurs mostly on the trunk. The face, hands and feet are usually free except in severe cases. It is symmetrical, appears mostly on the flexor surfaces, is circumscribed, recurs more frequently than does any other variety and is prognostically favorable. It is recognized with difficulty when sweat or other secretions cause maceration. On the scalp focal alopecia results. Annular or gyrate forms may occur. Some pigmentation is left.

The *papular syphilide* presents various forms: (*a*) The small miliary papules (lichen syphiliticus), usually develops on the forehead, chin, nose, shoulders, buttocks, back and flexor surfaces of the extremities and usually corresponds to the glands of the skin. Irregularly distributed, it may appear in rings or circles. At first red, it rapidly becomes brownish-red. Fine scales or crusts may form, which, on falling, leave a lacquered appearance. Recurrences are infrequent. It must be differentiated from psoriasis, the development of which on the scalp, ear and extensor surfaces is not observed in syphilis, where the scales are smaller, thinner and less glistening; the hemorrhage on their removal is parenchymatous and not punctate; the glands are swollen; and the hair thinned. On the palms of the hands and soles of the feet differentiation may be difficult, but in syphilis the centre of the papule is likely to be sunken. (*b*) The large papular syphilide is often associated with fever, constitutional disturbance or synovitis, and may occur on all parts of the body, as

the *corona veneris* on the upper forehead, around the nose or in the furrows of the chin and neck. Though infrequent on the palm, it is often seen on the dorsum of the hand. This variety occurs most frequently at the junction of the skin and mucosa and its polymorphism is pronounced, as psoriasis palmaris, rhagades, onychia or verrucose forms. (*c*) The *condyloma latum* or mucous patch is the papule altered by secretions or excretions, and is found about the genito-anal regions, axillæ, etc. *It is the most important syphilide in the propagation of syphilis* by its occurrence on the lips, breast and genitalia; it is flat, elevated, dirty, ill-smelling, and secretes a highly infective secretion. (*d*) The *pustular* syphilide includes the acne form, the varioloid, varicelliform and impetiginous forms; a brown pigmented scar usually results.

The broad *general characters* of *secondary eruptions* are: (i) Their circular form; (ii) tendency to circular groupings; (iii) slow evolution; (iv) symmetry of distribution; (v) polymorphism of the eruption or of its successive crops; (vi) induration; (vii) copper-color; (viii) diffuseness; and (ix) their usual resolution.

Gummata of the skin are asymmetrically disposed; are cutaneous or subcutaneous, involving deeper structures; are non-infective compared with the secondary eruptions; are prone to develop at the site of secondary involvements; occur in the skin more than in all other localities combined; follow incomplete mercurial treatment; are promoted by vicious habits and constitutional diseases; and are seen in 6 to 13 per cent. of cases, most frequently in the third year after infection. Small gummata may resorb by fatty degeneration, but the larger ones caseate, ulcerate, suffer secondary infection and leave a kidney-shaped scar with deformation. Gummata are differentiated from malignant ulcerations by the absence of adenopathy, by their slow growth, painlessness and multiplicity, and by the results of therapy. In gumma, ulceration is more rapid than in lupus; it is deeper, less sharply marked, redder and less symmetrical; recurrence after cicatrization is less frequent; the scars are less flat and smooth, and the bacteriological findings of lupus are absent.

Leukoderma syphiliticum is observed in recent, but rarely in late syphilis—*e. g.*, after macular or lenticular syphilides—as white spots with peripheral brown pigmentation, appearing mostly on the face and neck. Leukoderma usually disappears in from two to twelve years, is rarely seen in either extreme of life and occurs in 4 per cent. of syphilitic men and 45 per cent. of syphilitic women (Neisser).

The *hair* falls out except from the crown of the head, most frequently during the secondary stage, and is proportionate to the intensity of the eruption. It is greater in poorly nourished cases, and its regrowth is usually good. In the bald areas, there are no broken hairs.

The *nails* are sometimes affected. *Onychia syphilitica* occurs particularly in women, in whom it may be the only secondary symptom. The nails may hypertrophy, fall out or fail to grow. Peri- or paro-

nychia rarely occurs without other specific signs and rarely causes pain, ulceration or suppuration.

2. Lymph Glands.—The inguinal glands are first enlarged because they are nearest to the most common genital chancre. Some glands are more involved than others, yet adenopathy of any particular group, as the epitrochlear, is less pathognomonic than suggestive, of secondary syphilis. The posterior cervical glands are involved more than the anterior. Next in frequency are the axillary, epitrochlear and those along the pectoralis. It is well to recall Dietrich's observation that 99 per cent. of healthy individuals have palpable glands. Inflammation and pain are rare, and suppuration occurs in but 3 per cent.; these are due to secondary infection. In extragenital sclerosis, other glands are enlarged first,—*e. g.,* the anterior cervical, from chancre of the lip. The glands are freely movable; may remain swollen for years; and are not usually larger than a hazelnut, but occasionally become massive from trauma, scrofula or gumma formation, and then may resemble Hodgkin's disease.

3. Gastro-intestinal Tract.—On mucous membranes the eruption (enanthem) is not necessarily characteristic, is less frequent and is less polymorphous than the exanthem, with which it occurs synchronously.

Mouth.—This is *more involved than any other part except the skin,* and may precede the skin eruption. Recurrence is very frequent. (*a*) *Erythema* may occur as diffuse or circumscribed dark red maculæ, especially on the cheek, pharynx, uvula, palate and swollen tonsils—the *acute syphilitic angina;* it ceases sharply at the border of the soft and hard palate, and it often escapes recognition. Desquamation, infiltration or fissures, with a slightly granulated and vesicular appearance, may develop. Erosions may last long in users of tobacco and of alcohol.

(*b*) *Mucous patches,* which are papules modified by moisture, develop with the exanthem, and mostly on the tonsils, cheeks and lips. They are single or multiple, disseminated, elliptical or irregular, flat or slightly elevated patches which are circumscribed and of variable color, white, milky or red. On the lips they may measure 6 to 17 millimeters. The course is usually chronic; the patches swell, become granular, ecchymotic and often ulcerated. On the tonsils they may simulate diphtheritic patches, and in smokers a cure cannot be effected until the habit is stopped. Cauterization leaves scars. They are the *most important syphilide because so highly contagious.*

(*c*) *Chronic angina* is of a light red, granular appearance, and is accompanied by hyperæmia, some œdema and erosion, a gray coating on the swollen tonsils, and the general appearance of pharyngitis granulosa.

(*d*) *Pachydermia, ichthyosis* and *leukoplakia* occur on the mucosa of the cheeks, and on the angles of the mouth, lips and tongue, and are not always syphilitic. They are swellings with fissures, are gray in color, irregular, they vary in shape and size and are thick

and scar-like. They occur especially in users of tobacco and alcohol. They may become carcinomatous. Leukoplakia in tobacco users usually occurs on the tongue, or under the lip or palate, while in syphilis it is mostly on the cheek. Psoriasis linguæ is usually central, with round or elliptical, bright red, flat spots, with thread-like coating; fissures are frequent on the edges of the tongue and result especially from the use of tobacco and from bad teeth.

(*e*) *Gummata* of the oral cavity are tertiary. They appear on the lips, when the face is involved. They are rare and without glandular swelling, and occur chiefly in smokers. Gummata of the tongue are most frequent, occurring (*a*) as a diffuse glossitis or infiltration with a later cirrhotic shrinkage or (*b*) as circumscribed gummata, with nodes or ulceration, and without pain or glandular involvement. Gummata of the palate or pharynx are mostly multiple, and ulceration may reach the nose, spine, base of the brain, etc. They are dangerous because they are frequently diffuse, deep or phagedenic; they destroy the uvula, produce adhesions, obliterate the posterior nares or Eustachian tube, stenose the pharynx or draw the tongue back, thus stenosing the respiratory passage. In the tonsils they are very deep and may erode the carotid or palatine artery. Differentiation is required from tuberculosis and epithelioma, in which the glands are usually but not always involved. The diagnosis *ex juvantibus* is important.

Œsophagus.—*Strictures* result from deep gummata, ulcerations in the larynx, or extension from the bronchial glands. Differentiation is required from tumors of the mediastinum, aneurysm, carcinoma, round ulcer and nervous dysphagia. Diverticula may result.

Stomach.—(*a*) *Acute catarrhal gastritis* in the secondary stage is usually toxæmic,—rarely organic. (*b*) *Chronic gastritis* is more common, in part explaining the anæmia and malnutrition; it differs from the vulgar gastritis only in the influence of treatment. Amyloid and glandular wasting are infrequent. (*c*) *Ulcers* of the stomach may result from syphilitic arteritis; Rosanow and Lafleur describe convincing instances. Flexner has collected 16 definite cases. (*d*) *Gummata* are rare. They begin in the submucosa, are usually multiple, leave radiate scars and differ from the ordinary round ulcer in that they are not funnel-shaped, have undermined edges and are broader at the base. (*e*) *Hemorrhages* from the stomach are most rare and Hayem's case is almost unique. They may also result from cardiac, splenic, hepatic or renal lesions.

Intestines.—Except for the rectal involvement, little is known of intestinal syphilis in the adult. (*a*) *Acute catarrhal enteritis* may *induce icterus;* it resists treatment other than specific. (*b*) *Chronic enteritis* in the syphilitic new-born, is manifested by the viscid meconium and disappears with antisyphilitic treatment. Obstinate diarrhœa and stenosis may follow. (*c*) *Ulcerations* may result from enteritis in secondary syphilis, from gummatous and diphtheroid changes in the tertiary, and from amyloid disease in the tertiary or

quaternary stages. Meschede collected 54 cases of ulcer in the small intestines. They occur around the axis of the intestine and are characterized by productive inflammation which prevents perforation but leads to stenosis. (*d*) Intestinal *amyloid disease* is associated with amyloid liver, spleen and kidneys. The gut is pale, smooth, waxy, thick and rigid and there are obstinate diarrhœa, stinking stools and hydrops. It is most frequent in the small intestine and is essentially a vascular change. Amyloidosis in general occurs in 67 per cent. of tuberculosis and in 21 per cent. of syphilis (Hoffmann).

Rectum.—Ulceration occurs in papules near the anus, or results from fissures (Johnson). Rectal disease is usually secondary by contiguity. (*a*) Irritative *proctitis* is most frequently chronic, attended by evacuations of pus, by itching, tenesmus and external excoriations and by a long course. (*b*) *Syphilitic ulcers* of the rectum are tertiary. They occur high up, extend to the colon and often produce stenosis. Stricture without previous ulceration is rare. Perforation is rare. The periproctic tissues are invaded. Fistulæ may develop, but the symptoms are less marked than in cancer or tuberculosis. The fæces are covered with mucus and blood. Diarrhœa from catarrh of the colon, myositis of the sphincter with involuntary evacuations, burrowing of pus and sepsis necessitate at best a long course. Tuberculous ulcers rarely occur in the rectum alone, and are marked by the tubercle bacillus. In dysentery the involvement is higher up, colic is more frequent, diarrhœa is more profuse, and the resulting stenosis is higher in the intestine. In carcinoma, age, cachexia and local appearances are suggestive but not positive criteria. Stricture is lower in syphilis than in carcinoma, and adhesions are more commonly carcinomatous.

Pancreas.—Acute syphilitic pancreatitis was noted by Rokitansky. The interstitial inflammation with sclerosis causing parenchymatous atrophy is rarer in acquired (22 per cent.,) than in hereditary syphilis. Only 3 instances of gumma are recorded. The *peritoneum* sometimes participates in syphilitic disease of the organs which it envelops, *e. g.,* perisplenitis, perihepatitis, perinephritis or perityphlitis; it is extremely rare as an independent disease; Lancereaux, Aufrecht and Laurenzi have observed gummatous peritonitis.

4. Spleen.—(*a*) *Acute splenic tumor* (Walter, 1821) occurs in 31 per cent. of acquired and 61 per cent. of congenital syphilis. (*b*) *Interstitial splenitis* occurs chiefly with syphilitic liver. The organ is increased in size, hard, tender and painful, from perisplenitis. (*c*) *Gummatous splenitis* is very rare. (*d*) *Amyloidosis* occurs very frequently as the "sago" spleen, which is not necessarily very much enlarged; or as the diffuse amyloid spleen, which causes considerable swelling.

5. Liver.—Syphilis of the liver occurs in two forms, interstitial and gummatous, which are often associated with perihepatitis, amyloid or fatty change, and sometimes with parenchymatous changes

as acute yellow atrophy. (*a*) *Interstitial hepatitis* (syphilitic induration, syphilitic cirrhosis) is more frequent in congenital (in 65 per cent.) than in acquired syphilis. The volume of the liver is increased in children, and sometimes decreased in adults. The surface is usually uneven, with furrows or nodules; the edge is thin, indented and fairly sharp, whereas in other cirrhoses it is rounded. Perihepatitic adhesions to the colon or abdominal wall are frequent, and clinically evidenced by lessened respiratory excursion. The connective tissue forms in Glisson's capsule, follows the branches of the portal vein into the liver (peripylephlebitis), and shows as bands of grayish tissue, the protruding granules representing normal tissue. The liver cells are degenerated, even destroyed by the connective tissue or by obliterating endarteritis of the small vessels. The atrophy is most marked in the anterior border of the liver and in the left lobe. The connective tissue is not only perilobular, but reaches into the lobules more than it does in alcoholic cirrhosis. In acquired syphilis the liver is not equally nor completely diseased, whence protrusions of the sound tissue are noted. New bloodvessels from the hepatic artery develop in the connective tissue and thrombosis may occur in the portal vein and its radicles (pylephlebitis).

Symptoms.—The clinical development is latent or insidious,—*e. g.*, without pain, ascites or œdema. The abdominal veins may be distended; the urine is often dark, possibly icteric and often albuminuric. The concomitant splenic tumor results from hyperplasia, stasis or amyloid degeneration. Ascites is less frequent and develops late and gradually, herein differing from alcoholic cirrhosis. Icterus develops in 33 per cent. More bile is present in the stools than in ordinary cirrhosis. Gastro-intestinal dyspepsia and vomiting of blood are not infrequent. This form develops more often in men than in women; is promoted by alcoholism; has a chronic course; and its prognosis is rather better than in non-syphilitic cirrhosis, since relative recovery is possible. When the liver is small, recovery is impossible and death results from intercurrent pneumonia, pleurisy, cholæmia or hemorrhagic diathesis.

(*b*) *Gummatous hepatitis* (syphilitic hepatitis *par excellence*) is more frequent than the first form, and occurs oftener in acquired than in congenital syphilis. The gummata are either miliary or large nodes which may fill the abdomen. The nodes vary from one to over fifty, and may be superficial or deep. The surface of the liver is smooth or warty, thickened, or adherent to the diaphragm or colon. The liver is distorted, mammillated or *lobulated* (*hepar lobatum*). Deep, irregular, radiating *furrows* are frequent, resulting from absorbed gummata, affecting either lobe and occurring mostly on the anterior surface near the suspensory ligament (Virchow) or on the edge. In congenital syphilis gummata are often observed at the hilum, in the wall of the portal vein or near the bile vessels.

Symptoms.—Gummata are frequently latent—*i. e.*, are discovered only at postmortem. Considerable *pain* occurs in the shoulder, as well as

pain on motion or tenderness on pressure in the epigastrium or hypochondrium. Pain is due mostly to perihepatitis; a friction rub is heard when the inflammation is fresh; adhesions and loss of respiratory excursion result, in older cases. Icterus is infrequent and results from accidental gummatous compression of the bile ducts, from perihepatitis or from cicatrices. When icterus exists the affection is often mistaken for cancer. The liver usually shows *furrows* and *nodules*. *Early enlargement* is more frequent than in alcoholic cirrhosis but a final shrinkage has been observed. Ascites is rare except from syphilitic cirrhosis, amyloid liver or cardiac insufficiency. When present, ascites is usually terminal, independent of the size of the liver and often associated with albuminuria. Splenic tumor is infrequent, except from gummata or amyloid degeneration in the spleen, from coincident liver cirrhosis or gummata at the *porta hepatis*. Dyspeptic disturbances, bronzed skin, hemorrhages and inflammations of the serous membranes infrequently complicate liver gummata.

Diagnosis.—This depends upon other evidences of syphilis, and while sometimes easy, it is often difficult or impossible. All forms of syphilis of the liver may remain stationary for a time (Bamberger), and in general a *longer course* is expected than in alcoholic cirrhosis. Oppolzer and Bachdalek mistook gummata for cancer, and the reported cases of cure of *cancer* by the earlier writers are clearly confusions with syphilis; age is only a relative criterion, yet most cases of syphilis of the liver occur under the fortieth year; ascites, icterus, enlarged liver and cachexia may occur in both diseases, occasioning doubt and confusion. The nodules of syphilis are usually small but may be larger than those of cancer. Rapid growth is an indication of cancer; more constant size, of syphilis. Albuminuria and splenic tumor are more common in syphilitic liver. The best clinicians have confused syphilis with cancer, and the safest rule is always to think of syphilis when cancer seems the obvious diagnosis. Coincident amyloid disease renders the diagnosis difficult. The lungs are compressed in cancer more than they are in syphilis. The smooth edge of the liver, signs of fresh hepatitis, and lack of respiratory excursion, indicate syphilis (Lancereaux and Riedel). Riedel found similar symptoms in syphilis and *gall-stones*, such as pain, fever and tenderness. Fever is not uncommon in hepatic syphilis, which may then be easily confused with *liver abscess* (*q. v.*). Final differentiation may be made *ex juvantibus*. (See DIFFERENTIAL TABLE OF DISEASES OF THE LIVER.)

Prognosis.—This is more favorable than in the vulgar cirrhosis, for the disease lasts several years. Spontaneous recovery from gummata may result without specific treatment. Rupture into the peritoneal cavity is rare. Death from cachexia and amyloidosis may occur.

Amyloid liver (*q. v.*) is most frequent in the tertiary or quaternary stage of acquired syphilis. Amyloidosis rarely occurs in the liver alone.

Syphilitic *pylethrombosis* (see PYLEPHLEBITIS).

Icterus, especially investigated by French writers, has been attributed to swelling of the portal lymphatics, early hepatitis, cholangitis, injury to the liver cells or to gastroduodenal catarrh.

6. KIDNEYS.—(*a*) *Albuminuria* occurs in recent syphilis or in the later ulceration. It is usually transitory and it is thought to result from toxæmia or vascular changes. Its prognosis is usually good, since nephritis rarely follows it, but the later in the disease that albuminuria appears, the poorer is the prognosis. Mercury in moderate dosage does not cause it though energetic mercurialization may produce albuminuria, cylindruria and even calcification of the convoluted tubules. (*b*) Paroxysmal *hæmoglobinuria* occurs relatively often after mercurial therapy. The prognosis is better than in non-syphilitic cases. (*c*) *Acute parenchymatous nephritis* is focal and cortical. It occurs with the eruption, is toxæmic and is rare. When it develops late, from ulceration, the prognosis is unfavorable. There may be no symptoms; anæmia, vomiting or other symptoms of acute nephritis may be present, but uræmia is rare, since it is a focal disease (Bamberger). (*d*) *Chronic parenchymatous nephritis* may follow syphilis. Its relation and its percentage are difficult to estimate (0.5 per cent.). (*e*) *Chronic interstitial nephritis* differs from the ordinary form in its disseminated focal involvement, which leaves scars. Polyuria is rarely great, casts and albumin are present in greater amount, and the course is more rapid than the ordinary course of three to seven years, because of marantic, amyloid and other visceral changes. It often occurs at an early age when arteriosclerosis is rare. There is said to be no cardiac hypertrophy. Bamberger found a history of syphilis in 3 per cent. of cases of ordinary contracted kidney. (*f*) *Gummata* are found in the miliary or large forms in the cortex and sometimes in the medulla, usually multiple and generally unilateral. The renal tissue is partly destroyed. They coëxist with liver gummata, amyloid kidney and chronic parenchymatous nephritis. The symptoms are indeterminate. (*g*) *Amyloid kidney, q. v.,* under diseases of the kidney. (*h*) According to Frerichs, Cantani and others, syphilis is an etiological factor in *glycosuria.* The *suprarenals* are rarely involved, except in congenital syphilis; swelling, hyperæmia, fatty degeneration, gummata, interstitial inflammation and amyloid changes in the Malpighian bodies have been recorded. Gummata have been observed in Addison's disease.

7. CIRCULATION.—HEART. The heart was found involved but six times in 100,000 autopsies (Mrâcek).

(*a*) *Syphilitic myocarditis is coronary arteritis;* foci of interstitial involvement occur, as infiltration along the small vessels, especially in congenital syphilis, into the intermuscular septa. Microscopically, the general picture of myocarditis prevails. It involves the front or apex of the left ventricle, or the intermuscular septum. In congenital syphilis it occurs in the right more than in the left heart. Re-

sulting changes are cardiac hypertrophy, aneurysm of the heart and Dittrich's true heart stenosis (conus stenosis). *Gummatous myocarditis* is usually observed with a diffuse myocarditis. Only 97 (62 perfectly clear) cases of myocardial gummata are recorded (Stockmann, Goldfrank). Gummata may attain great dimensions in the interventricular septum. The disease is often latent until, in 50 per cent. of the cases *sudden death* results. Most fatalities occur in persons between thirty and forty years of age. The heart action may be incompetent or irregular, and there may be dyspnœa, asthma, angina pectoris or cerebral embolism. Hydrops is rare. Amyloid of the heart is occasionally associated with gummatous myocarditis. Heart rupture is recorded.

(*b*) Independent syphilitic *pericarditis* is unknown; it is invariably a complication of myocardial syphilis, occurring over the large vessels or anterior surface of the heart, as serofibrinous pericarditis or complete concretion. Gummatous pericarditis is always combined with the serofibrinous form, and sometimes with aneurysms of the coronary arteries. The symptoms are those of disordered compensation or of myocarditis, its invariable associate.

(*c*) *Syphilitic endocarditis* rarely develops. The nodules on the valves rarely retract or cause valvular disease. Mural plaques complicate myocardial gummata. Endocardial gummata may occur.

Bloodvessels.—Endarteritis syphilitica. Syphilitic arterial disease, known to Morgagni and Paré, causes wide-spread, often irreparable or fatal complications. (*a*) In the *fibrous* form, the wall is thick and opaque, the intima and elastic tissue are hyperplastic and the lumen is eccentric. When the muscular coat is not involved, thromboses occur; when both the muscular and elastic coats are diseased, aneurysms result; this type is known as the *obliterating endarteritis* of Heubner (see SYPHILIS OF THE BRAIN). (*b*) Gummatous arteritis or periarteritis is very rare. (*c*) General arteriosclerosis involves the vessels more widely than does the syphilitic arteritis which is largely localized in the aorta and brain-vessels and exceptionally in the other vessels. Coronary involvement is held by some writers to be more frequent than brain involvement. It may begin in the first year after infection.

Aorta.—Syphilitic aortitis (Heiberg, 1877) is oftenest seen in the ascending aorta, its arch and thoracic segment, and the lower part of the abdominal aorta. It begins in the adventitia or intima and ultimately involves all the walls in varying degrees. The early stages show gray, transparent, gelatinous patches of productive cellular inflammation and spirochætes in the intima; older foci are gray-white, round or oval, elevated and stellate plaques. The process sometimes becomes deep and calcification occurs in the intima with thrombosis. It may extend down to the aortic valves. Its origin may be (*a*) in the intima, (*b*) in the adventitia and media together (mesaortitis) or (*c*) in the intima and adventitia (vasa vasorum). The resulting loss of elasticity and resistance promotes aneurysm. Welsh held that

66 per cent. of aneurysms were syphilitic; personally, the author believes 80 to 90 per cent. is a more accurate estimation. Syphilitic aneurysms develop more rapidly than those due to other causes. They may be single or multiple and usually occur in the arch of the aorta, and sometimes also in the abdominal aorta or its branches (see ANEURYSM). Straub found this aortitis in 98 per cent. of cases of paretic dementia, and Chiari in 50 per cent. of syphilitics. Benda describes gummata, of which only six cases are on record.

Pulmonary Artery.—Valvular gumma is very rare, as are occlusions of the pulmonary trunk or its compression by scars. According to Neumann, syphilitic dilatation or aneurysm of the pulmonary artery has not yet been observed.

Vein Syphilis.—Thirty-three cases of phlebitis were collected by Roussy in 1903. Schuppel has described a periphlebitis originating in the adventitia and seen especially in hereditary syphilis. Gummata exist in the veins or involve them secondarily.

BLOOD.—Oligocythæmia and oligochromæmia occur early and most markedly when the glands are widely involved. The anæmia disappears under administration of mercury, to reappear if too much be given. A count as low as 1,700,000 may be observed. The hæmoglobin is reduced 15 to 30 per cent. and the loss may exceed that of the red cells. The "Justus' test" is based on a 10 to 20 per cent. reduction in the hæmoglobin after a large inunction or injection of mercury, in the florid secondary stage; a positive test is only suggestive of syphilis—not pathognomonic. The white cells are slightly increased, especially the lymphocytes; the eosinophiles, as in other skin diseases, are increased but not constantly. Myelocytosis may develop in the tertiary stage. The blood contains the spirochætes and by way of the blood syphilis becomes a general disease. Pernicious anæmia and leukæmia are rare sequels.

8. RESPIRATORY TRACT.—*Nose.* (*a*) An *initial nasal lesion* is most rare, only 95 cases being recorded. (*b*) *Acute rhinitis* is much rarer in adults than in the new-born. It is characterized by its persistence, erythematous papular foci, hemorrhagic erosions and by fetor. (*c*) *Rhinitis atrophicans* is the result of hyperplastic rhinitis. (*d*) *Gummata* usually develop late, though they have been seen in the seventh month (Mauriac); they begin in the floor and septum of the nose, the periosteum, bone and secondarily in the cartilage. They occur with diffuse necrosis and involve the cartilaginous and membranous septum and cartilages of the alæ; sequestra may be blown from the nose. Septal perforation may develop and a long course is usual, with discharge of pus and crusts; the bone is often denuded. The deformed *"saddle-back" nose* is due to loss of the triangular cartilage and vomer, and sometimes to simple atrophy. Differentiation from noma, tuberculosis, carcinoma and the ordinary perforating ulcer of Zuckerkandl (beginning as a hemorrhage and due to streptococcic and staphylococcic infection) must be considered. The diagnosis *ex juvantibus* is the best test.

Larynx.—(*a*) *Catarrhal laryngitis* occurs with the eruption. The larynx is red and swollen, with a secretion, at first thin and later purulent. Coughing and hoarseness are common. (*b*) In *papular laryngitis* the papules often ulcerate and are associated with diffuse infiltration and nodes. The epiglottis is also involved. Deforming ulcers are neither acute nor inflammatory. Stenosis of the glottis results if ulcers invade the submucous tissue. (*c*) *Gummata* of the larynx are usually multiple, and vary from the miliary size to that of a pigeon's egg; sometimes a diffuse infiltration occurs. They may soften, ulcerate and produce dysphonia, dysphagia, dyspnœa, hemorrhage or suffocation. They always leave adhesions, *stenosis,* scars or deformity.

Diagnosis.—In cancer the glands are usually enlarged, the growth is harder than in syphilis and the microscopic examination may be of value. Syphilis and tuberculosis may occur together. Though the round form of syphilitic ulcer is not of absolute diagnostic value, it usually begins near the epiglottis, is rounder and larger and has sharper edges, a more reddened border, and a whitish-yellow coating of its deeper base, while the tuberculous ulcer is less red, less infiltrated, less deep and less insidious in its development.

(*d*) *Perichondritis* may affect any cartilage. The cartilage is discolored, excoriated and surrounded by œdema; there may be submucous necrosis, or difficulty in respiration and deglutition. The prognosis is always grave. The lower the necrosis develops, the worse is the prognosis, for deformity and stenosis occur. Syphilis causes 13 per cent. of cases of glottis œdema; tuberculosis, 9 per cent.

(*e*) *Syphilitic vegetations,* due to irritating secretions, are sessile or pedunculated and cause dysphonia or even suffocation.

Trachea.—Only two instances of condylomata have been recognized. The trachea is red, swollen and sometimes ulcerated and distorted. Tracheal stenosis, resulting from healing gummata, is usually angular (Mauriac) and involves mostly the middle third. The symptoms are a burning sense of constriction; inspiratory dyspnœa at night, on exertion or on lying down; a cough resembling that of pertussis, with sanguineo-purulent sputum, which may contain fragments of cartilage; and cyanosis. The diagnosis is made by the laryngoscope.

Bronchi.—(*a*) *Acute bronchitis* is observed, with the secondary symptoms especially in tuberculous, cachectic and alcoholic subjects.

(*b*) *Chronic bronchitis* often accompanies laryngeal, tracheal or bronchial affections. Phthisis may be suggested. The breathing is disproportionately disturbed by irritation of the vagus from enlarged bronchial glands (Morton). (*c*) *Kidney-shaped ulcers* may cause stenosis and sometimes perforation into the pulmonary artery, mediastinum or œsophagus. L. A. Conner, of New York, collected 128 cases of bronchial and tracheal syphilis; in 56 per cent. the trachea alone was affected; gummata occurred in 15 per cent.; ulcers

in 44 per cent., frequently with severe hemorrhages; cicatrices and stenoses in 40 per cent.; and bronchiectasis in 20, fibrous peritracheitis in 6, and pulmonary syphilis in 7 per cent.

Lungs.—Syphilis of the lungs, recognized by Morton (eighteenth century), may cause symptoms like those of pulmonary tuberculosis. Sims holds that every patient with tuberculous signs should be searched for syphilis.

(*a*) The *white pneumonia* of hereditary syphilis may be described here although it presents no clinical symptoms. The lung is firm, heavy, airless and grayish-white,—called white hepatization by Virchow, the chief change being in the thickened and infiltrated alveolar walls. In the air cells, desquamated and swollen epithelium and spirochætes are found, with scattered miliary foci about the arteries.

(*b*) *Diffuse infiltration* differs from tuberculosis in that it occurs in the centre of the lung, and exceptionally in the apex (Fournier). It usually travels downward rather than upward. In the beginning the lung is red, hard, large, airless and smooth on section and later becomes gray, uneven and nodulated; the alveoli are filled with fibrin, epithelium and blood-cells. Infiltration of the connective tissue occurs about the vessels impinging upon the air-cells. The origin of this infiltration is disputed. It arises from desquamative pneumonia, alveolar connective tissue, the bloodvessels, peribronchial connective tissue, or from the lymph-vessels. Necrosis, fatty change and caseation may result from occlusion of the vessels, which later may form cavities. Disproportionate dyspnœa is present. Temperature may occur with or without ulceration.

(*c*) *Gummatous pneumonia* may occur alone or with diffuse infiltration. Gummata develop largely in the lower lobes near the hilum. Differentiation from tuberculosis is often difficult; the two lesions may occur together. Dulness and other signs of consolidation or cavity formation often result. The sputum is mucous, purulent, profuse, bloody, stinking, and contains masses of tissue but no tubercle bacilli. The course is usually chronic, and advanced cases may heal with appropriate treatment; Brambilla (1777) described a supposedly phthisical patient, who, by mistake, took mercury and recovered. Diagnosis is most difficult if the foci are small. Tuberculosis occurs more often on the left and syphilis, on the right side. Tuberculosis occurs more frequently in the upper, syphilis more often in the lower or middle lobes. Rise of temperature is more frequent in tuberculosis. In syphilis, hemorrhage is rare, as the vessels are obliterated, and night sweats are infrequent. In caseous tuberculous pneumonia, localization is of no diagnostic value.

(*d*) *Syphilitic interstitial pneumonia* occurs with the above affections. The lung may be lobulated (pulmo lobatus) like the lobulated liver; there is marked induration as irregular branching bands, which dilate or distort the bronchi; diagnosis is impossible.

(*e*) Syphilitic disease of the pleura is rare. It may occur with lesions of the lung alone or with pleural gummata.

9. Nervous System.—The changes are partly toxæmic, partly anatomical. Syphilis of the nervous system occurs in men more frequently than in women (8 to 1), mostly between the years of twenty-five and forty, in neuropathic subjects and after trauma; in brain workers or those debilitated by worry, excesses, lead or nicotine; and in children as perhaps the first symptom of parental disease.

The primary lesion often escapes recognition. In 20 per cent. of cases, there is no history of chancre. It occurs where the secondaries have been light and have had ineffectual or no treatment. Syphilis is said to occur in the brain in 16.5 per cent. of syphilitics, more, indeed, than in other viscera, excepting the liver. (Again, it has been held that only 10 to 15 per cent. of syphilitics have visceral, and only 3 per cent. brain, complications.)

Fifty per cent. of brain syphilis occurs within three years after infection. Formerly described as occurring many years after infection, instances are recorded where cerebrospinal syphilis developed even as early as five weeks after infection. Nervous symptoms may occur soon after an infection in those advanced in years (Kuh). Nervous symptoms in the secondary stage as headache, neuralgia, increased reflexes or fleeting paralyses, are probably chiefly toxæmic.

In general, the symptoms are marked by—(*a*) *variability,* undulation and inconsistency, due to the characteristic growth and vessel changes which regress and recur; (*b*) *incompleteness,* as partial paralysis or disturbance of consciousness; (*c*) the symptoms are partly tumor-like, partly vascular, or partly inflammatory; they are partly meningeal, partly basal or cortical, and rarely occur in the centre of the brain except from secondary vessel lesions and gummata. The disease may begin with diffuse neurasthenic, meningeal, psychopathic or focal symptoms. As Wunderlich stated, syphilis produces no symptom not produced by other disease; therefore, no typical picture obtains.

Types.—1. *Syphilitic arterial disease* occurs alone or with the other varieties of brain syphilis. It is the *most frequent form* and, aside from neuritis paralysis, *the most frequent cause of syphilitic paralysis.* There are prodromal disturbances; headache is usual but less constant than in meningeal syphilis; vomiting, vertigo, psychical changes, dementia, convulsions, intermittent hemianopsia and aphasia may occur; choked disk is rare; *thrombosis and obliteration of the vessels* is gradual in onset and is intermittent,—*e. g.,* it may involve the leg and, in a few hours or days, the arm. These changes are due to obliteration of the vessels and multiple softening which (in 95 per cent. of cases) occur in the Arteria fossæ Sylvii; pseudobulbar paralysis may rarely result from involvement of the bulbar vessels.

Course.—The first attacks are mild and short; the later ones are more severe, and occur with marked somnolence, very frequently with bilateral, alternating, or crossed paralysis, with general, unilateral, or rarely Jacksonian convulsions, or with partial or mixed aphasia,

mental changes and progressive bulbar phenomena, such as dysphagia. They result from arterial disease; hemorrhage is most rare except from ruptured cerebral aneurysm.

It may be difficult to diagnosticate primary syphilitic disease from the ordinary *arteriosclerosis;* in over 90 per cent. of cases the latter occurs in late life, its progress is slower, the changes are more disseminated, all coats of the vessels are involved, its fatty and calcareous changes lead more often to widening than to obliteration, psychical changes and convulsions are less frequent and hemorrhage is more characteristic than softening; whereas in *syphilis* the onset is more acute and headache more conspicuous, encephalomalacia occurs at a younger age (see Brain Embolism, Differential Table), the changes affect chiefly the aorta and brain vessels, the intima is principally involved, calcareous and fatty changes are rare and obliteration is usual, and there are characteristic psychical changes as somnolence or apathy; the hemiplegia occurs by "epochs" or "installments," associated with meningitis, dementia or delirium; finally the symptoms are more diffuse than in senile arteriosclerosis. Without treatment, the average duration is one to three months (Oppenheim).

Dieulafoy has described multiple *aneurysms* in the basilar, Sylvian and carotid trunks; they are generally *tertiary,* but may occur eight months after infection. Their rupture produces symptoms of apoplexy or meningeal hemorrhage. Otherwise hemorrhage is rare in brain syphilis. (See illustrations under Aneurysm.)

While *softening is usually ischæmic* and not inflammatory, we sometimes encounter foci of *encephalitis syphilitica* as (*a*) disseminated foci in the brain and cord; (*b*) indurated sclerosis; and very rarely (*c*) softening occurring apart from disease of the vessels.

2. *Basal gummatous meningitis,* the best known type, and, after arterial disease, the most frequent form, begins in the dura, especially along the chiasma opticum, in the interpeduncular spaces or along the cavernous sinus; less frequently in the fossa of Sylvius and the cortex; pathologically it consists of fibrinous exudate, granulation tissue, cicatrices and gummata.

General Symptoms.—Headache is the most important, constant and early symptom (in 75 per cent.). It is paroxysmal, or, if persistent, is increased at night, is sharp, boring or dull, is deep-seated (Fournier) and but rarely circumscribed. Other symptoms are neuralgias, cerebral *projectile vomiting,* vertigo and changes in mentality,—*somnolence,* semi-intoxication, or motiveless activity and loss of the æsthetic sense. Again, the brain may be normal, except during the attacks,—*e. g.,* coma may occur from which spontaneous recovery is possible. Persistent sleep, lasting for weeks or months, is usually ominous. The brain activity may be tardy or excited, while nocturnal automatism (Wood) or dementia, may develop, alternating with delirium, epileptic attacks or paralytic seizures. The brain symptoms may, on rare occasions, resemble those of uræmia, meningitis or typhoid. *Epilepsy* may be typical or unilateral, frequent or

violent. Polyuria or polydipsia (33 per cent. of cases), diabetes insipidus, resulting from disease in the third ventricle, or diabetes mellitus, of which Oppenheim has collected 20 cases, may occur. Fever may be present, but usually accompanies the secondary manifestations.

The *cerebral nerves* are often affected, especially the second and third. Involvement of the nerve branches successively is most characteristic (85 per cent., Uhthoff); *80 to 90 per cent. of cases of nerve involvement results from syphilis of the brain, tabes* or less frequently, from general paralysis and brain tumor. (*a*) The third nerve is involved most frequently—50 to 80 per cent. of the cases, "the sign of syphilis" (Ricord). (*b*) Disease of the optic nerve occurs anatomically in 82 per cent. and clinically in 40 per cent. of cases. Choked disk (10 per cent.) is almost always bilateral. (See Retinal Plates under Miliary Tuberculosis.) Neuritis is unilateral in 4.6 per cent.; simple atrophy, with complete blindness (in 6.6 per cent.), is most frequent in tabes and next most frequent in brain syphilis and general paralysis. Amaurosis is common and often unilateral. Hemianopsia is homonymous. Temporal heteronymous hemianopsia is very often syphilitic while the nasal form is rarely so. In 5 per cent. of cases the field of vision is contracted. The eye changes, due to meningitis, bone disease or gumma are susceptible to treatment. (*c*) The fourth nerve, (*d*) the sixth and (*e*) the fifth nerve are next in frequency of involvement. The fifth is unilaterally affected, and more in its sensory than in its motor branches. The olfactory nerve is rarely involved. The facial nerve may be paralyzed, nearly always unilaterally and peripherally. The eighth nerve is sometimes involved, and the Ménière symptom-complex results, which, according to Fournier, is ominous. The vagus or hypoglossus may be affected.

Together with the above changes *gummata may grow into the pons,* crus or medulla, and may cause hemiplegia and crossed paralyses, as hemiplegia plus oculomotor paralysis (Weber's paralysis); plus facial paralysis (Gubler's paralysis) or plus abducens and trigeminus paralysis (Leyden's paralysis). In basal meningitis, *arterial phenomena are often noted;* in the cortical region they are evidenced by syncope or apoplectiform attacks from sudden variation in the blood pressure; toward the base they produce encephalomalacia, hemiplegia, hemianæsthesia and hemianopsia. These, however, are usually later symptoms than meningitis and neuritis. The course of syphilitic meningitis is typically subacute with remissions and exacerbations, and lasts rarely more than a half a year. To summarize: Headache, convulsions, sopor, coma, delirium and neuritis are followed by irritative and paralytic symptoms with remissions, then with renewal of the old, or advent of new, symptoms.

Differential Diagnosis.—Carcinoma and sarcoma at the base of the brain, involving the meninges, run a continuous progressive course, with definite localization and with few changes in the vessels, while

in syphilis the course is intermittent or remittent, localization varies, and changes in the vessels may be marked. *Tuberculous meningitis* exceptionally may give identical symptoms, but there is less development of connective tissue, vascular changes are rarer, the nerves less frequently involved, the course more acute, febrile and progressive, and remissions less frequent and less marked; stiff neck and general muscular rigidity occur, which are rare except in the congenital type of syphilis. Mental obscurity is more marked and sudden, while it intermits in syphilis. The irritation, in the initial stage, is followed by paralysis later, while paralysis may occur at once in syphilis. Tuberculous meningitis is a disease of the first, second or third decennium, while syphilis usually occurs after twenty years of age. Other syphilitic or tuberculous foci and the results of therapy determine the differentiation. Differentiation from Quincke's *serous meningitis* may be difficult, but the syphilitic variations and the therapy are the final tests. Differentiation must be made from *primary syphilitic neuritis.* It is questionable whether primary involvement of the second and third nerves may occur in syphilis. It is rare at the base of the brain and these cases are generally caused by an anatomical meningitis with but few symptoms. The *multiple nerve root affections* of Kahler, involving the seventh and third cerebral and the cervical and dorsal nerve roots, were observed without symptoms of meningitis or tumor, and caused neuralgia and paralysis. *Optic neuritis* requires differentiation from similar changes due to tumor, alcohol, nephritis, diabetes and acute infections. *Periodic paralysis* of the third nerve is rhythmic in its attacks, usually involves the entire nerve, occurs in young children especially, and with attacks of migraine.

3. *Syphilis of the convexity* may occur as circumscript or diffuse meningitis or meningo-encephalitis, with symptoms like those of cortical tumor or with diffuse manifestations.

General Symptoms.—Headache is usually the first and most common manifestation. *Mental* symptoms may occur independently of the localization. *Focal* symptoms are the *Jacksonian attacks* of epilepsy, which occur with mono- or hemiplegia, develop by starts or "installments" and occur without an aura. *Ninety per cent. of convulsions in those over thirty years of age, not uræmic or alcoholic, are syphilitic* (Fournier). The symptoms are more diffuse than in vulgar epilepsy. The convulsions may even number 400 in twenty-four hours. Pain or paræsthesia occurs but anæsthesia is uncommon. Aphasia is frequent and is usually of the transitory motor type at first, word deafness being relatively rare; aphasia occurs less often from gumma than from vascular disease. Alexia and agraphia have been observed. When the process is diffuse it may be manifested clinically as an acute psychosis which answers only to antisyphilitic therapy; dementia is the most frequent type. Both the diffuse and gummatous forms recover more frequently than do other forms.

4. *Gummata* are the least frequent variety; they occur in any part

of the brain, usually in the meninges, sometimes in the medullary substance, central ganglia, cerebellum, pons or crus. If they occur alone, the symptoms are the same as in brain tumor, except that there is often regression, which is either spontaneous or results from anti-syphilitic treatment. *Cortical gummata,* producing cortical epilepsy and monoplegia, require differentiation from other tumors (*q.v.*) as tubercle or glioma; in *cortical tumor* the headache is duller and deeper; the pressure symptoms, mental depression and slow pulse are more marked; the disk changes follow focal symptoms and the process advances less by epochs; while in syphilis the headache is frequently local and the pressure symptoms are more diffuse and rapidly extensive; the disk, as a rule, is involved only when coincident basal meningitis occurs. Cortical paralysis may develop, and is often associated with Jacksonian epilepsy (*v.s.*). The symptoms are more undulatory, and finally treatment is successful.

5. *Cerebrospinal syphilis* produces no characteristic spinal symptoms; suggestive, however, are (*a*) their asymmetry, (*b*) their less pronounced character, (*c*) their variability, (*d*) meningeal and nerve root symptoms and (*e*) response to mercury and iodides. Syphilis is never a system-disease (Gowers). Syphilis of the cord is five to six times less frequent than brain syphilis.

The most frequent and characteristic form is *cerebrospinal meningo-encephalitis,* observed in both acquired and congenital syphilis. While the symptoms may be strictly spinal, anatomically, cerebral gummatous meningitis or meningo-encephalitis is frequently found. Of the three sets of spinal bloodvessels, the peripheral are most involved and Heubner's arterial findings are not noted in them. Brain changes usually occur first and may mask the spinal symptoms. The meninges are most involved in the cervical and upper dorsal regions, as evidenced by pain, stiffness or girdle sensation; while the cord itself is mostly affected in the lower dorsal region, and therefore involves chiefly the lower extremities, causing weakness in the legs, paræsthesia, sphincter disturbance and decubitus. Intercostal neuralgia, pains in the extremities (often most severe at night), spastic paraparesis, hemiparaplegia, triplegia, Brown-Séquard's syndrome, anterior poliomyelitis or amyotrophic lateral sclerosis, increased (varying) reflexes, involvement of the bladder and rectum, and general or selective sensory disturbances, are some of the symptoms of cerebrospinal syphilis. Erb has laid special stress on the spastic paraplegic type. It is now maintained that the so-called syphilitic myelitis is only softening—*i. e., myelomalacia.* Gummata of the cord are infrequent. Perineuritis gummosa of the sensory or motor nerve roots is uncommon.

Differentiation from combined system-disease is often difficult. Some cases of ascending paralysis have been helped by mercury. In cerebrospinal syphilis and parasyphilitic tabes or dementia, the lymphocytes predominate in the fluid withdrawn by lumbar puncture.

Prognosis of Brain Syphilis.—According to Naunyn, only 48 per

cent. of the cases recover. The later the disease, the worse is the prognosis. The prognosis is more favorable in meningeal (*i. e.,* extracerebral) involvement, when nerve symptoms are dominant, when epilepsy and other symptoms of peripheral brain irritation occur or when early treatment is instituted. Eighty-two to 88 per cent. of cases of syphilis receive insufficient mercurialization (Hjelman). The prognosis is less favorable in specific arterial disease (paralysis from softening), in bulbar forms, in diffuse involvement, in great psychic alteration, and in proportion as the primary and secondary lesions were poorly developed; also in extragenital infections, in hereditary syphilis, and in complicating trauma, tuberculosis or alcoholism. Recovery is rarely complete and recurrence may be observed. The prognosis is relatively poor in spinal lesions.

6. *Syphilis of the peripheral nerves* may cause facial and trigeminal paralysis, even in the secondary stage. The most frequently affected spinal nerves are the occipital and auriculotemporal. Other nerves are infrequently involved. Multiple neuritis is rare.

10. Eye and Ear.—Syphilis comprises 2 to 3 per cent. of all eye diseases and at least 3 per cent. of all syphilitics have eye disease. Any part may be involved, except the lens, which suffers only from arterial disease in the uveal tract. The *uveal tract is most involved;* next in frequency come the retina, optic nerve and eye-muscles. The lids are not often involved; chancre, condyloma and gumma may occur, and are easily confused with chalazion.

Conjunctiva.—Twenty-two scleroses (Guzeit) have been observed, as well as a few gummata; catarrh occurs in the secondary stage.

Cornea.—The cornea is frequently involved: (*a*) As *parenchymatous keratitis,* which is mostly observed in *congenital syphilis,* and occurs even in the third generation, while in acquired syphilis it occurs in but 2 per cent. of cases. *"Hutchinson's triad"* in congenital syphilis consists of *keratitis, notched teeth* and congenital or early acquired *deafness.* The cornea is diffusely clouded and milky, and on close examination shows patches or streaks of inflammation and sometimes new-formed vessels; (*b*) as keratitis parenchymatosa, circumscribed or punctate; (*c*) as gumma, which is infrequent; (*d*) as keratomalacia, which is observed in the congenital variety.

Iris.—Of all inflammations of the iris, 75 (Mauthner) or 90 per cent. (Klein) are syphilitic. The symptoms are those of other varieties of iritis—ciliary injection with discoloration, narrowed pupil, slow reaction and synechia or occlusion of the pupil. It is usually plastic, or exudative with tiny papules (Fuchs); iritis with nodules, if it is not the result of trauma, is strongly indicative of syphilis. Iritis is usually secondary and rarely tertiary. Without cyclitis it is rare in congenital syphilis. As it rarely heals completely, atropine should be administered early while mercury is being given, for expectant treatment results in severe lesions and inflammation tends to recur. *Ciliary* involvement is usually secondary, rarely tertiary and may be the sole symptom of syphilis. Injection and exquisite tenderness to touch are characteristic. Gummata usually heal.

Choroid.—The choroid is involved late in the secondary period; it may be involved secondarily to iritis or cyclitis. The several forms terminate with the same appearances—*e. g.,* the exudative choroiditis with bright yellowish, bluish or even rose-colored spots, which are small, oval or irregular, and are usually peripherally pigmented. The pigmented type is without spots. Both the spots and pigment lie behind the retinal vessels, which branch over them in the ophthalmoscopic picture. (See Retinal Plates under MILIARY TUBERCULOSIS.) Disseminated choroiditis consisting of spots of pigment, especially in the peripheral eye ground, usually indicates syphilis. When occurring with inflammation of the retina and choroid (choroiditis totalis) the prognosis is usually bad, as blindness may result from recurrent attacks. Though no objective signs positively point to a syphilitic origin, syphilis is always suspected. The *vitreous humor* is rarely involved without other lesions, especially those of the uvea. It results merely in opacities or dust-like bodies; in the absence of myopia and hemorrhage, it rather indicates syphilis.

Retina.—The retina is involved chiefly in its anterior layers, becoming cloudy about the disk, with opacity of the nerve and blurring of the disk outlines. The course of the arteries is lost or broken and the veins are tortuous and engorged. Hemorrhages are uncommon. Most idiopathic cases of retinitis are syphilitic, although no unique morphological characters exist. Nodules, either papules or gummata, constitute the specific retinitis. This is usually late or tertiary and its prognosis is usually good. Central recurring retinitis (v. Graefe), commencing at the macula, is of great importance. Retinitis with pigment formation occurs mostly in congenital syphilis. The *optic nerve* is usually involved with the retina. It may be involved anywhere from its central origin to the disk. Sometimes the change is simple blue atrophy; again the appearance is nearly normal, or again choked disk occurs. The causative lesions are gumma in the brain, basal disease or independent syphilitic inflammation of the intracranial portion of the tract. Neither its syphilitic nature nor its location can be diagnosticated by the ophthalmoscope. Atrophy may occur, as in tabes or progressive paralysis. Amblyopia or amaurosis may result from transitory circulatory, or permanent organic changes. Hemianopsia is often central, results from lesion in the optic tract and, in most cases, is transitory.

Eye Muscles.—The eye muscles are involved as frequently as is the iris. Syphilis explains many so-called rheumatic cases; more than half the paralyses of the eye muscles are syphilitic. Ocular paralyses may be (*a*) peripheral; (*b*) intracranial as at the base of the brain; or (*c*) central, comprising nuclear, fascicular and cortical localization. Anatomically the following lesions occur: Syphilitic meningitis, basal gummata and periostitis, cellulitis of the orbit, diffuse arterial disease, nuclear disease, poliencephalitis and gummata of the hemispheres or cortex. Other causes, as tabes, general paralysis and tumor, must be considered. Thirty-six to 50 per cent. of nuclear

paralyses are syphilitic. According to Gowers, syphilis frequently produces progressive paralysis of all the muscles of both eyes. Total paralysis of the *oculomotor* is rare; if it occurs, the lesion is usually at the base, is rarely orbital, or may be nuclear with localization in the third ventricle, especially if one twig after another become involved. If it is partial, it is usually nuclear, rarely neural or muscular. Ptosis occurs very frequently, and is highly suggestive of syphilis. Abducens and trochlear paralysis may occur. Ophthalmoplegia interna, involving the sphincter iridis and the ciliary muscle, produces mydriasis and paresis of accommodation. Thirty-six per cent. of all mydriases are syphilitic (Alexander). Its origin is purely nuclear (Mauthner). Ophthalmoplegia externa may occur with facial paralysis, involving the orbicularis palpebrarum, or with trigeminal paralysis involving the sensory filaments of the eye (in 25 per cent. of brain syphilis, Uhthoff).

Orbit.—Cellulitis and periostitis occur frequently. Periostitis is indicated by deep pain on pressure over the eyeball, or over the edge of the orbit, which is sometimes prominent; by pain on movement of the eye; more rarely by blindness, exophthalmos and thrombosis. Periosteal gummata may often be mistaken for tumors.

Ear.—Four instances of primary sclerosis on the external ear have been noted (Politzer). The ear in general is involved in 0.6 per cent. in acquired syphilis; 33 per cent. of children with hereditary syphilis are deaf. Secondary maculæ or condylomata, mastoid periostitis or gumma, sclerosis or gumma of the Eustachian tube, suppurative otitis media, sclerosing otitis media and labyrinthine disease (rapid deafness, vertigo and tinnitus), may be observed.

11. Genitalia.—Some primary lesions occur in the *urethra.* Secondary manifestations are infrequent; gummata have been occasionally observed in the urethra and bladder.

Penis.—Gummata may occur at the site of the primary lesion or elsewhere, and often have been confused with chancre, chancroids, pustules or carcinoma.

Testes.—(*a*) The *diffuse interstitial form* (orchitis fibrosa syphilitica) occurs with great enlargement; the swelling is pyriform, with flattening of the epididymis; it is more often nodular than smooth, due to periorchitis or sometimes to gummata. Serous effusion and partial or total synechia occur. Recovery by absorption of this sarcocele syphilitica may result. The connective tissue is increased and the canals are compressed, separated, fatty and infiltrated with round cells. (*b*) *Gummatous orchitis* is less frequent, and usually involves one testis, and also the epididymis and cord. Great symmetrical or irregular swelling may occur, and frequently the entire testis is elastic, firm and studded with gummata. It is often mistaken for sarcoma. The testes atrophy and sclerose. The symptoms develop gradually or after strain or trauma. The anterior portion of the testis is most involved, with single or multiple, hard, irregular nodules, which are usually not painful. The veins are dilated and

adhesions occur. The scrotum is infiltrated, nodular and red or violet in color. Perforation is frequent, with a crater-like opening and irregular edges. Oligospermia, azoöspermia or impotentia do not always follow, because islets of functionating tissue frequently remain. Differentiation is required from gonorrhœal orchitis, which is more rapid, painful and involves the epididymis first; plastic orchitis, resulting from stricture, traumatic orchitis and mumps orchitis; also from tuberculous disease, which is often bilateral, or which at least occurs in several foci, and frequently involves the cord, vesicles, epididymis and often causes perforation with pain, fever and prostration, in which case tubercle bacilli may be detected. Differentiation is necessary from sarcoma, which is harder, develops more rapidly, is more nodular, produces lancinating pains and frequently affects the epididymis and cord, which are less often involved in syphilis. Sarcoma also invades the glands and, when perforation results, is more rapid and painful and occurs with fever, necrosis, hemorrhage and cachexia. Carcinoma, which is often difficult to differentiate from the interstitial form of syphilis, does not respond to specific treatment.

Vas Deferens.—Gummata may rarely form. Syphilis of the seminal vesicles may convey the disease to the mother and child. Prostatic involvement is rare.

Vagina.—Chancres are relatively rare, because of the thick epithelium and the paucity of glands; secondary eruptions are rarely observed. Condylomata must disappear under mercury to make certain the diagnosis. Gummata rarely occur.

Vulva.—The vulva is very frequently the seat of secondary lesions, upon which tertiary lesions may develop. These may extend to the urethra or bladder. Enormous swelling, sometimes resembling blisters, is a common result of lymphatic involvement. Phagedenic ulcerations or strictures may occur. Gummata are readily differentiated from carcinoma and tuberculosis.

Cervix.—Chancre occurs in 5.5 per cent. of the cases (Fournier, Mrâcek and Neumann). Scars may result, with stenosis and sterility. Tertiary lesions may occur and recur. Diagnosis may be difficult.

Uterus.—Syphilitic *endometritis* is frequent; its symptoms are the same as those of the non-syphilitic type; metrorrhagia may respond only to mercury. Virchow describes a papular and a tuberous variety. Endometritis may produce sterility. *Metritis* syphilitica has been less studied, but clear cases are recorded, in which large tumors disappeared on administration of iodide. Specific perimetritis or simple perimetritis is secondary to rectal disease.

Tubes and Ovaries.—Salpingitis and oöphoritis syphilitica and gummata have been described.

12. Bones, Joints and Muscles.—Secondary *periostitis* produces pain, tenderness and considerable swelling, which is hard at first, and later is elastic even to fluctuation. It occurs especially on

parts subject to trauma, as the forehead, temporal bone, sternum, ribs, tibia or clavicle. Osteophytes develop. Fugitive pain may occur with the secondary eruption, but the pain of periostitis is fixed; it may radiate but rarely migrates, is increased by pressure, is largely boring in character, appears at night—the *dolores nocturnæ*—and disappears at one or two o'clock in the morning with sweating, but otherwise resembles the common types of periostitis. In 28 per cent. of cases it occurs in the frontal or temporal bones, and forms circumscribed, smooth, elastic and immovable tumors. On the ribs, it occasions pain on coughing, neuralgia over the sternum or possibly dyspnœa. Tibial nodes occur in 16 per cent. of cases. Periosteal changes last for five or six weeks. *Gummata* of the bones were the first well-studied form of gummata. They occur on the exposed parts largely as: (*a*) Diffuse subperiosteal infiltration, which causes molecular destruction of the bone, and possibly reaches the marrow or meninges (perforating type). Osteophytes develop as an effort of nature toward regeneration. The bone may rarefy or show foci of sclerosis; multiple areas exist in the pernicious forms of gummata. In large bones sequestra may form, especially in the femur; osteophytes in the diaphysis sometimes resemble arthritis deformans. (*b*) Circumscribed gummata, the more important form, develop chiefly in the periosteum; they form elastic, immobile, not especially tender tumors which afterward soften and, at a late stage, may fibrose and calcify.

Symptoms.—Gummata of the cranium are attended by dull pain, especially at night; have a slow growth; are at first soft, and later have a wall-like, hard edge. They rarely ulcerate and often disappear spontaneously, leaving a somewhat elevated periphery and a deep centre. They are very often mistaken for supraorbital neuralgia or migraine. Tuberculous ulcers rarely occur in this location. Pyogenic infection may produce ulceration; or meningeal symptoms may develop. Gummata *at the base,* where periostitis is observed infrequently, occur chiefly in the middle fossa and involve the nerves or bloodvessels. Diffuse gummatous infiltration develops very slowly, with little pain, possibly with ulcerative exposure of the bone and is diagnosticated with difficulty. The *spine* is rarely the seat of gummata; but when involved, the affection is largely cervical, rarely dorsal, very rarely lumbar, and affects the bodies of the vertebræ less than their processes and arches, in contradistinction to tuberculosis. Pain and immobility are observed. The head is held in the hands, if the atlas is involved; recovery is possible, but death may result from ulceration, pressure on the medulla or luxation. Paralysis of the upper extremities has been observed. The *humerus, radius* and *carpus* are less frequently involved. The phalanges may be involved, but this usually affects one finger only, the *spina ventosa,* the parts being swollen but soft, with periosteal thickening, shining appearance and rarely pain or ulceration. The clavicle, from its slight protection and its exposure to trauma, is very frequently involved; the

course is slow, often without pain. Ulceration of the sternum is frequent; of the ilium and sacrum rare.

Joints.—(*a*) *Acute synovitis* occurs alone or with bone changes at the time of the eruption; pain alone may be observed or the swelling may be multiple, constituting the pseudorheumatism of Fournier, with tenderness, nocturnal pain, immobility, temperature, fluctuation and involvement of the periarticular structures, as the muscles and tendons. The acute form is polyarticular, while the subacute variety is oftener monarticular. The knees are most frequently involved and, according to Fournier, more than three joints are never affected. The affection may last for weeks or months, does not induce cardiac complications, and responds to mercury and iodide and rarely to salicylates. (*b*) *Chronic synovitis* or arthritis is usually monarticular, indolent, with little or no pain or fever, and affects chiefly the knee and elbow, with fluctuation and slow loss of function. It responds to antisyphilitic treatment. (*c*) Joint disease of late syphilis originates in the bone or cartilages (*tumor albus syphilitique* of Richet), and affects chiefly the knee and elbow and next in frequency the ankle. The central portion of the cartilage is involved, and not its edge, as is the case in arthritis deformans. This is probably due to gummata originating in the bone and is most frequently seen in congenital syphilis. The symptoms begin slowly with moderate pain, swelling and localized tumors; no temperature is observed, the symptoms vary and contracture may result. There may be a close resemblance to tuberculous lesions, diagnosis from which depends upon other stigmata of syphilis or tuberculosis; while tuberculosis is usually inflammatory, incipient syphilitic lesions are non-inflammatory; in syphilis, sequestration and ulceration are infrequent; therapy is the final test.

Muscles.—Early involvement may occur from *toxæmia* or *myositis.* The latter is either a diffuse or circumscribed infiltration, which possibly eventuates in muscular contractures, especially in the arm and finger flexors, sphincter ani, masseters, deltoid and sternomastoid. The course is subacute or chronic, depending on treatment. The affection is most often observed from two to twelve months after infection. Differentiation from muscular rheumatism may be necessary. *Gummata* of the muscles are important because of confusion with neoplasms, especially in the arms and neck; they may be as large as the fist, single or multiple, usually with a firm connective-tissue capsule; they involve the long muscles, especially at their attachments. The course is long, and regression may occur with induration. *Myositis ossificans* is rare. Syphilitic *atrophy* of the muscles may follow arterial disease and, less frequently, central nervous lesions. The *tendons* may be involved, partly from changes in the bones or muscles. (*a*) The irritative form, occurring early as acute bursitis or tendosynovitis (Fournier), is sometimes confused with rheumatism. There is more tension than pain. It occurs most frequently in sewing women and washerwomen, and the exudate is usually absorbed. (*b*) The gummatous form, occurring in the flexor

tendons, is characterized by slow growth, little pain and usually a favorable prognosis.

13. Mamma.—The mammæ may be involved (*a*) by diffuse infiltration, *syphilitic mastitis,* with increased size, and even tumor formation. The affection occurs largely in the secondary stage, and often resorbs spontaneously. (*b*) *Gummatous mastitis* is deep-seated and is not very sensitive. Rapid growth may be observed with central necrosis, fluctuation, or even external ulceration. It requires differentiation from adenoma, fibroma and carcinoma; no glands are involved, it occurs frequently on both sides, without cachexia, but possibly with gummata elsewhere, and it responds to antisyphilitic treatment. (*c*) 206 cases of mammary *chancre* are recorded (Dimey).

Abnormal Course of Syphilis.—Of special importance is *malignant syphilis* (syphilis præcox, acute or galloping syphilis, synonymous with early tertiary development). The cause may be increased virulence of the spirochæte, or decreased physiological resistance, as in cases of tuberculosis, alcoholism and other cachexiæ. Early ulceration, grave general symptoms and hemorrhage are noted.

Prognosis.—The mortality is great in congenital syphilis (*v. i.*); tuberculosis, retarded development, idiocy and epilepsy may result later. On *pregnancy* the disease has a bad influence and mercury is less universally successful. *Exanthematous diseases* may cause syphilis to disappear but their curative influence is short. The effect of coincident measles and smallpox is unfavorable. *Chronic diseases,* as tuberculosis, are unfavorable and often induce the ulcerative form with early tertiary symptoms. Syphilitic cachexia may be complicated by tuberculosis; glaucoma may become aggravated, and syphilitic psoriasis may become carcinomatous. *Trauma;* in the treatment of fractures in syphilis, mercury is indicated. Syphilis is important in plastic operations. Wounds may become the seat of specific ulceration.

In *life assurance,* the outlook is uncertain; it has been estimated that 11 per cent. die before their expectancy (Runeberg); the main direct causes of premature death are cardiac disease, aneurysm, nephritis and nervous localizations (dementia, tabes and arterial thrombosis); the chief contributing causes are poverty, worry, overwork, tuberculosis and alcoholism.

II. Hereditary Syphilis.—That syphilis descends only to the first generation is the general belief. Fournier (1905) finds 115 cases recorded in the second generation, of which many are very doubtful.

If both parents are diseased the child mortality is 75 per cent., but this depends on treatment; 95 per cent. of living syphilitic children die, if not treated, and only 10 per cent. if treated (Etienne); only 3 per cent. die, if the parents were properly treated (Fournier). In 330 syphilitic pregnancies, abortion or premature delivery occurred in 40 per cent., and delivery at term in 60 per cent.

1. Syphilis of the Child Acquired from the Father at the Time of Con-

ception.—Purely spermatic (paternal) infection is the simplest and most frequent variety, in that 97 per cent. of cases of syphilis occur in men (Fournier), although a larger ratio occurs in women of the lower classes. It rarely occurs after the second year of infection has passed. This form of transmission is more probable, the more recent the paternal disease. The effects are shown by abortion, premature birth, less frequently by syphilitic children at full term, or all of the above successively (the virulence of paternal syphilis decreasing with time). The child mortality is 28 per cent. and the morbidity 37 per cent., this being the most favorable form. The mother, it was once maintained, remained uninfected; since the discovery of the spirochætes, it would appear that the mother is always infected.

2. Syphilis of Child from Mother, Syphilitic at the Time of Conception.—Infection is most dangerous in the first year of the disease and generally persists longer than in man. Abortions occur first, then premature deliveries, and finally, dead or diseased children at term. The mortality is 60 per cent. and the morbidity 84 per cent.

3. Infection of the Mother and Conception Occurring Simultaneously.—This form is dangerous to the child, though early treatment may modify the result. According to Neumann, in 29 per cent. of cases, the children are not viable, and 39 per cent. die soon after birth (*i. e.*, 68 per cent. die).

4. Postconceptional Syphilis.—The child is less liable to contract the disease, the later the infection of the mother; it may even escape direct infection and only suffer inanition. The virus may pass through the placenta to the child, although there is a theory that the placenta, if not diseased, may protect the child from infection.

Influence upon the Sound Mother of the Child Infected by the Father.—Colles' (or Beaume's) law is as follows: The child is held to make the mother immune to infection; she cannot be infected from her infected child; she can nurse her syphilitic child without danger. Some exceptions exist and 30 cases are reported in which the mother was infected after the birth of a syphilitic child. In such cases, Fournier held that the mother is already syphilitic. Sound children of syphilitic parents have apparently some immunity against syphilis because of action of toxins (Profeta's law,—*i. e.*, the syphilitic mother is said not to infect the sound child, which theory has many exceptions). If the children acquire syphilis, it is said to run a mild course. It is, nevertheless, a fact that the sound child may become infected *intra partum* (Grunfeld).

Influence on the Child.—Abortions and miscarriages have been overestimated as evidences of syphilis. They are frequent in animals. Abortion, atrophy, maceration, retarded development, congenital syphilis or evidences of the disease after birth are frequent. Many children, born first after infection, die, though those born later may be sound. A difference exists between the congenital syphilis with signs at birth, and the hereditary type, which develops later (possibly very late, syphilis hereditaria tarda). Even when specific

lesions are absent, pathological cell degeneration, rhachitis, scrofulosis, slow intelligence, neuroses, psychoses and a high percentage of mortality to all diseases may result.

Symptomatology.—The primary, secondary and tertiary stages are not usually present and lymphatic infection does not precede that of the entire organism. Secondary and tertiary manifestations may occur in the most intimate and synchronous connection. The symptoms are usually recognizable at birth or soon afterward, usually after a few days or weeks, and almost always within the first three months.

Appearance.—The *atrophic child* presents a characteristic appearance, with relaxed yellow-gray skin, poorly developed hair, bent or undeveloped nails (onychia) and "the little old man" appearance described by Trousseau. The palms and soles are lacquered, the voice and muscles are weak and the child does not nurse well. Such children, especially when several are born in succession, are suggestive of syphilis, even when no lesions are found. Other *suggestive symptoms* include the *heavy placenta* whose weight compared with that of the syphilitic child is 1 to 5, 4 or 3 (normally 1 to 6); increasing loss of weight for one to two weeks (normally three days), which may amount to 13 ounces; and enlargement of the spleen. In the placental villi end- and periarteritis occur, which may cause, by vascular occlusion, placental necrosis and adherence. Similar changes in the umbilical vessels may destroy the child.

Pathognomonic Symptoms.—The *exanthem,* as in adults, is polymorphous, but varieties occur which are absent or infrequent in the adult, such as pemphigus, hemorrhages, furuncles and diffuse infiltration of the skin. There is a general tendency to severe forms. The maculæ are dirty, brown-red and often confluent, and occur on the face, body, extremities and in the genital region. The papules occur principally in the folds of the joints, axillæ, groins and gluteal region and tend to recur; papules of various ages co-exist with desquamation, though never with condyloma, which is always a sign of acquired syphilis. They tend to ulcerate. Psoriasis of the hands and soles of the feet with desquamation is common. Pustular lesions occur in severe syphilis and are prognostically unfavorable. They occur not only in the new-born but also in the immature fœtus; pemphigus is a subtype of the pustule; gangrene and necrosis may occur. The copper-colored syphilitic infiltration especially on the nates and lower extremities, often may be mistaken for erysipelas. Coryza (*"snuffles"*) is very characteristic and almost invariable, the secretion being bloody and purulent, with crust formation. Fissures occur about the mouth, with an infiltrated base and cicatrix formation. Papular eruptions (erosions) occur in the mouth, especially on the tongue and the palate, with a tendency to necrosis. Eruptions and *rhagades* occur about the genital and anal regions.

Teeth.—Erosions, furrows and notches may develop. The teeth are small, often irregular and poorly developed. Hutchinson held

that the semilunar curving on the free edge of the upper middle permanent incisors was pathognomonic of tardy hereditary syphilis, but *Hutchinson's teeth* occur in other affections, such as scrofula and rickets, and are expressions of constitutional derangement and not necessarily syphilitic. Later the lateral borders of the teeth become curved; the teeth may become peg-like or the notches grind off even.

FIG. 19.

Notched teeth. Malformation of permanent teeth found in hereditary syphilis. (Jonathan Hutchinson.)

Bones.—The bones, as in adults, may present characteristic findings. There may be periostitis, tophi or perforated palate. *Osteochondritis* occurs in various bones but especially in the lower end of the femur; the diaphysis and epiphysis may be separated (Wagner). There is (*a*) overgrowth of the cartilage of the diaphysis which produces the wavy line of white cartilage cells; (*b*) the line of cartilage cells becomes thick and foci of decalcification occur; (*c*) the cartilage is as prominent as it is in rickets, the part nearest the bone being soft. The immobility of the leg may suggest paralysis (Parrot).

Viscera.—The changes are vascular or gummatous. There are characteristic changes in certain organs which are rarely involved in acquired syphilis;—*e. g.,* in the thymus, umbilical cord, pancreas, lungs, intestines and adrenals. The liver and spleen are greatly enlarged. To avoid repetition and to conserve the classification, these changes have been described under SPECIAL SYMPTOMATOLOGY. Vascular changes are especially marked, as in the umbilical vessels, with a tendency to hemorrhage—*syphilis hæmorrhagica neonatorum.* Hemorrhages in the skin, mucous membranes and viscera, may be profuse, especially in the liver and the lungs, where the circulation is precipitately altered after birth. Some vessels may be thickened or infiltrated (disease of the vasa vasorum). There is often deficient brain development or meningitis in the posterior cerebral fossa.

ACQUIRED SYPHILIS IN CHILDREN	*vs.*	HEREDITARY.
Less mortality and less malnutrition.		Marked.
After third month (are exceptions).		Before third month.
A primary lesion occurs.		Absent.
Secondary lesions, then tertiary.		Secondary and tertiary together.
Lymph glands enlarged.		Absent (except from accidental causes or suppuration).
Usual sequence of primary, secondary and tertiary symptoms.		Snuffles, anæmia, diffuse pigmentation, pemphigus, involvement of soles and palms, epiphyseal changes, etc.

Syphilis hereditaria tarda develops its first symptoms in later years, as for instance, at puberty. This variety is recognized if (*a*) the parents are syphilitic, and if (*b*) the individual has presented no previous signs of syphilis. Most cases begin between the tenth and eighteenth years (rarely before the tenth and even after the fortieth year). Most cases are seen in private practice. The symptoms are mostly those of infantilism, in regard to puberty, bone development, teeth, hair and brain.

Immunity.—Syphilis usually confers *immunity,* but this is neither absolute nor life-long, because (1) reinfection may occur (80 instances, Stern, 1907); (2) in hereditary syphilis, a later infection may be acquired (27 cases known); and (3) a sound child of syphilitic parentage may acquire syphilis (29 cases reported).

Treatment of Syphilis.—Prophylaxis.—The disease prevails in spite of the numerous opportunities for free treatment, for the sexual instinct is even stronger than that of self-preservation. Irregular intercourse is possible because of class differences, lack of barriers among the ignorant and destitute, and is promoted by the struggle for existence which often makes early marriage impossible. Prostitution, hidden or open, is the basis of syphilis. Sixty-nine per cent. of women in open houses have the disease for months before treatment is instituted, while hidden prostitution among waitresses, for example, is much more dangerous. In prostitutes, condylomata last as long as seven years and repeatedly recur about the genitalia. Continence is the sole prophylactic measure. Enforced medical inspection is ineffectual. American sentiment is against licensed prostitution, which, besides, is incomplete because it does not regulate men or unlicensed hidden prostitution. Licensing also teaches the young that immorality is safe. Many infections are acquired under the influence of liquor (Sine Baccho friget Venus). Where ethical considerations fail, an appeal to fear may be effectual. Metschnikoff comments upon the remarkable fact that diseases conveyed by insects are rapidly becoming preventable, whereas diseases like syphilis and tuberculosis conveyed by rational human beings, are not. Efforts to educate the public will doubtless avail much.

The importance of the disease lies in its long course, in the involvement of vital viscera, in its transmission to the wife and offspring whose mortality and morbidity are enormous, in its wide dissemination (13 per cent. of the population acquire the disease, and 1,650,000 persons are infected annually), and finally in our possession of specific remedies.

Marriage should be forbidden for at least three or better, four or five years. Coitus must be avoided when there are recurrences. In cases of doubtful infection of the wife or husband, both should be treated. Inunctions should be given the pregnant mother, whether she be infected in the first or second part of pregnancy. If the child be diseased and the mother healthy, the facts should be laid before her and some would then allow her to choose whether she

would nurse her child. Mercury should be given her. The physician should never countenance employing a healthy wet-nurse for the syphilitic child. Circumcision is thought to lessen chances of infection. Metschnikoff advises a prophylactic calomel ointment, to be applied at once after intercourse.

1. Initial Lesion.—Extirpation is inefficient. The early use of mercury does not prevent the secondaries or the recurrence of the disease; it may lessen secondary manifestations. Nevertheless, the writer believes early treatment is indicated, but only when the diagnosis is certain, *i. e.*, when there is a typical chancre and spirochætes are found. *The diagnosis must be positive,* as syphilis entails years of scrutiny and complicates the life of the wife and offspring. Local treatment includes the use of antiseptics, iodoform, aristol, mercurial plasters, iodoform in suppository in rectum or vagina, bichloride washes for mouth and calomel for condylomata (calomel 6 parts, boric acid 3, and salicylic acid 1 part).

2. Secondary Stage.—In the secondary stage *mercury is a specific.* It must always be used with care; the teeth should be cleaned first, and bad teeth filled. Calomel acts especially on the lesions of the mouth and is prone to produce salivation. Mercury is indicated for all of the irritative (*i. e.*, secondary) phenomena of syphilis, and Gowers always uses it with the iodides, even in the tertiary stage, especially in nervous lesions. Mercury may produce bad effects (*v. i.*) in tuberculous, cachectic, alcoholic, malarial or anæmic subjects, in bleeders and in cases of galloping syphilis, in which latter iodides are considered by some better than mercury. Mercury must be used with great care in nephritic subjects. During and after a course of mercury, *tobacco must be interdicted* because it perpetuates syphilitic sores in the mouth for years, even decades. Moderation in all matters of life is enjoined upon the patient.

Methods of Administration.—1. *By Mouth.*—Ninety-five per cent. of cases are treated by this method in this country: (*a*) Calomel (Hydrargyri chloridum mite) is especially adapted to children with hereditary syphilis, who are rarely salivated under three years of age; gr. $\frac{1}{4}$ to $\frac{1}{2}$ t. i. d. plus opium p. r. n. It is insoluble, and is probably converted by the alkaline carbonates in the intestines into the gray oxide which is absorbed with the fats. (*b*) Blue mass (Massa hydrargyri), gr. $\frac{1}{2}$ to 1, is milder than calomel and less irritant to the stomach, but produces salivation oftener than any other mercurial. Women are less subject to ptyalism than men. (*c*) Hydrargyrum tannicum oxydulatum (Lustgarten) contains 50 per cent. mercury, is non-irritant, does not salivate, produces less diarrhœa and is easily assimilated; gr. 1 to $1\frac{1}{2}$ plus pulv. opii gr. $\frac{1}{10}$; (*d*) The Hydrarg. iodidum flavum or protiodide is mild, insoluble and analogous to calomel.

℞ Hydrargyri iodidi flavi ..... gr. xv.
Extr. opii ..... gr. v.
Extr. gentianæ ..... q.s.
M. et ft. pilulæ no. 60.
S.—One after meals.

(*e*) The bichloride of mercury is irritating (Hydrarg. chloridum corrosivum); it is not prone to salivate, is absorbed by the intestine and stimulates the liver. Mercury, administered internally, produces more irritation and is less likely to be absorbed. Patients are more apt to persist in this form of treatment. Sublimate is readily soluble. It is used especially in children and in lighter forms of the disease. Mercury internally was first used by Benedictus (1525).

2. *Inunctions.*—These must be thorough and should be given where the skin is soft (flexor surfaces). The mercury is rubbed into the sebaceous or sweat-glands and is probably absorbed by the lymph-vessels. The skin must be cared for by daily baths. A simple diet and mild laxatives should be given, though, as a rule, mercury somewhat loosens the bowels. The rubbing should continue for twenty to thirty minutes. Hairy regions, the axillæ, navel, nipple and areas of accidental eczema, abscess, psoriasis or furuncle, are to be avoided. On the first day the inunction should be given on the abdomen, on the second day on the chest, on the third on the calf, then on the thigh, forearm, arm, etc. After inunction, the part is enveloped in cotton. If an attendant gives the rubbing, his hand should be protected by a rubber glove. Written instructions should always be given to the patient.

The number of inunctions necessarily varies with the case; twenty to thirty should be given for the macular and papular types; more than thirty, for all pustular or ulcerating eruptions, but not more than thirty should be given continuously. Inunctions must be interrupted for ulceration, diarrhœa or stomatitis. Salivation is not desirable, nor is it a sign that enough mercury has been given. The *unguentum* contains 50 per cent. of mercury, and the more elegant oleatum but 20 per cent., which should be considered, as about 3ss of mercury should be incorporated daily.

℞ Unguent. hydrarg. ..... ʒiv.
Div. in dos. æqual. no. xxx.
Dent. ad cartam cerat.
S.—One as directed daily.

In children, mercurial ointment may be applied on a binder and allowed to remain for many days (Westlander). Children stand relatively large doses but women usually endure only half the adult dose. The advantages of inunctions are as follows: They cause almost no digestive disturbance; they are especially good in children; they are the most rapid method, and are attended by the least general bad effects. The disadvantages are publicity, troublesomeness and dirtiness, all of which, however, are but relative objections. Inunctions were first used by the Galenists.

3. *Injections.*—A great diversity of opinion exists as to the value of injections. Their advantages are prompt action and exact dosage. Intravenous injections may be imperative in cases with coma and other urgent symptoms. The disadvantages are the risk of em-

bolism, suppuration, sloughing and pain; once given, their action cannot be regulated or decreased. The soluble salts are best:

℞	Hydrarg. chlorid. corrosivi	1.00
	Sodii chloridi	10.00
	Aquæ dest.	100.00

One c.c. daily equals 0.01 gm. or gr. $\frac{1}{6}$ of $HgCl_2$; forty injections are usually necessary.

4. *Baths.*—℥ij to iij of $HgCl_2$ to a full bath are useful for skin lesions, such as syphilitic psoriasis. 5. *Fumigations* are impracticable. As a working rule, mercury should first be given two months and stopped one month, through the first year *i. e.*, two thirds of the time; in the second year, about half the time, with intervals; in the third year, one third of the time.

Mercury is *incompatible with nearly everything.* Coincident anæmia is treated by iron, strychnia and arsenic. Syphilitics tolerate mercury well, in large doses, and for a remarkable period. Indeed, they sometimes seem to develop an immunity to its action. It is not out of place to consider briefly the general action of the drug; in the smallest doses it is tonic, for it increases the number of red cells. In small doses there develops a symptom-complex known as salivation, of which peculiar fœtor is first noted; then sore teeth, red, swollen, spongy and bleeding gums, and increased saliva, follow. The drug is never given beyond this point. In larger doses the tongue becomes swollen, the gums inflamed; the teeth become loose; the saliva is increased greatly, is viscid and stringy; the salivary glands become intumescent; there is low fever; fugaceous eruptions appear, resembling eczema, scarlatina, measles and more rarely, purpura and pemphigus. Severe salivation results in loss of teeth, necrosis of jaw, ulceration of the mouth and contiguous parts, marked changes in the blood and profound exhaustion. The treatment consists of potassium chlorate as an antiseptic mouth wash, and astringents, as tr. myrrh and tannic acid.

3. Tertiary Stage.—Iodides are specific in this stage and their resolvent action is little short of miraculous. They were introduced by Welch of Ireland and popularized by Ricord in France. They are sometimes used in the secondary stage (*v. s.*), especially in febrile cases, secondary osseous and nervous manifestations, and weakly persons with a marked macular eruption. They act more slowly than mercury. They are especially indicated when the bones, nervous system and viscera are the seat of gummata, and when the general health is broken. Patients may acquire immunity to iodides and their use should therefore be intermittent and should alternate with mercury. Mercury is conjointly employed when organs of great importance are involved, such as the larynx, brain and eye.

The *therapeutic tests for syphilis* are (*a*) cure with mercury and iodides; (*b*) tolerance by the patient of very large doses. Regarding their diagnostic value, Gower's criteria are: (1) That the influence

of the drugs must be unequivocal and fairly immediate. (2) That no other remedy be used. (3) That spontaneous remissions seen in other diseases must be excluded. (4) That the remedies used must not operate on any disease other than syphilis. (5) Before negative conclusions are drawn regarding the syphilitic nature of the lesion, it must be remembered that loss of tissue, *e. g.*, in arterial occlusion and paralysis from brain softening, cannot be restored.

The iodides contain the following percentages of iodine: KI, 75.5 per cent.; NaI, 84.6 per cent.; LiI, 95.5 per cent.; AmI, 87 per cent.

Sodium iodide is milder, produces less iodism, has a less bitter taste, and has less effect on metabolism than potassium iodide, which however, is better than other iodides (Schmiedeberg), though it irritates the stomach and bowels, depresses the muscles, and depresses the motor nerves.

The following proportions for a single ordinary dose may be employed; the mercury becomes mercuric iodide:

℞ Potassii iodidi ..... gr. xv. (gm. 1.0)
Hydrargyri chloridi corrosivi ..... gr. $\frac{1}{32}$ (gm. 0.002)
Tincturæ opii ..... ♏ 2 (gm. 0.13)
Syrupi aurantii corticis ..... ʒss (gm. 2.0)
M. et S.—After meals in a glass of water.

Large doses of iodide are sometimes given in urgent cases, ʒj–ij t.i.d., to break down more rapidly the new-formed granulation tissue. The author believes that enormous doses are unnecessary and are not without danger (*v. i.*). Fournier warns against their intemperate use—"iodide debauches." Iodide dissolves in its own weight of water; hence one minim of a saturated solution equals one grain of iodide. It is best disguised by Syr. sarsaparillæ co., dr. ss and Tr. iodi, ♏ 1.

Iodism is often produced by small doses, and usually ceases with larger ones. It is obviated by intermittent administration, by daily baths, by flushing out the kidneys, the inactivity of which is the usual cause, by giving aromatic spirits of ammonia, or arsenic and belladonna. Iodism is likely to occur in renal disease. Its leading characters are: *Increased secretion* from the eyes, nose, mouth, salivary glands, bronchi, stomach, bowels, skin and kidneys. The eyes, throat, larynx and salivary glands swell. The writer once nearly suffocated from œdema of the pharynx and uvula after ingestion of 10 grains of iodide for a cold. Laryngeal and pulmonary œdema and albuminuria, or even nephritis, may develop. *Gastric* irritation; and *nervous depression,* mental derangement or excitement. *Acne,* hemorrhages, blebs and telangiectases in the skin. *Cachexia and anæmia,* especially in goitrous subjects; emaciation, wasting of mammæ and testes; fever, increased pulse and neuralgias.

The *iodides are continued* for two months and their use then remitted for one month and so on. After several years, iodides are given for one month twice a year *e. g.*, in January and July.

A Syphilitic Should Never be Dismissed or be given any guarantee of the extinction of the disease (Ricord).

TUBERCULOSIS.

Definition.—A specific infection, characterized (*a*) etiologically by the Bacillus tuberculosis, (*b*) pathologically by nodes or tubercles, or diffuse tuberculous infiltration, and (*c*) clinically by symptomatology varying with the tissues or organs invaded. Tuberculosis, by derivation, refers to tubercles, but in the light of our present knowledge, only to tubercles caused by the Bacillus tuberculosis.

History.—The term phthisis (wasting) was first used by Hippocrates. The history of tuberculosis includes several epochs: *Epoch I.* The anatomical tubercle was recognized by Sylvius (1614–1672), Morton (1689) whom some consider the real pioneer, Mangetus (1700) and Bayle (1810), who specially described the miliary tubercle. Laennec, the pupil of Bayle, in 1819, spoke for the unity of phthisis. Virchow threw the weight of his influence for Laennec's views, but in 1847, showed that caseation was only a retrogressive change and not necessarily tuberculous. In 1857, Buhl described a case of miliary tuberculosis arising from old foci of caseation. Cruveilhier, Rillet and Barthez identified scrofula and tuberculosis.

Epoch II was opened by Villemin (1865) who positively established the inoculability of tuberculosis by experiments on animals. Klenkpe (1843) made some successful inoculations, but Villemin absolutely proved his point. It is interesting to note that from the time of Hippocrates, Aristotle, Aretæus and Galen, tuberculosis was feared as a contagion, and the older pathologists, as Morgagni, dreaded to section phthisical subjects. Villemin's formulations met great scepticism and opposition, but were supported by Fox, Charcot, Grancher, Thaon, Cohnheim and Salamonson. The histology of the tubercle was developed by Wagner, Schüppel and Virchow; Langhaus fully described the giant cells previously seen by Virchow and Rokitansky; tubercles were found in lupus (Friedländer, 1875), in lymph-glands (Schüppel, 1871) and in fungus joints (Köster, 1876). The firm and ancient belief in its contagiousness led to the successful search for the cause. It seems strange that Aufrecht, Baumgarten and von Ziemssen still practically deny the infectivity of tuberculosis.

Epoch III began with Koch's announcement, in 1882, of the tubercle bacillus. His report, as health officer in an obscure German town, was so complete that scarcely a single essential point has since been added to his initial communication. The bacillus was found in all tuberculous lesions, such as chronic phthisis, miliary tuberculosis, intestinal ulcers, "scrofulous" lesions, bone and joint disease, lupus and in the sputum, in cattle, etc.; it occurred in no other disease; it was cultivated by him and inoculated into animals with

reproduction of tuberculosis, thereby fulfilling what has become known as "Koch's law."

Bacillus Tuberculosis.—1. MORPHOLOGY.—It is a thin, narrow rod, measuring $1\frac{1}{2}$ to 4μ long and more often slightly curved than straight; it contains no spores, the light unstained areas in its body being vacuoles or degeneration; it is immotile; in young cultures it is shorter than it is in the sputum; in old cultures and in lung cavities it is longer and is frequently thread-like; it occurs singly or in groups and bundles. Pleomorphism; variations in form are often noted, as bulging sides or ends, thread-like or branching forms and even forms said to resemble actinomyces. (See Plate under PULMONARY TUBERCULOSIS.)

2. STAINING REACTION.—It stains slowly, except at body-heat, and once stained gives up the dye reluctantly. Ziehl's solution is the best for clinical purposes (fuchsin 1, absolute alcohol 10 and 5 per cent. carbolic solution 100). The sputum is spread out in a dish over a black background and the yellow-green areas selected are thinly distributed on the slide which is held in the hand, and heated until dry at some distance from the flame; it is then stained by the carbol-fuchsin, being passed repeatedly but lightly through the flame (without boiling) to deepen the stain; it is then washed, treated with 33 per cent. nitric acid, washed well in water and then alcohol and dried. If the sputum is thick or if previous examinations were negative, 15 c.c. of sputum with 30 c.c. of water are treated with 8 drops of a 10 per cent. caustic potash solution, are shaken and allowed to settle for some hours, after which the supernatant fluid is decanted, and the sediment is examined directly or after centrifugation. Spengler digests the sputum with pancreatin instead of using potassium hydrate. The bacillus resists destaining because of its fat, an almost pathognomonic feature. For clinical purposes only (*a*) the bacilli found in bronchiectasis and gangrene of the lung (*q. v.*), (*b*) the smegma bacillus and (*c*) the leprosy bacillus need be considered; the leprosy germs are intracellular, cover the nucleus of the containing cell, and are stained in six or seven minutes with alcoholic fuchsin, while tubercle bacilli are extracellular and stain slowly; the smegma bacillus causes many errors in genito-urinary examinations; it grows rapidly in twenty-four hours, does not show the beaded appearance which is seen in the Bacillus tuberculosis, and when stained and counterstained in the usual way, becomes tinged with methylene blue (Weichselbaum).

3. CULTURES.—These develop best at body heat in one or two weeks; blood serum is the best medium, but growths are also successful on bouillon, glycerine-agar and potato; they are dry, thin, grayish and scale-like. The germ is a facultative aërobe and multiplies by transverse fission. It seemingly does not lead a saprophytic life in nature, outside the bodies of animals and of man.

4. CHEMISTRY, METABOLISM, RESISTANCE, VIRULENCE, ETC.—It contains 2 to 16 times as much fat as other bacteria. Aronson

holds that a waxy substance envelops the bacillus; its other constituents are proteids, a glycogen-like substance, cellulose, nucleic acid, water and ash; it also develops *tuberculin* whose chief components are peptone, albumose and alkaloidal substances; it is soluble in glycerine. Its *resisting powers* are as a rule limited. (*a*) Drying is endured by the bacillus strikingly well, for a few months (six or even ten months). In decomposing media its average life is one to one and a half weeks. (*b*) Heat (106°) kills it in a few minutes. (*c*) It may survive 14° below zero for weeks. (*d*) Sunlight kills the bacillus in a few minutes to a few hours.

Koch was inclined to assume that it had an equal *virulence* independent of the source of the germ, while the Arloing school finds that it has an attenuated virulence in glandular and osseous lesions, and Theobald Smith holds that bovine cultures are more virulent for rabbits than is human sputum. *Large numbers* are found in fresh tubercles; few are found in chronic articular, osseous or glandular lesions, and inoculations may be necessary to demonstrate the nature of long-standing lesions. Arloing and Courmont (1898) found that serum from inoculated rabbits or goats, agglutinized *tubercle bacilli* in a dilution of 1 to 5 or 20, in two to twenty-four hours; agglutination was noted in 95 per cent. of the cases of lung tuberculosis and in 50 per cent. of the cases of surgical tuberculosis. Its differential value has not, however, been generally conceded.

Associated or Mixed Infection.—Symbiosis has been noted with the streptococcus, staphylococcus and pneumococcus, to a lesser degree with the Bacillus pyocyaneus, influenzæ, etc. This has been much emphasized by Ortner, Weichselbaum, Strümpell, Roger, Dieulafoy, Mosney, Marfan, Osler, Wolf, Spengler and King, though Straus, Leyden, Baumgarten, Fränkel and Troje lay but little stress upon it.

Prevalence in Man.—Tuberculosis is the greatest foe of man; it causes 14 per cent. of all deaths, and 33 per cent. of deaths between fifteen and forty-five years of age; it causes more deaths than war, famine, plague, cholera, yellow fever and smallpox combined; in Germany, in 1894, the deaths from diphtheria, croup, measles, scarlatina, pertussis and typhoid, together, were 7,000 less than those from tuberculosis. Five million die yearly from this plague, one million in Europe, 70,000 in England, 150,000 in France, 150,000 in the United States, and 170,000 in Germany. Postmortem and hospital statistics do not exactly depict the tuberculosis situation; in Austria, the general death rate from tuberculosis is 11 per cent.; the hospital rate from the poorer classes is 25 per cent.

Quiescent (latent) and healed tuberculosis is found in many postmortems upon those dying of other diseases; Heitler, of Vienna, found obsolete pulmonary tuberculosis in 4.8 per cent. among 16,562 autopsies. Latent tuberculosis occurs in 25 to 33 per cent. of the population (Baumgarten, Birch-Hirschfeld and Bollinger), 39 per cent. (Harris) and 50 per cent. (Cruveilhier, Roger and Déjérine), 91 per cent (Burkhardt) and in 97 per cent. of all necropsies (O. Naegeli).

Prevalence in Animals.—(*a*) *Cattle.*—In Germany, it is found in from 4 to 20 or 30 per cent.; in Leipsic, it even reached 20 per cent. in bullocks and 48 per cent. in cows. In New York, the tuberculin test was positive in 17 per cent. In 1898, Theobald Smith, and later Koch, contended that bovine and human tuberculosis were separate infections; most writers incline to the view of Smith and Koch. In cattle, tuberculosis is less often attended by softening of the tubercle, and the serous membranes are more often involved; the lungs and pleuræ are involved in 40 per cent., the lungs alone in 20 per cent., and the pleuræ and peritoneum in 15 per cent. of tuberculous cattle. (*b*) *Swine* are affected more often in Europe than in this country; in Saxony tuberculosis occurs in 1 per cent. and in Copenhagen and Dantzig, in 11 per cent.; because their infection commonly results from eating tuberculous offal, the type is usually alimentary (90 per cent.). (*c*) *Other mammals.*—Dogs, cats, horses, sheep and goats, are rarely diseased. Monkeys in captivity die frequently (43 per cent.) from tuberculosis, referred by Dieulafoy and Krishaber to foul cages. (*d*) In *reptiles,* it is rarely seen, except in those in captivity. (*e*) In most *birds,* tuberculous lesions are more frequent in the liver, spleen, intestine, mesenteric glands, bones and joints, than in the lungs, though parrots suffer from skin and lung localization. Avian, bovine, reptilian and human tuberculosis are closely related types. Tubercles are not uncommon in animals from nematodes, protozoa and bacteria, and are designated pseudotuberculosis. In man, Flexner has described a streptothrix pseudotuberculosis causing caseous pneumonia and peritoneal nodes; the streptothrix differed both from the tubercle bacillus and the ray fungus. An aspergillus tuberculosis has been described (*v.* diagnosis Tuberculosis of Lungs).

Atrium.—The details will be especially considered under the different clinical localizations of the disease.

1. By the Respiratory Tract.—The bacillus enters the respiratory tract by inhalation. In the vast majority of cases *the sputum* of diseased persons is *causative* both *of the disease,* and its great frequency of localization in the respiratory organs. It has been estimated that a phthisical person voids 7,200 millions of tubercle bacilli daily. As the expired air is proven germ-free, the sputum must be the chief factor in dissemination. Cornet holds that the greatest danger lies in the *drying of the sputum* in the room, on the handkerchief, the lips, beard, hair, fingers and clothing. Flügge contends that dried sputum is less dangerous than the moist expectoration, and demonstrated that in coughing, which seemingly voids no secretion, in singing, in sneezing or talking, *small and almost invisible drops* of moisture containing the bacilli are voided; he has cultivated bacilli from glass plates which were covered with solid culture media and held near the patient; he believes that minute particles remain in suspension in the air and thus infect other individuals. Drying of the sputum voided in the street or other open

places is less significant than was formerly thought, since the sun soon kills the bacilli. Hence the ubiquity of the tubercle bacillus is generally exaggerated, though it is of great importance in poorly ventilated, dark dwelling-rooms, hospitals, cloisters, hotels or prisons, which are frequented by victims of tuberculosis. Cornet found the bacilli in the dust of such rooms in large numbers, yet in about 10 per cent. of tuberculous wards no bacilli were found; he experimentally demonstrated that animals confined in rooms with dried tuberculous sputum readily acquired tuberculosis. Straus, of Paris, collected the germ from the nose in 31 per cent. of the attendants in tuberculous wards. *The closer the contact* with tuberculous subjects, *the greater is the danger of infection,* as tuberculosis is surely an infection. In crowded prisons, the death-rate is three or four times that of the general community; in Austria, for example, the general mortality is 11 to 15 per cent., and the prison mortality is 40 to 60 per cent. In nursing sisterhoods, the death-rate exceeded 75 per cent. Six cases were observed by the writer, in two years, in one small, closely cloistered community. In Philadelphia, Flick's studies showed that 33 per cent. of infected houses contributed more than one case. In Falkenstein, Germany, and Brompton Hospital, London, acquisition of tuberculosis by attendants is infrequent, while in Paris, Straus puts their mortality at a high figure. In our dispensary service, seven rapidly fatal cases were observed in one family which had no hereditary stigma or previous disease (Harpole). Oral breathing, caused by adenoids, inhalation of coal-dust or stone-dust, syphilis, cancer and leprosy are favoring factors.

Dusting, sweeping or shaking the bed clothes disseminates the virus; there is less danger of infection when the windows and doors are open and when dusting is done with moist cloths rather than with feather-dusters. In hospital wards there are more germs in the air during the cleaning hour than during the other twenty-three hours. However, if bacilli are in the dust, they are not necessarily in the inhaled air. If inhaled, they do not necessarily reach the lungs, for they may be caught in the upper air-passages whose secretions antagonize their development, and are often waved back by the ciliated epithelium of the lower air-passages. Cornet holds that the statement that "every man lives in the environment of tuberculosis" is surely exaggeration. Married persons, because of their intimate association, may contract the disease from each other, as was noted by Ettmüller and Morton, in the seventeenth century; the husband more often conveys infection to the wife than the wife to the husband; in Weber's series of cases, a husband lost four wives, another three wives, and in four other cases two wives contracted tuberculosis. When bacilli are inhaled into the respiratory tract (*a*) no lesions may result, (*b*) the lungs may become diseased, or (*c*) the germs may pass the mucosa of the mouth or bronchi and cause cervical or peribronchial tuberculous adenitis.

2. By the Digestive Tract.—Bacilli may pass the mucosa, espe-

cially in children, and produce the first lesions in the mesenteric glands. Milk is dangerous (Gerlach, Bollinger, Bang and others), though lately attempts have been made to minimize the danger from this source of infection. It was formerly thought that, in cows, a tuberculous mastitis was necessary to milk infection, but Ernst and others demonstrated that milk may be infective from cows with tuberculosis which does not invade the udders. Pigs, fed on tuberculous milk, develop intestinal tuberculosis. Butter may contain tubercle bacilli. Meat is less dangerous, as it is usually well cooked, but tubercles in raw offal frequently infect swine.

3. Direct Inoculation.—Inoculation through the skin, eye, genito-urinary tract, by trauma or operations, is far less significant and will be considered under the various tissues occasionally involved.

Predisposing Factors.—*These are important, but if practically considered, tuberculosis is best prevented and treated as an infection, communicated and acquired by direct personal contact.*

1. Heredity.—(*a*) *Hereditary Predisposition to Tuberculosis.*—A tuberculous family history is obtained in infected individuals in 25 per cent. It is remarkable what statistics can be made to establish, and one might prove by figures a formidable hereditary influence in measles, pneumonia and other common diseases. Kuthri found essentially the same percentage of family tuberculosis in non-tuberculous as in tuberculous cases. Tuberculous parents may infect their children (50 per cent.), as they may infect others, but 97 per cent. escape the disease when removed from their parents. According to Rieffel, 3 per cent. of children acquire tuberculosis when both parents are sound, 22 per cent. when both parents are tuberculous, 10 per cent. when the mother and 7 per cent. when the father is tuberculous. Hereditary predisposition is no obstacle to prevention and treatment.

(*b*) *Congenital Tuberculosis.*—It is thought by Baumgarten that children actually inherit the bacillus, which remains latent for years, and then develops an active tuberculosis. If his views are correct, we are confronted with a difficult problem. Congenital tuberculosis can be acquired only through (i) the spermatozoön, (ii) the ovum or (iii) placenta. The spermatozoön and ovum can not carry bacteria. Placental tuberculosis has been observed clearly in about 22 human cases (according to Warthin and Cowie in only 5), and in slightly over 100 cases in animals (Friedman, 1905). The first indisputable case was that of Schmorl and Birch-Hirschfeld, which was confirmed by inoculation and finding of the bacillus. Usually there is a placental focus before the fœtal blood is infected, but the bacillus may sometimes pass directly to the fœtus. Many experienced observers, like Virchow, have never seen a case. Schmorl and Geipel recently found tuberculosis in nearly 50 per cent. of placentæ of tuberculous mothers.

The arguments against hereditary tuberculosis are (1) that children born with the disease die very early; (2) that tuberculosis in

the first months of life is extremely rare; (3) that the disease seldom remains latent until adult life, as most cases of infantile tuberculosis develop during the period of special growth, namely from the second to third year; (4) in congenital infection, the liver is chiefly diseased because of the nature of its vascular relations to the placenta; in tuberculosis of children, the lungs, glands and other organs are more often diseased,—*i. e.*, the infection is respiratory, and therefore extra-uterine. (5) When removed from their tuberculous parents, children thrive well.

2. Environment.—This covers many points mentioned under Atrium. Environment is but *another name for exposure—or contact-infection.* Dampness and poor drainage, emphasized by Bowditch and Buchannan, are factors which *reduce the physiological resistance.* Trudeau's experiments show that inoculated animals, kept in dark, damp cellars, die, while others, allowed to run at large, outlive the infection. Density of population raises the proportion of tuberculous cases. In recent years the large cities have shown an apparent decrease of tuberculosis, as shown by Edward F. Wells, of Chicago, and Jas. B. Russel, of Glascow. It has also decreased in New York and London. In Massachusetts the total is less by half than it was fifty years ago. The decrease is attributed to more intelligent hygiene. The figures show a decided decrease in Chicago (*v.* Pneumonia); it is possible, in recent years at least, that this is explained by the fact that pneumonia carries off the less resistant of the population, as in Munich the death-rate from tuberculosis was 12 per cent. at a time when typhoid was epidemic; when typhoid was practically abolished, the tuberculosis rate increased to 16 per cent., —*i. e.*, 4 per cent. of those with tuberculosis had previously died from typhoid.

3. Occupation.—Occupations involving work in close, damp rooms, in dust, in *crowded quarters,* particularly when associated with poor food, alcoholism, worry and over-exertion, *lower resistance* and promote tuberculosis when *exposure to infected individuals* exists. Clerks, stenographers or waiters are naturally more easily infected than are those working in the fresh air, for example, cab-drivers. It is rather surprising that miners do not suffer more frequently, but the explanation is the dampness, which prevents the drying of infected sputum.

4. Age.—Cornet's figures show the following death-rate per 10,000 of population: First year 2 to 3, the child being in close contact with the mother. These figures grow smaller, until the lowest figure is reached, namely ½, between the fifth to tenth years; fifteenth to twentieth years, 17 to 19; twentieth to thirtieth years, 32 to 36; thirtieth to fortieth years, 44; fortieth to fiftieth years, 55; fiftieth to sixtieth years, 76; sixtieth to seventieth years, 100; seventieth to eightieth years, 70; over eightieth year, 20. In the first decade of life, glandular, meningeal, osseous and cutaneous tuberculosis are more frequent.

5. Sex.—Between five and twenty years of age, girls are more prone to tuberculosis, because their life is more sedentary. After twenty years males more often contract tuberculosis, because of their greater exposure to infection. Repeated pregnancies and protracted lactations may favor infection by lowering physiological resistance. Skin lesions are more frequent in women; in 68 per cent. lupus occurs in women.

6. Climate.—Warm, dry climates, high altitudes and lack of sudden variations in temperature, generally are inimical to tuberculosis. The French Alps were long free of tuberculosis until crowded by infected subjects, and the same seems true of other localities. Some writers even hold that the chief advantages of many resorts is their sparse population and inaccessibility.

7. Race.—In this country the Irish and negroes are most frequently diseased. The Indians have justly come to dread the "coughing white man," and they die in large numbers from consumption. Infection among Jews is peculiarly infrequent.

8. Individual Predisposition.—This was considered important by the oldest medical writers, who described the winged scapulæ, paralytic thorax and depressed sternum; but poor thoracic and general development is very often an expression of already existing tuberculosis; phthisis often occurs in splendidly developed chests, *exposure to infection* being the most important factor. Tuberculosis confers no immunity to relapses or to new localizations; a lung lesion may heal while a coincident intestinal lesion progresses.

9. Trauma.—This is a somewhat determining factor. Bacilli in the blood may be localized in a bone or joint by trauma; a blow on the head or spine may initiate meningitis in a tuberculous subject; pleurisy or pulmonary tuberculosis may be initiated by injury to the chest, which is thought to awaken latent lesions in the peribronchial glands.

10. Other Factors.—These are *diabetes,* in which 23 to 50 per cent. die of tuberculosis of the lung; *psychoses,* especially of the depressive type, as melancholia; anæmia, which, however, is more often the result than the cause of tuberculosis; inflammation of the mucous membranes, acute processes, such as *heavy colds, measles* and *pertussis,* and less often scarlatina, influenza or diphtheria, may predispose to infection, or they probably more often awaken slumbering glandular tuberculosis by the irritative action of products absorbed from the bronchi or lungs. Enterocolitis in children, or typhoid in adults, may in a similar way awaken or initiate abdominal tuberculosis. *Contracted kidney, cirrhotic liver, sclerosed arteries,* stenosis of the pulmonary artery, aortic aneurysm and narrowed hypoplastic aorta are predisposing factors. As Rokitansky first pointed out, valvular disease of the left heart prevents or checks infection, because it congests the lungs; Bier's treatment of surgical tuberculosis, by inducing congestion through constriction, is based on this point; Birch-Hirschfeld, in 4,359 autopsies, found chronic pul-

monary tuberculosis in 21 per cent., and in 107 autopsies of valvular heart disease, it was present in but 2.8 per cent. Potain, in 54 cases of mitral stenosis, found pulmonary tuberculosis in 16.6 per cent. (see VALVULAR DISEASE OF THE HEART). Syphilis, pleurisy (*q. v.*) and hæmoptysis (*q. v.*) are occasional predisposing factors.

General Histo-pathology of the Tubercle.—The only absolutely *pathognomonic feature of the tubercle is Koch's bacillus,* as similar infective granulomata are seen in strongylus in sheep, in actinomycosis and in syphilis. Baumgarten described the changes following inoculation of the anterior chamber of the eye: (*a*) On the first day the bacilli increase and spread somewhat along the lymph-spaces. (*b*) By the sixth day, the fixed connective-tissue cells proliferate under the stimulus of the tubercle bacillus, which acts as a foreign body; these cells, known as *epithelioid cells,* are large, rounded or polygonal cells with distended nuclei, and sometimes contain tubercle bacilli; in another day or two they show karyokinetic figures; the bacilli multiply and the endothelial cells of the vessels proliferate. The tubercle of this stage is sometimes called the epithelioid tubercle. (*c*) On the tenth or eleventh day the leukocytes emigrate. This inflammatory reaction results from the toxins secreted by the bacillus; at first most of the white cells are polymorphonuclear, many of which degenerate; later they are *mononuclear,* which do not suffer retrogressive changes; the mononuclears crowd the epithelioid cells and dominate the histological picture, especially with complicating trauma (lymphoid tubercle). (*d*) The *reticulum* of the tubercle consists of the stretched fibers of the infected tissue or coagulation necrosis. Tubercles contain no bloodvessels. (*e*) The epithelioid cells increase in size and are often multinucleated. From them or from the extravasated leukocytes, by growth or by fusion, *giant cells* develop; they contain many large, oval, vesicular nuclei and tubercle bacilli; the bacilli may number 50 to a cell in very active processes but are few in chronic tuberculosis; the giant cells are particularly abundant or even may be the chief histological finding in chronic forms, such as lupus, scrofulous glands or fungoid joint-disease; they are few or absent in acute processes, as in miliary tuberculosis; there seems to be a certain antagonism between the nuclei and bacilli, for if the nuclei lie on the margin of the cell, the bacilli are centrally grouped, and if the nuclei lie at one pole of the cell, the bacilli concentrate at the other pole. The cellular inclusion of the bacilli is considered a phagocytosis by Metschnikoff, which Baumgarten, however, denies. The completed miliary tubercle is non-vascular, cellular, millet-seed (miliary) in size, gray in color and translucent; opacity marks the later caseation (*v. i.*). It is the invariable pathological unit of all tuberculous lesions, however extensive, and, more or less, is a continuous process. In the diffused inflammatory tubercle, the diffuse areas result from the fusion of myriads of miliary tubercles (see CASEOUS PNEUMONIA).

The tubercle then undergoes one of two *degenerations,* caseation or

induration: (1) *Caseation* results from degeneration of the tubercle, beginning at its centre, from ischæmia (vascular obliteration) and from coagulation necrosis (action of the bacilli and its toxins). Inoculations with dead bacilli may cause tubercles and even caseation. The area of coagulation necrosis then gradually becomes more yellow and granular, and finally results in an amorphous, homogeneous, cheesy debris. Caseation is a dangerous, destructive degeneration. (2) *Induration,* sclerosis or encapsulation, results from growth of connective tissue around the tubercle or conglomeration of tubercles; it limits the tubercle, is the measure of the resisting power of the surrounding tissue, of the process of bacteriolysis, and is a conservative change. It occurs most frequently in the peritoneum, but also in the lungs and other tissues. *Inflammation,* varying with the tissue involved, may cause other histological findings. Suppuration may result from mixed infection with streptococci, though Koch held that pus may be formed by the tubercle bacillus itself; the ordinary tuberculous "cold abscess" is not really pus, but merely thick, amorphous, granular debris.

Extension.—(1) Tubercles develop at the point of infection and also in the nearest lymph-glands, which permanently or temporarily arrest the process. (2) Infection may occur through the uninjured mucosa or skin, but the process rarely passes beyond the tributary lymph-glands. (3) The later advance of the process occurs interruptedly or gradually, so that it can be traced clearly at autopsy. (4) Extension usually occurs along the lymph vessels and glands.

I. Tuberculosis Miliaris Acuta.—Definition.—An acute general, sometimes almost universal, embolic dissemination of the tubercle bacillus in great numbers, by the blood or lymph stream. It results pathologically in the wide-spread formation of miliary tubercles in various tissues, and it produces, clinically, acute toxæmic (typhoid-like), pulmonary, meningitic or other symptoms, and is invariably and rapidly fatal.

Etiology.—1. Development.—Acute miliary tuberculosis *always develops from a tuberculous focus.* This is found in direct ratio to the carefulness of dissection; von Buhl, who first fully described the affection (1856), recognized that miliary dissemination occurred by absorption from old tuberculous foci, just as pyæmia results from local suppuration; and he found the primary focus in 90 per cent. of cases. It may be small or even miscroscopic and may easily escape detection but Simmonds found the initial lesion in 100 per cent. As Cornet remarks, it is curious that miliary tuberculosis occurs from foci so small as to be easily overlooked, and that large tuberculous lesions so seldom result in miliary dissemination. A description of all primary foci would be an enumeration of every possible localization of tuberculosis.

2. Lesion.—*Pulmonary tuberculosis* is the most frequent lesion and causes over 50 per cent. (Rühle, Orth, Litten) of cases of acute

miliary tuberculosis. Glandular lesions rank next, and articular, osseous, genito-urinary and serous-membrane tuberculosis cause most of the balance. The more the tubercle is indurated, the less is the danger of local or general dissemination. Acute generalization only follows the introduction into the circulation of *large numbers of bacilli,* for they do not multiply in the blood. A few bacilli reaching the circulation cause a solitary tubercle in some organ—for example, in the brain. Repeated ruptures of moderate numbers of bacilli lead to the so-called chronic miliary tuberculosis (*v. i.*).

3. Mode of Access of Bacilli into Blood Stream.—Sir Astley Cooper, in 1798, found tubercles in the thoracic duct, and this was later confirmed by Ponfick, Weigert, Koch and Meisels. In 1877, Weigert demonstrated that *tubercles invade the vessel walls,* which Rokitansky had declared immune. Tuberculosis reaching a vessel usually causes its gradual obliteration and in this way localizes the process, but in some cases bacilli reach the lumen of the vessel and are distributed in the direction of its current. Two views are held as to the nature of the involvement of the vessels: (*a*) That of Weigert, who thought the most frequent process was periangitis, by which a tubercle adhered to and emptied into a vein; and (*b*) that of Benda, who contended that the common lesion was endangitis, by which tubercle bacilli lodged in the intima, and, grouping there, further disseminated the infection. One of the most common methods is *periadenitis of a tuberculous bronchial gland,* its adhesion to a pulmonary vein and final rupture into it. Tuberculous lesions may rupture into the vena azygos, pulmonary artery, aorta, thoracic duct, jugular or other veins, into the cavæ or into the brain sinuses. From the pleuræ and peritoneum infection may travel directly to the thoracic duct.

4. Organs Involved.—(*a*) Rupture into a pulmonary vein floods the *arterial circulation* with bacilli, whence miliary deposits occur in the brain, choroid, liver, spleen, kidneys, myocardium near pulmonary artery, bones, serous and mucous membranes and in the blood-vessels themselves. (*b*) Rupture into a portal venous radicle affects chiefly the liver. (*c*) Into the pulmonary artery, the lungs and (*d*) into a peripheral artery, the tissue supplied by it.

5. Predisposing Factors.—Though miliary dissemination may occur at any age, it is most frequent *in children at the developmental period,* especially between one and four years of age, because the circulation is faster, metabolism is quicker, thrombosis of the vessels is less frequent, tissue reaction is less marked and the lymph-vessels and spaces are wider. *Depleting factors,* pregnancy, the puerperium, trauma, bone operations, *measles, pertussis,* mixed infections and the spring and summer months, are somewhat predisposing causes.

Symptoms.—The clinical picture varies greatly, depending on (*a*) the quality (virulence) and quantity of the bacilli; (*b*) the toxins thrown into the circulation from the primary ruptured focus; (*c*) the toxins developed elsewhere by the bacilli; and (*d*) the localization of the tubercles formed. Naturally *toxæmic precede local symptoms.*

PLATE VII

Retinal Findings in the More Important Diseases.

1. Retinitis diabetica. 2. Atrophy (and pigmentation) of retina and optic nerve. 3. Choked disk in brain tumor. 4. Optic neuritis in meningitis. 5. Medullated nerve fibers (normal).

Types.—Waller (1845) distinguished the following forms:

1. Typhoid Type.—The febrile onset may be gradual or sudden, with or without chills. The patient often exhibits little beyond a marked toxæmia. The typhoid type is due to *greater toxæmia with less local reaction* from the tubercle, and therefore with few local findings. The *status typhosus,* delirium, headache, moderate continuous or weakly remittent fever, enlarged spleen and bronchitis suggest typhoid but the fever is more variable and the pulse runs higher; less frequently roseolæ, meteorism, diarrhœa and intestinal hemorrhage may cause even a closer resemblance to typhoid (*q. v.* for differentiation). Unless the meninges or lungs are involved the diagnostic confusion may be insuperable. Epistaxis is less common; hyperæsthesia and herpes are more common but all symptoms may be deceptive except the typhoid bacillæmia and the Widal reaction in typhoid, as opposed to the tubercle bacillæmia and choroidal tubercles in miliary tuberculosis. Rosenberger recently found tubercle bacilli in the feces. Acute miliary bacillæmia is more common in children than in adults, and runs a course of ten to twenty days, or somewhat longer. Typhoid and miliary tuberculosis have rarely occurred in the same subject (Birch-Hirschfeld, Burkart, Massini, Schütze, and two personal observations).

2. Respiratory Type.—In the respiratory type *the toxæmia* is *rather subordinated by the local miliary eruption. Tachypnœa, dyspnœa* and *cyanosis, without proportionate physical findings* are always suggestive. This variety is unmistakable; the respiratory urgency and the rapid pulse, without heart lesion and with a normal or *tympanitic pulmonary resonance* declare the diagnosis. The following forms are less characteristic: *Pneumonia* may be closely simulated by the fever, chills, pain, cough, rusty sputum, increased breathing, dyspnœa, cyanosis, or even by tubular breathing (in children). *Capillary bronchitis* (bronchopneumonia) should be differentiated after recent measles, or after pertussis with patches of atelectasis. Diagnosis should be made from senile bronchitis and emphysema in the aged, in whom this type is particularly frequent without fever. Tubercle bacilli in the sputum or urine may occur accidentally. The course averages three to four weeks.

3. Meningeal Type.—In the typical meningeal form, tuberculous meningitis appears as the only apparent trouble, whereas it is but a part of a miliary dissemination. Its symptoms will be detailed later under a separate topic (page 304). Meningitis occurs in 80 per cent. of cases of miliary tuberculosis. The increased respiration, with occasional deep breathing and increased irregular heart action, are suggestive (Rühle). The choroid findings, the results of lumbar puncture and Kernig's sign are corroborative. Lumbar puncture often shows the presence of bacilli. The symptoms may be clearly organic, and yet, in children who are especially prone to this form of miliary tuberculosis, toxæmia may very closely simulate actual inflammation, as in Henoch's cases, where only brain œdema was found instead of the anticipated meningitis.

4. Other Types.—These are less frequent, but their mere mention may show the great and almost necessary liability to diagnostic error in atypical forms. They are: The *latent type* (Leudet), marked only by catarrhal symptoms, and often called grippal; the *ambulant type,* as in Grawitz's case, where an apparently healthy soldier died after four days of indisposition; nephritic; arthritic; apoplectiform, delirium tremens, pharyngeolaryngeal, suffocative or asthmatic type; fulminant type, where the patient dies in a few hours, or in his sleep. In some cases, sepsis, miliary carcinosis, uræmia or the cyanosis of nitro-benzol poisoning may be suggested.

Individual Symptoms.—1. Temperature.—Continuous fever may be observed in very acute cases with wide-spread localization, but as a rule it is lower than that of typhoid. *Remittent or intermittent* fever is more common; miliary tuberculosis following pulmonary phthisis may change neither the hectic fever of the latter nor its euphoria. The *typus inversus* may occur, though it usually lasts but a few days (Bruniche). In 17.5 per cent. of cases (Rheinhold) the course is afebrile; involvement of the brain or meninges lowers the temperature or renders it variable; hemorrhages, vomiting or diarrhœa may depress the fever (Litten); low temperatures are common in the aged, in the ambulant and in bronchitic or cardiac types.

2. Circulation.—(*a*) The *pulse is faster than is proportionate to the degree of fever,* being 120 in the early stages and 140 to 160 later; the blood-pressure is low but dicrotism is infrequent; a pulse under 100 is very rare and nearly always occurs in old people or in cases of meningitic localization. (*b*) The heart is somewhat dilated and an apical systolic murmur, with accentuation of the second pulmonic sound, is fairly frequent. A pericardial rub is heard on rare occasions, due to roughening of the serosa by the miliary eruption (Litten). (*c*) Tubercle bacilli were first found at autopsy in the blood by Weichselbaum and Meisels, and clinically by Lustig and Rütimeyer, Sticker, Ulacacis and Doutrelepoint. In the death agony from lung tuberculosis, bacilli may enter the blood. (*d*) The *leukocytes* are usually not increased.

3. Respiration.—Early symptoms are frequent. (*a*) There is very often a severe *continuous cough,* which is *always a suspicious symptom.* It is due to the toxins or to irritation of the vagus twigs by abundant miliary nodes in the lung; it causes considerable muscular pain, but little voiding of sputum and ceases later if the brain becomes involved. (*b*) The *breathing* is *increased* to 40 or 60 in adults, and to 80 or 90 in children. (*c*) *Dyspnœa* and *cyanosis* develop from irritation of the vagus (Rühle) or from diaphragmatic involvement (Rheinhold). The *tachypnœa, dyspnœa and cyanosis are peculiarly significant* diagnostically in that they have no adequate explanation in the physical findings. (*d*) The sputum is scant, foamy white and mucous, in acute cases. It seldom contains dots of blood, and is rarely rusty without the presence of complicating pneumonia. It may be more abundant in subacute cases. (*e*) The

physical findings are not as marked as the urgent respiratory symptoms would indicate. A *tympanitic note* is very frequent, resulting from relaxation of the lung, or from acute emphysema from coughing. Dulness only results from older tuberculous foci in the lung, or from complicating atelectasis or consolidation. Auscultation often reveals *fine rales,* which are especially suggestive when they occur over the upper chest, where stasis is uncommon. Jürgensen notes a fine pleural rub, which is due, he thinks, to pleural tubercles. A terminal pulmonary œdema is frequent. At autopsy innumerable translucent nodes are scattered through the lung, and are most apparent on oblique illumination.

4. Nervous System.—Nervous symptoms may predominate, and may result from toxæmia (like the symptoms following tuberculin injection), or from central organic changes. (*a*) The *toxæmic symptoms* are headache, dulness, vertigo and tinnitus; these are followed by delirium, muscular adynamia and tremor, and result in stupor, which is broken by fits of delirium, and finally in coma. In rare instances coma may develop early in an apoplectiform fashion, or the sensorium may be normal. (*b*) *Meningitic symptoms* may develop, such as headache, variable pulse and respiration, retraction of head and spinal tenderness and rigidity, narrow and then widened pupils, strabismus, paralysis of other cranial nerves, Kernig's sign, clonic contractions, convulsions and coma. The findings at autopsy are those of tuberculous meningitis (*q.v.*).

5. Digestive System.—The tongue is coated and there is anorexia. Nausea and vomiting are usually meningitic. There is usually constipation and the abdomen is normal. Diarrhœa is infrequent, but bloody stools may occur with tympany. Ascites is infrequent in miliary tuberculosis of the peritoneum (as distinguished from tuberculous peritonitis). Litten once heard a friction rub over the liver. Miliary tubercles may occur in the stomach and bowels, most frequently in children.

6. Choroidal Tubercles.—These are said to occur in 16 (Rheinhold) to 75 per cent. (Litten) of the cases. They are most numerous in very generalized tuberculosis. They occur singly or in large numbers, usually in both eyes as whitish-yellow protuberances with irregular washed borders. They become quite large, for they cause so much atrophy of the retinal pigment layer which covers them that they can be seen through it. Daily examination for them is necessary, as they frequently develop in half a day. They are seen only after one or several weeks. (See Plate VIII, Fig. 6.)

7. Other Symptoms.—Emaciation is often pronounced; one of Wunderlich's cases was reduced to one-quarter of his ordinary weight. The *spleen is enlarged* in 70 per cent. (Litten), but is usually smaller than it is in typhoid, except in children, where it may assume great dimensions. Collin observed swelling of the lymphatic glands, which in one instance became enormous. The urine is concentrated and dark, from the fever and from sweating, may contain albumin or pep-

tone, and quite often shows the diazo reaction. Rosenstein observed anuria, especially in children. Hemorrhages into the gums, nose, retinæ and intestines are infrequent and possibly result from mixed infections. The skin is pale and cyanotic; profuse sweats, and therefore sudamina, are frequent. Herpes may be observed, as well as rose spots, miliary tubercles in the skin, petechiæ, erythema nodosum and terminal œdema.

Remissions may occur for a time, even for a month or two, as the toxæmia may subside, while the tubercles continue to develop. The fall of the fever resulting from brain implication may be deceptive. As a rule the course is acute, even stormy or peracute, and death intervenes from exhaustion, meningitis, heart weakness, dyspnœa or pulmonary œdema. Severe infection is fatal in two or three weeks, moderate infection in one to two months. Chronic miliary tuberculosis is rare. Recovery is almost impossible, but Waller, Wunderlich, Burkart and Spengler believed that it exceptionally occurred, and Stricker saw recovery of a case in which the diagnosis seemed incontestable, as he found bacilli in the blood. Therapy is useless. The symptoms should be treated expectantly, as in typhoid, pneumonia and meningitis.

II. Tuberculous Meningitis.—**Etiology.**—Most cases occur between the *second and tenth years,* some cases develop in adolescence and few late in life; at least three times as many cases occur in children as in adults. Tuberculosis in children is usually generalized, and miliary tuberculosis is in 80 per cent. associated with meningeal disease. Tuberculous meningitis is almost always secondary, and follows pulmonary or glandular tuberculosis, less frequently joint disease, genito-urinary, brain and other localizations. Extension by contiguity from the nose, eye or spinal cord is possible. Strümpell and Leube believe that infection may creep from the pleura, along the nerve sheaths to the spinal cord, whence it reaches the brain. *Measles, whooping-cough,* typhoid or pneumonia may seemingly arouse latent glandular or other tuberculous foci. Trauma is not a frequent cause but is of distinct clinical and medico-legal importance. In the case of a baggage man thrown upon his occiput by the sudden stopping of the train, an acute tuberculous meningitis developed, which resulted in death in three weeks. The autopsy disclosed an apical focus, from which the bacilli infected the injured brain. In one instance, meningitis followed an anæsthetic, given to examine a tender spine, and was fatal within a week.

Pathology.—The diagnosis is made *macroscopically* in the majority of cases, although in some instances the changes are slight. The exudate is gray, yellow or green, gelatinous, sticky, purulent or serous and covers the base (basilar meningitis) from the chiasm to the medulla. Tiny tubercles are found on the basal nerves and deep in the fissure along the Sylvian artery where "they look like lice eggs on a hair." Careful search is sometimes necessary for their detec-

tion. They are most clearly seen in the translucent arachnoid, and on superficial examination look like minute air bubbles. They are seen quite frequently over the convexity and may cause adhesions between the two hemispheres. The ventricular fluid is increased in 80 per cent. of cases, and usually measures a few ounces; tubercles and inflammation of the ependyma are not uncommon; the external and internal communications of the ventricles may be obliterated. The substance of the brain shows ischæmic or encephalitic softening. The cerebrospinal fluid is usually increased and is clear or cloudy. In the cord the exudate may be a quarter of an inch in thickness, and is largely disposed over the posterior part of cord because of the patient's dorsal decubitus. The cord membranes are almost regularly affected, and sometimes appear granular, as "though sown with fine sand." Tubercles in the substance of the cord are not considered common, but Wartmann found them in 87 per cent. of his cases. The cranial and spinal nerve sheaths are bulging. *Microscopically,* exudation is found. The nodes are proven to be tubercles; bacilli are found in them and in the cerebrospinal fluid. The microscope differentiates between tuberculous inflammation and tuberculosis of the meninges (without inflammation), and again in some cases proves the existence of tuberculous meningitis not visible to the naked eye. Neuritis of the cranial and spinal nerves is constant. The optic, oculomotor and facial nerves are the most often affected. In the cortex, base and ependyma of the brain, the usual histopathology of tubercle is seen, with tuberculous endarteritis and periarteritis, small encephalitic and myelitic foci in practically every case, swelling of the neuroglia and degeneration of the nerve cells and especially of the axis cylinders.

Symptoms.—*Prodromes* are more prominent in children than in adults; fever, emaciation and anæmia are due to preëxisting lung or glandular tuberculosis. Irritability, change in disposition, tendency to cry, inability in children to play or in adults to work, unrest, insomnia, headache, nausea and constipation may develop in a week or two or in one to three months. Trauma, measles or pertussis may be the apparent cause. The outbreak develops with much the same symptoms as those described under the epidemic form, but in 86 per cent. (Koplik) they appear more gradually, so that the classical description comprises *three stages:* (*a*) The *stage of irritation,* with headache, vomiting, remittent fever reaching 102° in the evening, the *cri hydrencéphalique* of Coindet, night terrors, pain and rigidity in the neck and spine, small irregular pupils, delirium, grinding of the teeth, hyperæsthesia, rigidity of the limbs and Kernig's sign (in 22 of Koplik's 82 cases); (*b*) the *stage of brain pressure,* with paralytic and irritative conditions in the eye muscles, as ptosis or strabismus; with paralysis of other cranial nerves or of the extremities, as monoplegia or hemiplegia from involvement of the cortex, and very frequently aphasia and dulness; with general or Jacksonian convulsions, slow, irregular and quite variable pulse;

retracted abdomen, constipation, continued vomiting; sighing respiration; and very frequently with optic neuritis; and (*c*) the *stage of paralysis,* in which the vomiting and often the headache cease, the sensorium is dulled, absolute coma develops, the pulse becomes rapid and vacillates in rate and rhythm. Biot's alternating apnœa and hyperpnœa or Cheyne-Stokes' breathing appears, emaciation is extreme, the pupils are wide and immobile, the eyeballs roll upward, and a typhoid state with dry tongue or abdominal distention exists. Temporary remissions of days, weeks, or even a year are occasional, in which the pulse remains irregular, but the patient lapses again into coma, the fever rises very high (107°) or falls low (even to 93°), and death occurs in several weeks from cardiac or respiratory failure, dysphagia, inanition or hypostasis. This division into stages is not wholly satisfactory, because the symptoms of one stage may merge into those of another but the progression of symptoms is somewhat as described.

Tubercles in the choroid are rare clinically, being found by Garlick in 4 and Krämer in 13.6 per cent. of cases. Heinzell did not find them in 41 cases. At postmortem examination Litten found them in about 75 per cent. (See Plate VIII.) *Leukocytosis* is absent except during the agony or from mixed infection. The spleen is enlarged in 33 per cent.

Many variations exist. The process may be wholly *latent,* being masked by miliary tuberculosis (or meningeal symptoms may dominate when the greatest pathological changes are in the lungs). Coma may be sudden and the disease may run a short course, resembling apoplexy, especially in adults; in these cases the hemiplegia probably results from changes in the arteries. It may appear to be a psychosis. In adults the course is more rapid, the headache and delirium more marked and the convexity more frequently involved than in children.

Death is usual, but rare recoveries, numbering about twenty, are recorded, even after bacilli were found by lumbar puncture or after choroidal tubercles were demonstrated. (See Cerebrospinal Fever for Differentiation and Treatment.)

III. Tuberculosis of the Lungs (Phthisis, Consumption).—The lungs are involved with great frequency, not because they are peculiarly susceptible, but because infection by inhalation is the most frequent cause of tuberculosis. Besides this aërogenous infection, secondary infection by the bloodvessels, as in miliary tuberculosis (hæmatogenous infection), and by the lymph vessels (lymphogenous infection), is possible.

Pathology.—The tubercle bacillus localizes in the bronchi (tuberculous lymphangitis), bronchioles or alveoli (tuberculous pneumonia), and causes a tubercle as described in the general pathology. There is cellular hyperplasia of the alveolar, epithelial and endothelial cells, and fibrinous inflammation develops. Isolated tubercles by fusion cause the conglomerate tubercle.

PLATE VIII

6. Tubercles in choroid. 7. Syphilitic choroidoretinitis. 8. Albuminuric retinitis. 9. Hemorrhages in pernicious anæmia. (Figures 1 to 3 are from Jaeger and the balance are from Sahli.)

Extension.—(*a*) *Lymphatic extension* is the most common form. In young individuals the lymph paths are wider and absorption generally is greater, so that intoxication and bacillary migration are more active. Those who have lost weight, as convalescing typhoids, show greater absorbing powers; absorption is also greater after trauma, pneumonia, etc. Bacilli may pass the bronchi and reach the glands at the hilum, which in turn may infect the blood or break into the lung. Extension is usually along the lymph vessels in the bronchial walls (lymphangitis tuberculosa). (*b*) Extension by the *air passages* is quite common; when a tubercle ruptures into the bronchi, its contents may be coughed up or waved upward by the ciliated epithelia if they are intact; on the other hand, a deep breath may aspirate, or coughing may impel, infected particles into sound areas, where they develop into new foci; much depends on whether the bacilli are living or dead; if living, an active tuberculosis develops; if dead, a pneumonia (lobar or lobular) is more common. (*c*) Extension by the *bloodvessels* may cause a local miliary dissemination in the lungs or in the entire body. Preliminary to the clinical description in which special pathological features will be fully described, it may be said that some caseated areas fuse into large infiltrations; some may encapsulate or calcify and thus effect a temporary arrest or a permanent recovery; other caseated areas soften, forming ulcers and cavities.

Symptoms.—A clinical description covering all types is impossible, as the course may be galloping or extremely chronic; one form is marked pathologically by caseation, another by ulceration; one symptom, such as hæmoptysis, may indicate the type and may be the first and chief symptom; in some persons the symptoms suggest other diseases and the physical findings are indeterminate or lacking; in others the physical findings are remarkably dissociated from the usual symptoms. In the following description, the symptoms and signs usually found will be covered first and the special types will be dealt with afterward:

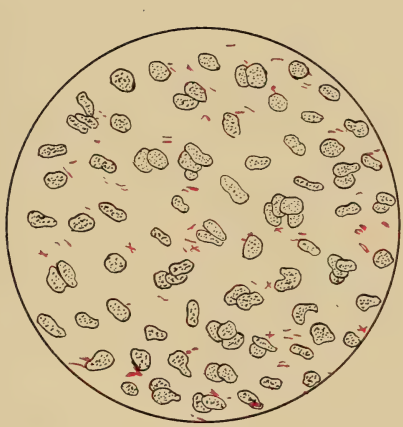
1. Local Respiratory Symptoms.—These are usually the first noted, and may only appear months after the primary focus has begun to spread; they are due to the local tubercle and its toxins.

(*a*) *Cough.*—Cough is one of the first symptoms and is *the most constant symptom;* it is usually present from the incipiency to the close of phthisis. It is due to *toxins irritating the vagus* twigs, which irritation is referred to the medulla, from which issue muscular contractions, with the glottis closed in order to evacuate the slight secretion; as shown by Nothnagel, Stärk, Kohts and Reyher, the sensitive interarytenoid space and tracheal bifurcation initiate this reflex. Less often coughing is due to pleurisy, catarrh of the pharynx, enlarged bronchial glands or laryngitis. It is slight at first, and has no peculiar characteristics, other than being dry, difficult and paroxysmal. Later it becomes more frequent but less difficult as the secretion increases. In some cases it is most severe in the

morning, the secretion having accumulated over night, while in others it is most severe at night, for the lungs become congested by the dorsal decubitus; saliva reaching the larynx during the night, may cause a sudden explosive cough. It may be provoked by dust, smoke, variations in temperature, opening windows or getting into the cool bed, by exertion, laughing or taking alcohol. It varies with the individual, being more marked in neurotics and less conspicuous in the phlegmatic, the aged or the insane. When quite obstinate it may constantly invoke vomiting and lead to great emaciation.

(*b*) *Sputum.*—The sputum is first *caused by the toxins*, and later by the *secretion* from the tuberculous focus. At first it is glairy and contains sago-like bodies composed of alveolar cells transformed into a myelin substance. It later becomes yellow from admixture of pus, yellow, green or red from saprophytic organisms, and blood may give it a red, rusty, flesh-like or chocolate hue. Other bacteria and saprophytes are frequent (*v.* Bacteriology). It tastes sweet and smells slightly fœtid. Its amount varies from a few globules to over a pint. It is most abundant in cavities; it stagnates before death, because of the patient's muscular weakness; women and children are prone to swallow the sputum. *Nummular sputum* from cavities is flat, "coin-like," greenish-gray, extremely abundant and sinks in water, as it is airless; it occurs in other than tuberculous cavities, as in bronchiectasis. The *only characteristic* of the sputum is the *tubercle bacillus* (*v. s.*), which is found only in tuberculosis; it may be seen before the advent of any physical finding, but in other cases it must be sought repeatedly, as it appears *only when ulceration communicating with a bronchus*, develops. The tubercle bacillus is found in only one-third of the cases of incipient tuberculosis. If the patient expectorates into water a crumbling precipitate, rich in bacilli, is obtained (Virchow). *Elastic fibers* are found in 70 to 90 per cent. and originate from the vessels or parenchyma of the lung. The elastic fibers from the vessels are elongated and twisted, and sometimes considerable shreds of the vessel-wall are seen; elastic fibers from the bronchi are also long and twining; those from the lung substance often show its alveolar arrangement. Elastic fibers can often be located by flattening the sputum between two plates of glass and picking out the yellowish areas for microscopic examination. Treatment with caustic potash and centrifuging, facilitate their detection when they are scanty. They merely denote a destructive process, hence are also found in abscess, gangrene, infarction and pneumonia. Detritus, pus cells, red cells, alveolar, bronchial and buccal epithelium, myelin droplets and pigment, are also found. The so-called lung calculi are sometimes voided; they are calcified particles from old areas of caseation, from bronchial obstruction, or from peribronchial lymph glands; from the glands large calcareous masses may cause fatal obstruction of the trachea, or suffocation may be averted only by immediate low tracheotomy. (See Plate IX for tubercle bacilli and elastic fibers.)

PLATE IX



Tubercle Bacilli and Elastic Fibers.

(*c*) *Hæmoptysis.*—Hæmoptysis (hæmoptoë, spitting of blood) occurs in a variable percentage. Prior saw it in 37 per cent. of 1,000 cases; others place it at 66, 80 or even 90, per cent.; it is more frequent in adult males than in females and is rare before puberty. (*a*) *Early hæmoptysis* is due to *erosion of a small vessel* by tubercles in its perivascular lymph sheath. The toxins usually prevent extensive early hemorrhage by causing coagulation in the vessels in and near the affected lung area. Hæmoptysis is the first symptom in 4 per cent. of phthisis cases and in 16 per cent. the only symptom, the patient never having future trouble; in most cases, however, other symptoms develop; bacilli and elastic fibers may be seen at the time of bleeding. Reiche recently corroborated the old statement that early hæmoptysis is not unfavorable. The bleeding develops in quietude in 66 per cent. of cases but may be induced by coughing, laughing, exertion or excitement. Often without cause, it comes on suddenly with a warm, salty taste in the mouth and the evacuation of a dram or more of bright frothy blood, clear or mixed with mucus; small particles of darker blood are brought up, for a few days, after which it disappears; in 4,125 of the Brompton Hospital cases, 69 per cent. evacuated less than half an ounce. Bubbling rales may be heard on the side of the lesion, and moulds of the bronchioles may be voided. F. Stricker collated 900 cases of hæmoptysis in the German army among 1,728,505 soldiers; in 480 cases there was no apparent cause and of these 87 per cent. were tuberculous; of 43 cases brought on by exertion, 74 per cent. were tuberculous; and of 24 cases due to trauma, half were tuberculous. The occurrence of early hemorrhage, before other symptoms of phthisis, led Hippocrates, Boerhaave, Van Swieten, Morton, Graves and Niemeyer, to think that hæmoptysis caused phthisis, while Laennec, Louis and Traube, correctly regarded it as a symptom of already established phthisis. The earlier idea is explained by the fact that in bleeding, blood and bacilli may be aspirated into other parts, in which the process develops anew; fever, rapid pulse and dyspnœa following hæmoptysis are ominous (*v. i.* Acute Forms). (*b*) *Late hæmoptysis* may be slight or profuse. Profuse bleeding is usually due to erosion of blood vessels running across, or in, the walls of cavites, which are frequently aneurysmatic (Rasmussen); Kidd found ruptured aneurysms in 70 of 82 fatal cases; occasionally larger vessels, as the pulmonary artery, are ulcerated. A pint or a quart may sometimes spurt out of the mouth, causing acute anæmia, suffocation or rapid exsanguination. Blood may accumulate in large cavites, never reaching the air passages. Hæmoptysis is usually a sign of tuberculosis but also occurs from other causes (*v.* Diseases of the Lungs).

(*d*) *Dyspnœa.*—This is infrequent, even when the respiratory area is enormously contracted, just as it is absent in pleurisy of *gradual* onset; it is often absent even with great infiltration, great cavity formation or induration of two-thirds of the respiratory space, whereas in sudden miliary tuberculosis the dyspnœa is great. In some cases

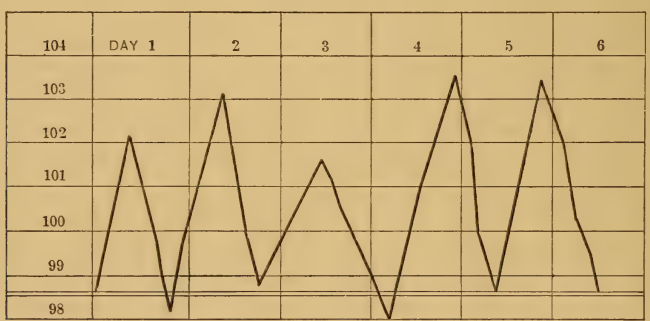
dyspnœa is neurotic, or it may follow pressure of swollen glands on the vagus, anæmia, stagnating secretion, extensive vicarious emphysema or massive pleural adhesions. It is *most often due to the rapid heart action.* In the last stages it may cause orthopnœa and can result from intercurrent bronchitis, pneumonia or pneumothorax. Germain Sée has described a tuberculous pseudoasthma.

(*e*) *Pain.*—Pain is observed in 66 to 75 per cent. of cases. Its location may be apical, basal, diaphragmatic, anteriorly from the clavicle to the third rib or under the scapula. Its character is usually sticking and intermittent, but not intense; the skin is often hyperæsthetic to percussion and palpation. Its cause is *chiefly acute pleurisy,* for lung disease and chronic pleurisy are generally painless; other causes are muscular fatigue from coughing, intercostal neuralgia and less often intercostal neuritis. Pain may be intense in pneumothorax.

2. General Symptoms.—These result *from absorption of the toxins* secreted in the local lesions.

(*a*) *Fever.*—This is present in the great majority of cases and is almost invariable in the early stages. Probably the dominant cause is the tuberculous toxin, whose febrile action is analogous to tuberculin; secondary infections undoubtedly may cause fever, though not necessarily, for the associated streptococci are usually far from virulent. Fever marks the tubercle formation, fusion of foci, advance of the disease, degeneration of the tubercle, and probably secondary infection; its cessation is synchronous with temporary or permanent arrest of the disease, as manifested by improved health, strength and weight. Its height and course are somewhat influenced by the individual; labile individuals possess labile temperature centres (de Renzi); it is easily increased by exercise, excitement or more trivial causes.

Fig. 20.



Hectic fever-curve in phthisis.

Types.—There is no typical curve nor can fever be classified as initial or ulcerative fever. The vesperal hectic is the most common curve, and indicates slow disintegration and absorption of toxins. Intermittent fever may resemble malaria and usually indicates rapid and marked tissue-disintegration; remittent fever most often occurs late

in the clinical course; continuous fever occurs early or in acute consumption, but rarely resembles the typhoid curve; in some cases the fever falls from high to subnormal registers, sometimes with collapse, which was regarded as unfavorable by Rühle; the *typus inversus* with morning exacerbation and vesperal solution is very uncommon. Fever may be absent in cases with old cavities and fibrous induration. Chills have no relation to the fever or its intensity. As fever is indicative of toxæmia, it is often associated with anorexia, emaciation and other symptoms (*v. i.*).

(*b*) *The Blood.*—(i) The red cells rarely number less than 2 million, though Limbeck records a case with but 730,000; anæmia may antedate all other symptoms, whence the frequent confusion of chlorosis and incipient phthisis. Again, anæmia may be inconspicuous or late. In some cases the red cells are increased, notably in individuals prone to hæmoptysis (Mircoli). (ii) The hæmoglobin averages 75 per cent.; it may be decreased more than the number of red cells, therein resembling chlorosis (*chloranæmie tuberculeuse*). (iii) The leukocytes may be normal; in cavity formation (secondary infection) their number may rise to 12,000 or 15,000; they are increased in cases of caseous pneumonia or protracted pyrexia; after injection of tuberculin the eosinophiles show a decided increase. (iv) Tubercle bacilli are seldom found in the blood except in the death agony.

(*c*) *Sweating.*—Night sweats are more important than in any other disease and occur in 90 per cent. of cases (Louis). The most plausible cause is the action of toxæmia on the sweat-centre in the medulla. Heavy coverings sometimes induce sweating. The term "night sweats" is used because nearly all of them occur in the night, chiefly between 2 and 5 A. M. It is more frequent in acute than in chronic phthisis, in younger than in older subjects, when cavities are forming and the fever is variable.

(*d*) *Pulse.*—The rapid pulse is associated with disturbed digestion, reduced muscular strength and depreciated nutrition (*v. i.* COMPLICATIONS, for details).

3. PHYSICAL SIGNS.—In adults, *the apex* of an upper lobe, more often the right than the left, *is first affected* (Laennec). The reasons ascribed are various, such as a poor apex blood-supply, slower lymph current and less expansion,—which are scarcely proven. It is more correctly held that coughing drives infection upward from the bronchi, and that the clavicle or first rib compresses the bronchi passing to the apex and thus lessens its ventilation. The summit of the apex is not invaded first. Birch-Hirschfeld, in early cases which died from causes other than tuberculosis, found that in 24 per cent. the disease began in the fourth or fifth order of bronchi in the upper lobe and that the posterior were affected earlier than the anterior divisions.

(*a*) *Inspection.*—(i) The nutrition may be good, but, later, emaciation is marked. The face is pale, the cheeks hectic, the nose pointed, the eyes hollowed and glistening, the neck thin and the chest is altered in various ways; in some cases it is long and narrow, with

wide interspaces, sharp costal angles, prominence of Louis' angle, atrophy of the scapular muscles and more vertically directed ribs; in others it is flattened anteroposteriorly and the ribs are closely apposed; in still others the funnel-chest may be seen. Laennec recognized that the "paralytic chest" was the result and not the cause of phthisis. Examining the patient in a good light, the following may be noted: (ii) *Deficient expansion* above or below the clavicles, especially when examined from behind and above; in the lower chest, an impaired or absent excursion of the diaphragm. Deficient expansion may be due to pleural adhesions, to atelectasis of the lung resulting from caseous nodes obstructing the bronchi, to a cavity, or to gradual induration of the focus, which is an effort towards encapsulation and recovery. (iii) Distention of the chest may be due to vicarious emphysema, effusive pleurisy or pneumothorax. (iv) The cardiac impulse may be widened by retraction of the left lung away from the heart.

(*b*) *Palpation.*—Palpation shows (i) *decreased expansion,* elicited by standing behind the patient with the thumbs above either clavicle and the fingers below it, or by standing in front of the patient and applying the spread fingers and hands over the anterolateral parts of the thorax. (ii) *Increased vocal fremitus,* obtained when the lung is infiltrated (caseous foci or pneumonia), indurated or is the seat of cavities, all of which increase conduction of the voice-sounds from the bronchi. Superficial show more clearly than deep lesions; thick pleural adhesions or plugging of the bronchi with caseous material decrease the fremitus. (iii) The systolic distention of the pulmonary artery and the second pulmonic closure are better felt when the lung retracts from the heart or when it is infiltrated in its vicinity.

(*c*) *Percussion.*—(i) Incipient infiltration of the apex gives a short percussion note, which later becomes *dull.* According to Grawitz the apex is first involved in 88 per cent. of cases. Dulness is detected in the apex or under the middle of the clavicle, at which level Birch-Hirschfeld found the anatomical beginning of tuberculosis. The apices are best percussed when standing behind the patient, and it should be remembered that the right apex is normally less resonant than the left. The lung tissue lying behind or near, the clavicle can be well tested by gently tapping the clavicle with one finger. To detect slight early alterations in note, percussion should be gentle, symmetrical parts should be compared and the patient should hold his breath in full inspiration; the muscles must be relaxed, and it should be recalled that each patient has his own percussion note, which varies with the individual's muscles, fat and bones. The apex reaches 1 to 2 inches above the clavicle, and behind to the *vertebra prominens.* Piorry, Friedreich and Eichhorst demonstrated that impaired resonance is produced only by consolidation measuring $1\frac{1}{2}$ by $2\frac{1}{2}$ inches in area and within $\frac{3}{4}$ inch of the surface, and that foci of these dimensions lying deeper than 2 inches escape detection. Oesterreich holds that a focus as large as a cherry may cause dulness.

Small, multiple or deep foci give a tympanitic note. The dulness may extend from the clavicle to the second and third costal cartilages, then to 1.5 inches below the apex of the lower lobe at the level of the fifth dorsal vertebra; this area is almost always involved when the upper lobe shows distinct consolidation; extension occurs outward and downward along the interlobar fissure; then it extends to the apex of the upper lobe on the other side. Often the first dulness of consolidation is found behind, in the supra- or interscapular region. (ii) Shrinking, fibrous induration and a thickened pleura give apical or other dulness. Sometimes the lung becomes vicariously emphysematous about an old focus which is thus hidden clinically and pathologically. (iii) A tympanitic or cracked-pot resonance may be obtained over cavities (*v. i.*).

(*d*) *Auscultation.*—This is *the most important aid,* next to the microscopic examination of the sputum. (i) The vesicular murmur, especially at the apex, is altered,—for detection of which the patient is best examined without deep breathing or coughing; the jerky *"cog-wheel" breathing,* described by Jackson (1833), may be due to small tubercles in the bronchi, somewhat stenosing their lumina; it is only suggestive and occurs in normal individuals in forced, muscular breathing; Potain considers it due to pounding of the heart on the lung during inspiration. The breathing may be *wavy, weak, indeterminate* or *puerile.* A rude, rough inspiration may be heard, which is due to peribronchial tubercles compressing the bronchioles. The *expiration* is frequently *prolonged, rougher* and *higher pitched,* in apical "catarrh"; it is long, sharp and bronchial, in older foci. It must be remembered that what seems clinically to be catarrh is often consolidation pathologically. The breath-sounds are physiologically higher pitched on the right than on the left side. (ii) *Bronchial breathing* is caused by various consolidations (tuberculous, pneumonic, neoplastic or gangrenous) and by cavities, tuberculous and otherwise. Bronchial breathing of moderate degree is physiological in the interscapular region, but is considerably less intense than that of consolidation. (iii) *Rales* are rather an evidence of infiltration (consolidation) than of "catarrh." If they are heard in the apex, the presence of tuberculosis should be suspected, though they may occasionally occur there physiologically. The vibrations of the accessory muscles of respiration, when the patient takes a long forcible breath, may be confused with rales, but are easily differentiated after a little experience. Rales may not be heard unless the patient breathes deeply or coughs; auscultation should be made before, and then after, coughing noiselessly and inspiring with the least muscular force. In incipient consolidation, small crackling crepitant rales are heard over the alveoli and smaller bronchioles, at the end of inspiration; they later become larger, ringing and resonant, which proves that the alveoli and bronchioles have become airless over a considerable area, or that softening or formation of cavities is in progress; scattered, clear, large, crackling rales are heard over small cavities in the apex or in

the smaller bronchi, and indicate a viscid secretion. Clicking rales are generally attributed to softening; they may occur near the heart in the systole, as the heart beats upon an adjacent infiltration. Gurgling and consonating rales suggest cavity or bronchiectasis. In emphysematous patients, whose chests exhibit sibilant and bubbling rales, diagnosis of developing tuberculosis may be most difficult. (iv) *Bronchophony* (a bronchial quality imparted to the voice-sounds) is heard when the patient counts or speaks during auscultation; it indicates infiltration or cavity formation but is absent when the bronchi are plugged. *Ægophony* is merely a higher grade of bronchophony and occurs as Skoda demonstrated, in cases of consolidation, in medium-sized cavities and above pleural exudates. (v) *Pleural friction* results from fibrinous pleurisy or rough tubercles in the pleura. (vi) A systolic bruit over the subclavian artery is probably due to pleural adhesions to the artery.

(*e*) *Roentgen Rays.*—Shadows may be due to consolidation, thick-walled cavities, calcified foci, gangrene, pleural callus or pleural exudate. The excursion of the diaphragm can be followed; shadows toward the apices, due to the muscles, may be readily eliminated by moving the arms; the results are often confusing.

Summary.—The findings of inspection, palpation, percussion and auscultation may now be grouped together, because they are found together clinically, one part showing the signs of consolidation, another those of cavity formation or of induration. (*a*) The *signs of consolidation* are decreased expansion, increased vocal fremitus, initial tympany followed by dulness, high-pitched expiration, then distinct bronchial breathing, crepitant rales and bronchophony. (*b*) The *signs of induration* are retraction or lagging behind of the fibrous area on breathing, increased vocal fremitus, dulness, usually large rales, bronchial breathing and bronchophony. (*c*) The *signs of cavity formation* are: (i) Increased vocal fremitus, if it is near the surface; (ii) tympanitic note, which may increase in intensity when the cavity is percussed with the mouth open (Wintrich's change of note), when it is percussed during inspiration (Friedreich's change of note), or when it is percussed with the patient in the sitting posture (Gerhardt's change); the note may be metallic; the cracked-pot note, Laennec's *bruit de pot fêlé,* is obtained in cases of superficial cavities measuring $2\frac{1}{2}$ inches with thin walls, which communicate with an open bronchus by a small opening; variation in the percussion note from accumulation (dulness), or emptying of secretion (tympany), is most suggestive of cavity formation; percussion may cause the patient to cough and void much sputum; (iii) the breathing, which is loudly bronchial, or if the cavity is quite large, metallic, amphoric or metamorphosing; metamorphosing breathing is breathing which begins normally and suddenly becomes bronchial or *vice versa* (Seitz); (iv) bronchophony and ægophony; (v) large, resonant rales, which are the most frequent and important finding; metallic rales in the apices are most suggestive of cavities; they may acquire an amphoric character in very large,

thin-walled excavations; (vi) nummular sputum; (vii) systolic murmurs, which are sometimes booming or metallic, and are due to propagation of the heart tones; they are occasionally heard at some distance from the patient.

Cavities are due to the breaking down of caseous areas and are usually connected with the bronchi. Their evacuation into the bronchi is partly favorable, in that the infective material is evacuated; partly unfavorable, because it opens up avenues for infection by aspiration. Once formed, they generally remain or increase; they may leave fistulous cicatrices (Laennec's *cicatrices fistuleuses*); if they are held by adhesions the cavities remain, contracting cicatrization being impossible; there is always danger of their further extension by secondary infection, or of gangrene. The cavity has a well-marked limiting membrane, from which pus, nummular sputum, elastic fibers, and grayish granular detritus composed of masses of tubercle bacilli, are voided; the cavities are frequently multilocular, may heal on one side and extend on another, and sometimes occupy an entire lobe or even an entire lung. They may rupture into the pleura, pericardium or bloodvessels; in one instance the author saw a cavity rupture into the mammary gland, resulting in diffuse subcutaneous emphysema.

Stages of Pulmonary Tuberculosis.—Tuberculosis usually develops for six to eighteen months before any symptoms appear.

1. Phthisis Incipiens.—In incipient phthisis the general and local symptoms appear as above described, with fever, anæmia and rapid pulse. The modes of onset are various. The most frequent picture is that of a patient with an obstinate cold or a "grippe" which does not resolve; the sputum may show tubercle bacilli and the apex some rales. In others, disordered digestion and a rapid pulse awaken suspicion. The voice may be lost, passing as a simple laryngitis. There may be only a moderate depreciation of health and, in young girls, a chlorosis may be hastily diagnosticated. Malaria is a common escape in explaining an indeterminate fever. Tuberculosis may appear in other more directly suggestive forms, such as hæmoptysis, pleurisy or cervical adenitis. The physical findings are uncertain or absent.

2. Phthisis Confirmata.—The appearance of distinct physical findings indicates not incipient phthisis, but confirmed established phthisis.

3. Phthisis Consummata.—This is advanced consumption, with marked hectic fever, cavity formation and emaciation.

Course.—The average case of "chronic ulcerative phthisis," with the symptoms and signs already described, stands midway between the following very acute and extremely chronic groups:

1. Acute, Galloping or Florid Consumption.—This type constitutes 10 per cent. of phthisis and probably always develops from some old focus, such as tuberculosis of the bronchial glands, or from a small cavity which by intrabronchial rupture disseminates the

process; possibly the *virulence of the germ* is greater in these cases; pregnancy, diabetes, alcoholism, *measles, whooping-cough,* influenza or trauma, may be the immediate cause.

(*a*) *Acute Pneumonic Form.*—The acute pneumonic form is acute caseous pneumonia. The term is employed because of the great clinical resemblance to, and confusion with, acute lobar pneumonia; but it must be remembered that genuine lobar pneumonia does not caseate, and that this form is not synonymous with tuberculosis complicated by acute pneumococcic pneumonia, which, in this association, runs its usual course. Acute caseous pneumonia begins in an individual in seemingly perfect health, or perhaps in one whose health is somewhat but not suggestively reduced. An initial rigor is fairly common, or the spitting of blood may be the first symptom. There is often herpes, cough, pain in the side, rapid breathing and sudden fever, which frequently exactly imitate the onset of genuine pneumonia. The resemblance may persist but often certain variations arouse suspicion. The fever is remittent or intermittent, and more variable than in lobar pneumonia; dyspnœa and cyanosis are less frequent than a gradually increasing pallor; sweating is usually copious. In a few days dulness prevails over a lower lobe but much more often over an upper lobe or over an entire lung. The breathing, which is at first indistinct, may remain so (Traube), but quite often becomes bronchial and is associated with crepitant and coarse mucous rales. The sputum is often typically pneumonic, viscid and rusty, or of an olive or grass-green color (Traube), but elastic fibres are seldom found. Tubercle bacilli are sometimes found, but in this case originate not from the fresh pseudopneumonic patches, but from the causal focus; in few cases the physical signs of the primary cavity in an upper lobe can be elicited. The physical findings change but little, though the crepitant rales are replaced by coarse mucous rales; the anticipated crisis does not occur, and most cases are still called unresolved pneumonia until the increasing anæmia, loss of strength or rapid pulse, suggests a tuberculous pneumonia. Albuminuria is uncommon; the diazo reaction is nearly always present. Of 13 cases reported by Fränkel and Troje, 11 died within six weeks, and Tindeloo observed a fatal issue in six days. In some few cases considerable regression may occur and it is barely possible for the course to last three months or more. The *findings at autopsy* are most interesting; usually an old focus of tuberculosis is found, a ruptured cavity or gland; from it caseous material, containing bacilli and tuberculous toxins, is aspirated into many finer alveoli and bronchioles, where they excite a miliary caseous lobar pneumonia, depending on the size of the affected area only. The inflammation is seemingly due wholly to Koch's bacillus or its toxins, and is (i) partly fibrinous and desquamative, red and white polynuclear and mononuclear cells being poured out; proliferative changes are noted, as the epithelioid and giant cells seen in the typical tubercle; caseation ensues, as shown in the already fused and fusing yellowish-gray

areas; (ii) the most rapid exudation is serous, Laennec's "gelatinous or smooth pneumonia," which is probably due, as Fränkel and Troje insist, to the toxins of the tubercle bacillus and not to mixed infection, as Ortner and others maintain; this part of the inflammation may regress, and does not necessarily caseate. Cavities rarely form in this type.

(*b*) *Acute Disseminated Form.*—Acute disseminated tuberculosis may occur as a special acute type, or may complicate the ordinary form of phthisis. Following the rupture of a small centre of softening, which is usually located only at the postmortem, and following dissemination by aspiration, there develop (i) fever, repeated hæmoptysis, chills, rapid pulse, rapidly increasing dyspnœa, diffuse crepitant or bubbling rales, considerable delirium, or even a pronounced status typhosus. (ii) The sputum is absent or scanty, and the bacilli are sometimes absent throughout the course. At this stage death may occur acutely in three to four weeks without physical signs having appeared and without breaking down of the foci, or a remission may take place, after which there is (iii) breaking down of the *indistinct* foci which cause slight *dulness* and distant *bronchial breathing;* lobular pneumonia is frequently diagnosticated. At autopsy, scattered, opaque, yellow, partly caseous peribronchial nodular foci are seen; in some cases the presence of tuberculosis is proven only by microscopic examination, for, without caseation, the foci may resemble the ordinary bronchopneumonic patches. Secondary infection with streptococci is usually said to cause this form, but Prudden demonstrated that the above lesions may result from the tubercle bacilli alone.

(*c*) *Acute Disseminated Ulcerative Form.*—The acute disseminated ulcerative type is often observed in children between two and six years of age (Grancher), in pregnancy, alcoholism, diabetes and influenza. The foci, which vary from the size of a pea to that of a walnut, are widely disseminated; they fuse, break down early and develop cavities without walls, which are only ulcers or consist of necrotic caseous material. There are present clinically hectic or continuous fever, pronounced anæmia, exhausting sweats, rapid emaciation and viscid sputum, which is purulent and contains many bacilli; diffuse fine and large rales and bronchial breathing may be heard, but the physical signs are often vague. The course is galloping, and ends fatally in one to four months.

(*d*) *Acute Miliary Form.*—Acute miliary tuberculosis (*q. v.*) develops in 3 per cent. of the cases of pulmonary phthisis.

2. Chronic Tuberculosis.—(*a*) *Chronic Ulcerative Form.*—The ordinary chronic ulcerative phthisis may run a course which is long latent, except for anæmia, dyspepsia or other undistinctive symptoms. Improvement may follow the detection of such positive lung findings as an apical catarrh, and the cough, fever, sweats and anorexia may subside; another relapse may occur after years of quiescence, or exacerbations develop, with loss of weight and strength.

Recovery may occur, even with pronounced physical signs. Cornet regards the breaking out, after years, of new tuberculous manifestations as reinfections rather than relapses.

(*b*) *Phthisis Fibrosa.*—Phthisis fibrosa is the slow development of fibrous connective tissue excited by the tubercle bacillus. It may begin acutely or follow an insidious course from the onset. Its explanation is difficult; Auclair thinks the bacillus secretes two toxins, one with a necrosing action which leads to caseation, and another with an indurating action which leads to fibroid phthisis. There may be no cough, sputum, fever, emaciation or anæmia. Bacilli may or may not be found in the sputum. Ulceration may result only from mixed infection. Sometimes there is hæmoptysis. If left-sided, the spleen rises higher and the retraction of the lung away from the heart makes its apex wider, the cardiac shock more diffuse, and the pulsation of the pulmonary trunk more visible. If right-sided, the liver rises, the right ventricle is apparently enlarged (from lung retraction) and is often actually hypertrophied and dilated from increased tension in the pulmonary circuit. The heart may be drawn to the right or left by the inevitable pleural adhesions, bronchiectases are not uncommon, the sound lung is vicariously emphysematous, and the diseased lung is smaller, flatter and depresses the shoulder, impairing expansion and often presenting bronchial breathing and large rales.

(*c*) *Pleuritic Form.*—The pleuritic form follows small latent foci in the lungs, lymph glands, etc. (See PLEURISY.) Exudates not due to rheumatism, inflammation in contiguous organs, tumor or nephritis are always suggestive of tuberculous pleurisy. Pleurisy, in fact, more often precedes than succeeds pulmonary tuberculosis and repeated attacks are frequent, with moderate irregular or continuous fever, perhaps with rales in the apex of the affected side (not the rales frequently heard just above the upper level of the exudate), or with dulness in the contralateral apex. Bowditch, following up 90 cases of pleurisy, found that 33 per cent. developed pulmonary tuberculosis.

Variations According to Age.—1. CHILDREN.—Tubercle bacilli easily pass the bronchial mucosa and produce tuberculosis of the bronchial and mediastinal glands; the wide lymph-vessels and more active metabolism have already been noted. In children the process in the glands attacks secondarily the hilum of the lung and the lower lobes (80 to 98 per cent.). This invasion explains the frequency of miliary tuberculosis from involvement of the thoracic duct and blood-vessels.

(*a*) *Chronic Generalized Tuberculosis.*—The younger the child the less local and the more general is the tuberculosis. Measles, pertussis and influenza are very prone to arouse a latent tuberculosis in the bronchial glands. The course is generally more active than chronic, like that of tumors in children. Emaciation, anæmia, enlargement of the spleen and liver and moderate intumescence of the peripheral lymph-glands are the variant features of this type. Fever

may occur only toward the end of the process. Hectic fever is seen chiefly in children over three years. The sputum is swallowed, but washing the stomach recovers bacilli-laden sputum.

(*b*) *Acute Miliary Tuberculosis* (*v. s.*).

(*c*) *Localized Lung Tuberculosis.*—Localized lung tuberculosis may be (i) of the acute caseous type resembling pathologically bronchopneumonias of other causation, and the microscope may be necessary to detect its nature; hæmoptysis is rare, though Henoch has observed over a dozen cases under six years of age. (ii) Chronic localized forms are very infrequent under six years of age.

In children, the physical findings are often absent; when present they are usually over the lower lobes, very seldom near the clavicles. Cavities are rare and if softening develops, do not freely communicate with the bronchi. In half the cases the wrong diagnosis is made (Henoch). Percussion is more difficult than in adults, because of the small chest and its thinner walls; for example a cracked-pot resonance can be obtained in a normal young chest by strong percussion. Children, too, breathe irregularly, and puerile respiration is physiological. Induration is rare.

2. The Aged.—In the aged, hæmoptysis is rare, physical findings are less frequent than in adolescence, because the breathing is weaker and emphysema is more common. Cough and fever are also less conspicuous.

Diagnosis of Pulmonary Tuberculosis.—Early diagnosis is most important. A positive family history is of some value, but it does not establish the diagnosis in a doubtful case, nor does its absence exclude tuberculosis. (i) The *Bacillus tuberculosis* distinguishes pulmonary tuberculosis (*a*) from other catarrhs, such as acute and chronic bronchitis or influenza; (*b*) from other consolidations, *e. g.*, pneumonia, bronchopneumonia, actinomycosis, neoplasm or syphilis; (*c*) from other vomicæ, such as the cavities of abscess, gangrene, actinomycosis, echinococcus, syphilis or bronchiectasis; (*d*) from indurations other than tuberculous. *Every sputum, however trivial, should be examined. Cultures and inoculations* may be made in doubtful cases. (ii) In doubtful cases, *repeated physical examinations* are imperative; auscultation is more valuable than percussion; too great reliance should not be placed on negative findings; any lesion in a lower lobe with findings in an upper apex is almost surely tuberculous.

(iii) *Tuberculin Tests.*—(*a*) The *ophthalmo-reaction* of Wolff-Eisner and Calmette (1907) consists of dropping in the eye one drop of 1 per cent. old tuberculin which is followed in a few hours (12 to 24) by a reactive conjunctival hyperæmia. In 6300 tests, Calmette found a positive reaction in 90 per cent. of tuberculous patients, in 57 per cent. of suspected tuberculosis and in 17 per cent. of apparently healthy persons; the only complications were keratitis in three instances, conjunctivitis in 20 and a reaction lasting over three weeks in 72 instances. Ulcers may develop if tuberculin is in-

stilled into diseased eyes. The reaction fails in advanced tuberculosis and may develop in typhoid. (*b*) *Von Pirquet's test* is vaccination of the skin under a drop of 25 per cent. old tuberculin (in salt solution); in 24 hours a small papule appears. This method is absolutely safe, most adapted to children, more prone to show latent tuberculosis and rather too delicate to be valuable in adults unless it is negative. (*c*) *Hypodermic injections* of Koch's *tuberculin* produces a local exudative reaction, and after four or five to ten hours, a systemic reaction, which is characterized by chill, fever elevation of even 5 degrees, headache, general pains, malaise, nausea, sometimes by erythema nodosum, an increase of sputum, rales or even a slight area of pulmonary dulness; the first injection of Koch's older tuberculin should be small, in adults 0.2, 0.5, 1 or 5 milligrams—in children 0.1 to 1 milligram. The reaction is probably specific and generally means that there is a tuberculous focus in the body, but not necessarily in the lungs, unless rales develop at the suspected spot after the injection. Positive reactions have been recorded in syphilis, leprosy, cancer and actinomycosis, but they were not proved by autopsy (to show absence of tuberculosis). Latent tuberculosis lessens the value of positive reactions; thus, a case with enlarged mediastinal and supraclavicular glands, without other physical findings, gave a positive tuberculin reaction, though the autopsy showed that they were carcinomatous from a clinically latent gastric carcinoma, and the reaction came from a single tuberculous mediastinal gland which was of no diagnostic significance. In veterinary medicine it is more valuable, leading to a correct diagnosis in more than 90 per cent. (Bang) or 97 per cent. (Voges). The author admits a reluctance to use tuberculin freely; four cases of miliary tuberculosis apparently resulted from its employment.

Sticker recommended the use of *potassium iodide* in small doses (gr. iij); it congests the lungs at the seat of the lesion, possibly makes the auscultation findings clearer, and carries bacilli into the sputum; its use is not wholly free from danger.

Pseudotuberculosis.—The Rhizomucor parasiticus (Lucet and Constantin), the ray fungus, streptothrix (Eppinger) and the aspergillus may cause pseudotuberculosis. The form caused by the Aspergillus fumigatus is described as a primary lung affection by Dieulafoy and others, but many writers consider it secondary only. The symptoms of *streptothricosis* are hæmoptysis (23 per cent.), fatigue, anorexia, dry cough and, later, a greenish purulent sputum. The physical findings are bronchitis, less often slight consolidation or cirrhosis of the lung. Ashton and Wright (1905) collated 26 cases of streptothricosis pulmonum, of which most had symptoms resembling phthisis; over 50 per cent. were unmixed infections and 76 per cent. died.

Prognosis of Pulmonary Tuberculosis.—Two issues are possible, recovery and death.

1. Recovery.—Autopsies on hospital cases show 25 to 97 per cent.

of healed tuberculosis. Many of the recoveries are only relative, since Kurlow's inoculations from seemingly latent caseous foci into guinea-pigs were positive in 77 per cent.; such foci may be aroused by intercurrent disease, though some writers regard recurrence as fresh infection rather than recrudescence. Watson observed a clinical course of fifty-four years, and Fagge one of thirty-five years. Recovery is effected by *agglutinins* (precipitins), elaborated by the infection, and by other specific antibodies (*opsonins*) which aid the leucocytes in the process of phagocytosis. The result is encapsulation, scar formation and the deposit of lime salts. Liebermeister believes recovery is possible in 50 per cent. of incipient cases. The outlook depends on several factors: (*a*) The *hereditary vital endowment* of the patient; (*b*) his *environment, social situation,* habits and intelligence; (*c*) whether the localization of the disease is single or multiple; (*d*) secondary or mixed infection; (*e*) digestion; (*f*) degree of toxin absorption (fever and emaciation); (*g*) youth, diabetes, pregnancy and lactation are unfavorable; (*h*) pulse rate; 80 per cent. with normal pulse were alive after 4 years and 97 per cent. with rapid pulse were dead (Schneider); and (*i*) complications (*v. i.*).

2. Death.—The clinical course is variable, even in chronic cases; it averages two years (Bayle, Laennec, Andral), over two and a half years (Pollock), three years (Cornet) and seven years (Williams and Dettweiler, though the latter's cases did not include acute forms or cases in children). The process of death is usually peaceful and painless, though at times it comes with dyspnœa (stagnant secretion), delirium or convulsions. Mechanism of death: (*a*) Heart failure is the common cause, associated with œdema, asthenia, anæmia and marasmus. Other causes are less frequent, as (*b*) profuse fatal hæmoptoë; (*c*) pneumothorax; (*d*) pulmonary embolism (clots or gas); (*e*) uræmia; (*f*) meningitis; (*g*) perforative peritonitis; (*h*) sudden death without adequate pathological findings. In cases of sudden death, Arnold, Brissaud and Toupet, have found in the liver large numbers of bacilli, which probably caused an acute intoxication, since Mafucci's experiments demonstrated that the injection even of dead bacilli causes rapid marasmus and death.

Complications.—Tuberculosis in other organs will be considered below.

1. Respiratory.—(*a*) Tracheitis and *bronchitis* occur in almost every case; and laryngitis in many cases results from the toxins. (*b*) *Dysphagia* usually results only from local involvement of the pharynx or larynx; swallowing into the larynx is usually the result of paralysis of the epiglottic depressors. *Hoarseness,* dysphonia and aphonia always suggest *laryngeal tuberculosis,* but may develop from such other causes as syphilis or catarrh; from paralysis of the recurrent laryngeal nerve from pressure by lymph glands; from inflammation, on the right side by pleural scars, and on the left by large pleural effusions; or from thickening at the hilum. (*c*) Pulmonary *gangrene* may develop in the cavity wall. Lobar pneumonia is not in-

frequently a cause of death. (*d*) Dry *pleurisy* is an almost invariable complication, and is a conservative process, which lessens the probability of pneumothorax. Exudative pleurisy more often develops previously to lung phthisis than during its course. In the latter instance it is usually serofibrinous, and is generally not extensive. Hemorrhagic pleurisy is more common in pleural than in pulmonary tuberculosis. Chyliform or adipose effusions may occur (see CHYLOTHORAX AND CHYLOUS ASCITES); purulent forms result from mixed infection, from cavities or from pneumothorax. Some pleurisies appear to result from toxæmia rather than from direct bacterial invasion. Effusions, by compression, sometimes seem to exert a favorable effect on a lesion of the lung. Litten thought that their sudden absorption might occasion miliary tuberculosis. (*e*) *Pneumothorax* is observed in 5 per cent. of the cases (Powell, West), and results from rupture of superficial cavities which were forming rapidly without a limiting membrane. Perforation occurs most frequently between the second and fourth interspaces, and between the mammary and axillary lines. Its onset is spontaneous, or results from straining or coughing. Its occurrence is usually marked by sudden symptoms, as great dyspnœa, orthopnœa and cyanosis, and sudden physical signs, as lying on the side of rupture, sudden ectasia, immobility, dislocation of the heart, spleen or liver, weak vocal fremitus, voice sounds and breathing, a tympanitic note, and when serum or pus forms, succussion. The onset is occasionally gradual and less urgent, so that pneumothorax may be an accidental finding. Death in a few hours may result, but life may be prolonged weeks or months. Infrequently pneumothorax (*q. v.*) may exert a beneficial effect on the lung trouble.

2. CIRCULATORY.—(*a*) The typical *rapid pulse* first appears only after meals or exertion, but later becomes permanent. It is *probably toxæmic,* but may be due to mechanical pressure of large lymph-glands on the vagus, or to compression or inflammation of the vagus from mediastinitis, pleurisy or pericarditis. There is a constant and early *lowering* of the *blood-tension,* and the vasomotor instability causes the bright-red cheeks. The pulse may be soft and full throughout the course. (*b*) The *heart* is pale, fatty, flabby and weak, and has been described as atrophied since the time of Laennec, but Potain considers that the small heart is merely accommodative to the malnutrition and anæmia. Cardiac hypertrophy is due to renal disease, pericardial or pleural adhesions, valvular disease, arteriosclerosis or emphysema. Hypertrophy and dilatation of the right heart may follow pulmonary induration or massive pleural adhesions, and the second pulmonic tone is accentuated in one third of the cases. Adhesive or effusive *pericarditis* may result by extension from pleurisy or caseated lymph-glands, but may be due to tuberculosis or pyogenic infection. Endocarditis tuberculosa is very infrequent; tubercle bacilli may be deposited in cardiac clots, or in endocarditic excrescences of non-tuberculous origin (Kundrat and Heller); Tripier's

case of tuberculous endocarditis was the first clear one; according to Benda, this endocarditis is due to miliary deposits in the intima, and clinically escapes recognition in most cases. (*c*) A functional, systolic murmur over the pulmonary valves or apex is very common. (*d*) *Thrombo-phlebitis* occurs in about 1 per cent. of the cases.

3. Nervous and Muscular.—(*a*) In some instances the mental power may be normal; oftener there is a *slight mental weakness,* irritability, psychical lability, self-concentration, *lack of endurance* and triviality. Sleep is often disturbed. Consumptives are usually *optimistic,* and they may plan for the future on the day of their death (*Spes phthisicorum*); occasionally melancholia or mania develops; fever, anæmia, toxæmia, marasmus and vasomotor changes in the brain, such as œdema, may cause terminal aberration. (*b*) *Neuralgia* and hyperæsthesia are not uncommon. Multiple *neuritis* not infrequently results from toxæmia; it may be latent, cause slight sensory disturbance or result in amyotrophy; it may in part explain œdema not due to cardiac weakness or thrombo-phlebitis. (*c*) The same toxins which reduce the body weight one-half or more, result in marked *muscular degeneration* and atrophy, which cause the flat chest, and possibly also the cardiac wasting. The muscular and adipose wasting is expressed in the terms synonymous with tuberculosis —consumption, *phthisis,* tabes. The muscles are dry, pale, fatty; they show transverse rents, separation of the sarcolemma, poor markings, granular degeneration or hyperplasia of the connective tissue; myositis is described by Delorme and Reverdin. *Their wasting and the nervous toxæmia explain the asthenia* and incapacitation for work, a matter of national economical importance. The muscles are often irritable, show fibrillary contractions and when tapped or pinched show local contraction or lumps, known as *myoidema.*

4. Digestive.—(*a*) The tongue may be furred, or red and clean. Aphthæ are frequently distressing. Excessive *redness* of the *gums* is frequent. (*b*) *Stomach* disorder is present in 33 to 66 per cent. of cases; it may be the initial symptom of toxæmia, or appear only late in the process. The appetite may be unimpaired, and Cornet instances an advanced patient who complained that he could only eat four beefsteaks for dinner. "The patient, with fever, who eats and digests well, is tuberculous" (Lesègue). *Anorexia* varies in degree and occurrence, being most marked toward evening, when the fever is higher, and most frequent in those with previous dyspepsia or poor appetite; it is sometimes nervous (anorexia nervosa) with normal gastric chemism; again it may be due to glands compressing the vagus (Peter, de Mussy). Brieger found the chemism normal in 50 per cent. in the incipient stage, in 33 per cent. in moderately severe, and in but 16 per cent. in severe advanced cases. After eating there may be a sense of fullness or pain. Nausea, gagging and *vomiting* may result from pharyngitis, forced feeding or coughing (Morton's "emetic cough"). In advanced cases, nausea and vomiting occur from over-eating, *toxæmic gastric catarrh,* swallowing of sputum, weak-

ness of the stomach musculature and deficient secretion; anatomically, the stomach shows anæmia, catarrh, fatty degeneration of the glands (36 per cent.), interstitial hyperplasia and sometimes amyloid infiltration (12 per cent.).

(*c*) *Intestines.*—Constipation may be caused by gastric hyperacidity. Diarrhœa is usually late; it may be caused by the toxin in the blood or in swallowed sputum, by intestinal catarrh, toxæmic obliteration of the lymph- and bloodvessels in the gut, tuberculous ulcers or amyloid degeneration in the gut. Tuberculous *ulcers* may exist without symptoms or may cause colic, tenderness, pain and diarrhœa; much depends on their location. According to Nothnagel, ulcers in the small gut, cæcum and ascending colon probably cause diarrhœa only when combined with catarrh or amyloid degeneration; ulcers in the lower colon and rectum almost constantly induce diarrhœa, which may, therefore, occur from causes other than ulceration, and ulcers may exist without it. Blood, pus, hæmatin crystals and tubercle bacilli may be present, but not always. Tubercle bacilli do not necessarily indicate intestinal tuberculosis, as they may pass through with the sputum swallowed. Anal fistula occurs in 1.3 to 5 per cent. of the cases. (*d*) The liver is fatty (in 10 to 15 per cent.); Jonescu (1902) collected 33 cases of cirrhosis cardiotuberculosa,—induration from stasis, with tuberculous granulations and fatty degeneration. (*e*) Tuberculous *peritonitis* (*v. i.*) occurs in 5 to 15 per cent. of pulmonary tuberculosis.

5. Amyloid Degeneration.—This occurs mostly in chronic ulcerative phthisis. According to Blum, 80 per cent. of all amyloid degeneration is due to tuberculosis, and 68 per cent. to pulmonary tuberculosis; it is found in 8.8 per cent. of tuberculous autopsies. The spleen is affected in 93 per cent., the kidney in 77, the liver in 62, the intestines in 28 and the stomach in 12 per cent. of cases.

6. Genito-urinary.—*Urinary.*—The loss of weight causes *phosphaturia,* increase of nuclein and chlorides (0.5 to 1 or even 12 gm.). More proteid is excreted than is ingested. Albumose is sometimes present. *Albuminuria* usually indicates nephritis, amyloidosis or local tuberculous disease. Acute and chronic parenchymatous nephritis is not infrequently due to long-standing toxæmia. *Amyloid* disease of the kidney produces albuminuria in but 50 per cent. of the cases; in amyloid of the kidney, nephritis is also present in 75 per cent., and the chronic parenchymatous type in 66 per cent. Flick and Walsh found tubercle bacilli in 84 per cent. by inoculating the urine in guinea-pigs; tubercles were found in 63 per cent. Ehrlich's *diazo reaction* is very frequently present, and is usually of unfavorable import; the prognosis is not necessarily good if the reaction is absent.

Sexual.—In some individuals, the sexual desire is more intense, though its increase is grossly exaggerated. The *menses often disappear early,* and pregnancy is less apt to occur. Pregnancy often temporarily betters the condition of the lungs, but a more rapid course

is usual after delivery. The occasional hypertrophy of the mammary glands, described by Bedor in 1836, particularly in males, is rather a chronic interstitial mastitis than an actual hypertrophy.

7. Skin.—Cyanosis is not common. *Pityriasis versicolor* occurs very frequently in the tuberculous; it is due to the tinea furfur (Eichstedt). Chloasma, desquamation, coarseness of the hair, herpes zoster, lichen scrofulosorum and purpura are sometimes observed. *Clubbing of the fingers* (the "Hippocratic fingers") and toes develops chiefly in chronic cases, is largely due to hypertrophy of the soft tissues (see Acromegaly) and is not pathognomonic of tuberculosis, for it occurs also in congenital cardiac disease, aneurysm, bronchiectasis, emphysema, etc. Emphysema of the skin is very uncommon; it follows interstitial emphysema of the lungs, and results from rupture of the alveoli, pneumothorax or cavities. Œdema results from anæmia, cardiac weakness, phlebitis, nephritis or neuritis.

Treatment of pulmonary tuberculosis is considered later, after description of other localizations of the tubercle bacillus.

IV. Tuberculosis of the Lymph Glands.—Of glandular adenopathies 68 per cent. are found in the first ten years of life (Wohlgemuth). It occurs fairly often in adolescence and sometimes even in the aged. It is found anatomically in 99 per cent. of tuberculous children (Sterner and Neureuther), or according to other statistics in 80 to 90 per cent.; it is found in 25 per cent. (Müller) to 50 per cent. (Babes) of all autopsies on children. *Scrofula* is glandular tuberculosis, but it presents different characteristics from those of pulmonary infection, in explanation of which it is held by Arloing that the virulence of the bacilli is less in scrofulous glands; others explain it as an infection with very few bacilli. Neither of these explanations is wholly adequate, whence the assumption of a special glandular tendency to infection; it has been suggested that the glands invaded are poorly developed. The skin, mucous membranes and lymph-vessels are physiologically more patent or more permeable in the very young, and an increase of this condition or its persistence in older children is regarded as an infantilism or embryoism, *i. e.,* a structural tendency toward infection. The accessory causes are the same as in pulmonary tuberculosis: (*a*) An hereditary tendency; (*b*) poor hygiene, dark damp dwellings, inadequate nutrition and coarse food given to the very young; (*c*) infections, such as pertussis and measles, to a less extent scarlatina, diphtheria, variola or syphilis.

General Characteristics.—(*a*) It usually runs an extremely chronic course. (*b*) Recurrence is very frequent. (*c*) Multiple lesions are usual. (*d*) Glandular involvement is frequently associated with affections of the skin (as eczema, scrofuloderma or lupus), inflammations of the mucous membranes (as conjunctivitis, otitis, rhinitis or bronchitis), and disease of the bones or joints (as coxitis or Pott's disease). This association is frequently known as scrofula, of which some authors, as Cornet, distinguish two varieties—(i) the

tuberculous and (ii) the pyogenic, caused by the staphylococcus or less often by the streptococcus. It seems incongruous to unnecessarily associate tuberculous lymphadenitis and arthritis with non-tuberculous forms.

General Anatomy.—(*a*) Few bacilli cause but little inflammatory reaction and little cellular proliferation; regression is possible. (*b*) If the infection is sharper, conglomerate tubercles form, with the following issues: Death of the bacillus in the central caseation, softening and external rupture, sometimes calcification or extension locally by the lymph, or generally by the blood current. (*c*) Hyperplasia with epithelioid proliferation may occur, without caseation or with very late caseation (Ziegler); this form results from infection with few bacilli. The lymphadenoid tissue is compressed by epithelioid spindle-shaped cells; the glands may reach the size of an egg, and their cut section may show no gross evidence of tubercle. (*d*) Mixed infection causes rapid caseation, necrosis, softening and early rupture; their cut section appears "like that of a raw potato but not so moist" (Virchow); the inflammation is often not limited to the glands, but causes a periadenitis, whereby the glands fuse, a phlegmon results, and generalization of the process throughout the body ensues. In purely pyogenic lymphadenitis, the glands may become homogeneous and dry (the hyperplastic lymphoma of Virchow), or productive inflammation may result in fibrous induration.

1. Cervical Tuberculous Adenitis.—Infection occurs through the nose, eye, scalp, pharynx or ear; the primary atrial lesion may leave no mark; or the bacillus may pass through the mucous membranes without producing any lesion in them. Writers who describe scrofula as a special symptom-complex, distinguish four stages: (*a*) The initial skin or mucous membrane inflammation; (*b*) the lymphadenitis; (*c*) involvement of the joints and bones; and (*d*) various complications, as amyloid degeneration or miliary tuberculosis.

The figures of Balman (first column) and Wohlgemuth (second column) show the following localizations:

	Per cent.	*Per cent.*
Cervical and occipital glands	81	93
Axillary (from the mamma)	6	2.78
Inguinal (from the hip, ilium, testicle and knee)	7	0.93
Cubital (from the fingers)	5	0.23
Popliteal (from the foot)	0.7	0.23
Pre- and postauricular	——	2.9

The cervical glands constitute 96 per cent. of Wohlgemuth's series. In cervical scrofula, the glands at the angle of the jaw and beneath or anterior to the sternomastoid muscle are most often involved; in bilateral involvement, the glands of one side are usually larger. The glandular swelling is slow in onset, intermittent in progression, usually unattended by pain or other symptoms and frequently an accidental finding. In some cases the first symptoms are acute, but the

trouble subsides in two or three weeks. The intumescence first is discrete and freely movable. Later the nodes may blend into large nodular masses, and periadenitis may fuse the glands and cause their adherence to the skin, which becomes œdematous, dough-like and reddened. Softening and fluctuation are then frequent, with burrowing of the pus in various directions and external rupture through small fistulæ. The fistulæ widen, discharge cheesy and milky material and form ulcers covered with granulation tissue and difficult to heal. Cicatrization leaves deep, deforming, depressed scars. Extension may occur to the deeper or subpectoral or axillary glands. Fever, anæmia and other toxæmic manifestations may develop as in lung tuberculosis, or the adenopathy may be the sole clinical fact.

2. Bronchial Glands.—In autopsies on tuberculous children they are almost invariably affected (in 98 per cent. of Steiner's and Neureuther's and in 100 per cent. of Northrup's series); in adults they are affected in 40 to 80 per cent. In children, tuberculosis of the bronchial glands is primary, the bacillus passing the intact bronchial mucosa, and tuberculosis of the lung is secondary. In adults, lung involvement is usually primary and bronchial adenitis is secondary. Small tuberculous foci cause neither glandular enlargement nor symptoms. Biedert, in 1,346 autopsies on tuberculous children, found the lungs diseased in 80 per cent., the bronchial glands in 78, the intestines in 32 and the mesenteric glands alone in 40 per cent.

Symptoms.—In most cases no symptoms develop; in others, pulmonary lesions overshadow the bronchial involvement; in others, miliary tuberculosis develops, promoted by the close contact of the bronchial glands with, and easy rupture into, the pulmonary vessels; in still others, mediastinal pressure symptoms result; in very few cases are there actual physical findings.

Pressure Symptoms.—Pressure on the vagus, known to Wrisberg, causes the rapid heart, so frequently remarked in tuberculous children; paroxysmal asthma, dyspnœa, or spasmodic cough which sometimes closely resembles that of whooping-cough; lessening of the sensitivity of the bronchi, which favors infection; and it finally leads to cardiac dilatation and death. Pressure on the *recurrent laryngeal* nerve is marked by hoarseness, aphonia, dyspnœa or laryngospasm. The *air passages,* trachea or bronchi may be compressed, as evidenced by dyspnœa, impaired expansion of one or both sides of the chest, and holding the head backward. The pupils may be unequal from pressure on the sympathetic nerve. Other pressure symptoms are: Hypertrophy of the heart, or the paradoxical pulse, from pressure on the aorta; œdema and cyanosis of the face and arms, from compression of the superior vena cava; cough, rales or hæmoptysis, from stenosis of the pulmonary veins; a murmur over the innominate vein on throwing the head backward; and dysphagia, from œsophageal compression.

Signs.—Bronchial breathing is occasionally heard between the

second and fourth dorsal vertebræ, more clearly behind than in front; more on the right than on the left side; and more markedly than the high-pitched breathing heard physiologically over this region. If the bronchus is compressed there is neither bronchial breathing nor increased vocal fremitus. An irregularly outlined dulness is elicited on exceptional occasions either between the clavicle and third rib, on either side of the sternum, or behind between the third and fifth dorsal vertebræ. It is due to enlarged glands near a bronchus, or sometimes to a distended obstructed vein. Sometimes a tympanitic note in the upper lobes results from relaxation of the lung. Both the symptoms and signs are extremely variable, appearing suddenly and remitting unaccountably.

Issues.—(*a*) Tuberculosis of the lung (in children); (*b*) miliary tuberculosis; (*c*) tuberculous pleurisy or pericarditis; (*d*) pressure by calcification or fibrosis; (*e*) adhesion to and rupture into the air passages, causing sudden suffocation; (*f*) fatal hemorrhage into the air tubes, which is less frequent, as the vessels yield before pressure, but which may result from rupture simultaneously into a bronchus and a vessel; (*g*) rupture into the œsophagus or traction upon it, causing an œsophageal diverticulum.

Diagnosis.—The direct diagnosis rests upon tuberculous adenopathies elsewhere, pressure symptoms, physical signs and evidences of extension, general or local. As Henoch puts it, "A diagnosis can rarely be made (from the physical signs), but if made, is nearly always correct," as the bronchial glands are nearly always enlarged in infantile tuberculosis.

3. Mesenteric Glands.—The bacilli pass the intact intestinal mucosa and settle in the mesenteric glands, precisely as they pass the bronchial mucosa and lodge in the bronchial glands. Carrière found that but 1 per cent. of his cases were primary; of the secondary cases, the primary focus was in the peritoneum in 40 per cent., in the lungs in 30 per cent., in the intestines in 20 per cent., in other glands and bones in 5 per cent. each. Hess (1908) holds that in over 60 per cent. of the primary cases, the infection is by bovine tubercle bacilli; in adults, it is by the human type. There are few characteristic symptoms. The glands may be palpable as thick nodular masses through a thin abdominal wall, but are often obscured by the abdominal distention. The *general emaciation* (*tabes mesaraica*) contrasts strikingly with the *tumid belly*. Digestive disturbance is most frequent, and there is usually *a diarrhœa* with thin, stinking stools. Compression is less frequent in the more distensible abdomen than in the less yielding thorax, though the veins of the abdomen may stand out and œdema of the legs may appear. The glands sometimes heal by induration and calcification, or, by softening, rupture into the intestine or peritoneum (plastic or exudative peritonitis). Amyloid degeneration may result from cases of long standing.

4. Generalized Lymphadenitis.—This may closely resemble Hodgkin's disease, as was first pointed out by Richard Paltauf and

his co-worker, Sternberg, though the latter goes too far in declaring that most cases of pseudoleukæmia are glandular tuberculosis. In two personally observed instances of supposedly clear pseudoleukæmia, the clinical and gross pathological findings were typical of that disease, and only the epithelioid hyperplasia and the bacillus tuberculosis stamped them as tuberculous. This form is rather common in the negro.

The *treatment* of tuberculous lymphadenitis is that of tuberculosis in general (*v. i.*), and is surgical if the process is accessible and localized. In 175 cases of cervical adenitis treated surgically, 58 per cent. were cured after three years, 8 per cent. had recurrence, 11 per cent. developed pulmonary tuberculosis and 23 per cent. died. In another series of 700 cases treated medically, 28 per cent. developed tuberculosis in some other organ (Demme). The writer relies largely upon full feeding, ample sunlight and fresh air and, if possible, a warm sea-side climate. The syrup of the iodide of iron and cod-liver oil are very largely used. Inunctions of green soap, recommended by Richter (1846) and Kappesser (1878) are considered beneficial by Hoffa; 3ss–j should be thoroughly rubbed into the back two or three times a week.

V. Tuberculosis of the Serous Membranes.—These membranes may be involved simultaneously or separately. Multiple involvement is known as multiple serositis or polyorrhomenitis. Multiple serositis may be acute, subchronic or chronic, serous, fibrinous or purulent, solitary or with visceral tuberculosis.

1. Pleura.—Tuberculous pleurisy has been touched upon as a complication of phthisis, and must be separately considered under Pleurisy (*q. v.*). It may develop as an apparently primary infection, it may be clearly secondary to unmistakable phthisis, or it may appear as a terminal infection. Miliary tubercles in the pleura may occur without coincident pleurisy. Pleurisy develops in some form in 66 to 100 per cent. of pulmonary phthisis. It may be acute, subacute or chronic; it is most often serous or serofibrinous; it may be serohemorrhagic, purulent, less often chyliform or adipose. Pleurisy may result from direct bacillary invasion from the bronchial glands, lung, spine or peritoneum, or possibly from simple toxic action without the agency of bacilli. In some cases the pleura proliferates and forms a thick, massive, adhesive, obliterative membrane. Sometimes the interstitial tissue of the lung is invaded, resulting in pulmonary cirrhosis (pleurogenous cirrhosis).

2. Pericardium.—Accidental miliary deposits must be sharply distinguished from tuberculous pericarditis wherein inflammation predominates. It is less common than pleural and peritoneal involvement, but is a pathological finding in 2 to 3 per cent. of pulmonary tuberculosis. Tubercles may be detected beneath the fibrin deposits or adhesions, as they are in pleurisy of the same type. Without association with some other tuberculous lesion (especially that of the

pleura) it is rarely separable clinically from the ordinary forms of pericarditis. Like these forms it may be symptomatically latent, it may form an unessential part of a generalized tuberculosis, or it may present the ordinary symptoms of pericardial effusion or concretion (*q. v.*). Only eight primary cases are recorded (Scagliosi, 1904).

3. **Peritoneum.**—Tuberculosis of the peritoneum was known to Bayle, Laennec, Bichat and others, a century ago, but became a clinical entity when Louis, in 1825, declared that nearly all chronic peritonitis was tuberculous. Though some chronic peritonitides are due to trauma, alcoholism, nephritis, portal and cava-stasis, most of them are tuberculous.

Frequency.—It complicates 10 per cent. of pulmonary tuberculosis and was found in 2 per cent. of 13,422 autopsies (Grawitz and Brunn). It constitutes one-quarter of all peritonitides (Cummins).

Age.—It is almost equally frequent in the second, third, fourth and fifth decades (20 per cent. in each); 66 per cent. of all cases occur between ten and forty years of age; it is infrequent in the extremes of life.

Sex.—Ninety per cent. (Nothnagel), or 78 per cent. (König), occur in women. More cases are found in autopsies on males, though more women are operated on. In America it is more common among the negroes. It is very seldom primary (0.7 per cent.), but is *secondary* to (*a*) *lung, bronchial gland* or *pleural tuberculosis* (in 80 per cent., Leube); (*b*) intestinal ulcers or mesenteric and retroperitoneal adenitis, especially in children. Tubercle bacilli may pass the intestinal wall without localizing in it and reach the peritoneum. It is thought by some that intestinal lesions are the most frequent cause; (*c*) tuberculosis of the Fallopian tubes, which some consider causative, is secondary to some small focus in the bronchial glands. The writer believes that *tuberculous peritonitis is hæmatogenous.*

It is a frequent complication of liver cirrhosis (Rokitansky, Förster and Weigert), though it is strange that tuberculosis should develop in any stasis. It also complicates ovarian tumors or trauma, which disseminates infection from a preëxisting quiescent focus. Sixty-two cases are reported in a hernial sac (tuberculosis herniosa).

Types.—A clinical description is impossible, for the disease may be acute, subacute or chronic, latent or stormy, circumscribed or diffuse; it may be marked by adhesions, by free or encapsulated ascites or by large tumor-like masses; it may be obscured by coincident miliary tuberculosis, liver cirrhosis, exudative pleurisy or ulcerative phthisis, or may appear as a seemingly primary clinical fact. Though the forms are similar and atypical cases are common, certain types may be described. The unessential forms of miliary tubercle, and the small and usually non-inflammatory tubercles seen over intestinal ulcers or in chronic phthisis, are not taken into clinical consideration.

1. The *acute form* usually begins with severe symptoms, which include fever, rapid pulse, abdominal tenderness and pain, free fluid

in the peritoneal sac, which shifts with change of position, considerable meteorism from paresis of the gut, and vomiting. This type may resemble *typhoid* when the intoxication is more marked than the local signs. The *peritonitic manifestations*—local signs—may be sharply marked, but always less so than in suppurative peritonitis. On the other hand, certain cases simulate *ileus,* which at times actually results from tuberculous adhesions or incarceration beneath bands. The writer has seen three cases precipitated by delivery, *resembling a puerperal infection.* Indicanuria, so frequent in other acute peritonitides, is usually absent. The ascitic fluid is usually lemon-colored, serous or slightly flocculent, and of a specific gravity of over 1,014; red blood-cells sometimes impart to it a hemorrhagic hue; the leukocytes are usually mononuclear. In rare instances the acute *peritonitis is suppurative;* 26 cases are on record, due to perforation of a tuberculous ulcer in the gut. In some cases the acute type is a *terminal infection.* Though commonly diffuse, there is sometimes acute local pain or circumscribed effusion, for instance, over an intestinal ulcer. Plastic adhesions occasionally modify the freedom with which this ascitic form shifts with alteration of posture. Acute tuberculous peritonitis comes more often under the care of the physician than of the gynæcologist or surgeon.

2. *Subacute or chronic forms* present diffuse peritoneal involvement. (*a*) The *fibrino-plastic form,* causing adhesions, may result from the acute form or may develop slowly and insidiously; symptoms and signs are usually lacking, or when present are not distinctive. (*b*) The *fibrino-caseous form* is characterized by the development of tubercles, which by fusion often form large solid masses. There are adhesions and thickening of the intestine, mesentery and omentum. Between the recesses formed by the inter-intestinal adhesions, or by the growth of the gut to the liver or spleen, pockets of exudate frequently occur, containing blood-stained serum, cold abscesses or cheesy material; the intestine is thickened and contracted, the mesentery deformed and the omentum shrunken. (*c*) The *effusive form* is marked by serous, serofibrinous, serohemorrhagic and less often by puriform or adipose exudate, which is more or less encapsulated.

All these types and subtypes vary so much that the *individual symptoms* will be considered separately.

Individual Symptoms.—1. Adhesions and Retraction.—The infiltrated *omentum* gradually *indurates and retracts* so that it is found above the navel (Bamberger) or along either costal arch as a fibrocaseous tumor-like mass; it may often be palpated, even when there is much exudate, and becomes clearer after paracentesis; it may be mistaken for carcinoma of the stomach, enlarged liver or distended gall-bladder, especially when in chronic forms there is little fever; it is more suggestive of tuberculous than carcinomatous peritonitis. The *walls of the gut* are frequently so *thickened* that intestinal stenosis results; the gut may measure but one-half or one-third of its original length; it may be thickened, adherent, surrounded by exu-

date, and may also resemble a tumor. Adhesions may continue to retract until the entire peritoneal sac is deformed (Kleb's *peritonitis deformans*); this is a species of spontaneous recovery but is attended by danger of intestinal stenosis. The mesenteric thickening and retraction (*mesenteritis retrahens*) affects the character of the physical findings and the tympany and the dulness; the mesentery may measure one-half to one inch in thickness.

2. Ascites; Exudate.—The abdomen is often moderately enlarged by serous exudation and meteorism. Large exudates may cause some widening of the abdominal veins, as in ascites (*q. v.*), because the cava is compressed. When the abdomen protrudes, its centre is not usually flattened nor are its sides bulging as in the ordinary ascites of liver or cardiac disease, but it is often prominent in the median line, as in ovarian cyst. Löhlein states that most of his cases were sent to him with a diagnosis of ovarian cyst. In cases without adhesions the dulness shifts easily with change of posture, so that tympany exists over the supernatant gut and dulness is found over the settling fluid. The physical signs differ from those of a common ascites in that the tympany is often on the right side; Thomeyer pointed out that the *mesenteritis retrahens* pulls the small gut to the right side, and that its chief cause is tuberculous (less often carcinomatous or simple) peritonitis; nevertheless, a tuberculous mass may develop in the right iliac region, in which case it dislocates the tympanitic intestines to the left, or in the median line or pelvis. In general the abdominal distention is asymmetrical and less extensive than in liver cirrhosis, with which it may co-exist. In very rare cases the fluid may rupture into the bowel or externally through the navel. Dulness does not always indicate fluid but may result from adhesions, thickening of the gut, caseous masses or enlarged glands.

3. Meteorism.—This may result from sudden paresis of the gut in the acute type, or from chronic fibrous obstruction in cases of long standing. Tuberculous tumors may also impede peristalsis and thus cause gaseous accumulations. Meteorism is one of the stigmata of tuberculosis of the mesenteric glands (*tabes or phthisis mesaraica*) and then is often due to coincident peritonitis; in both conditions but most often in children, the stools are fatty (Berggrün and Katz).

4. Palpation.—A *doughy sensation* is often experienced on palpation, which is usually referred to peritoneal adhesions or constricted intestine, but in some instances is due to early infiltration of the preperitoneal, subserous tissues.

5. Other Symptoms.—In some cases the *Beatty-Bright friction-rub* can be elicited. *Pelvic effusion* is particularly common in women, who also suffer from painful and disturbed menstruation. *Fever* frequently is irregular, sometimes continuous, remittent or hectic; it is most frequent in acute types or in acute exacerbations of chronic cases. In some chronic cases it is absent or subnormal. The disease therefore cannot be excluded by the absence of fever. Fever, pain and tenderness usually distinguish tuberculous encapsulation

from simple ascites. *Pain and tenderness* are generally conspicuous, though moderate in degree and by no means constant; they are less acute than in suppurative peritonitis. Emaciation has been especially emphasized by Vierordt; König, however, remarks upon the frequency with which cases of tuberculous peritonitis present a good color and robust appearance. *Gastro-intestinal disturbances* are fairly frequent; the usual constipation is occasioned by impaired peristalsis; in some cases diarrhœa is possibly caused by toxæmia. The spleen is difficult to find in the majority of cases. In rare instances there are icterus, pylethrombosis and other complications due to special localizations of the process.

Diagnosis.—The diagnosis is usually made correctly, though sometimes there are no just grounds for even suspecting the presence of the disease. Considerable importance should be attached to tuberculous lesions elsewhere, as in the lungs, pleuræ, glands or Fallopian tubes. Multiple serositis is extremely suggestive. If another tuberculous lesion cannot be established, tuberculous peritonitis, which constitutes nearly all chronic peritonitides, is very probably present; malignancy and traumatism must be considered. The tuberculin reaction does not prove that the lesion is peritoneal or tuberculous. Inoculation may be made with fluid withdrawn by paracentesis (see PLEURISY). Exploratory laparotomy is justifiable, as simple abdominal section is the foremost therapeutic measure.

Course.—The onset is usually insidious with marked but not extreme pain, tenderness, anorexia, vomiting, colic, fever and constipation. The course, independent of all therapeutic measures, shows spontaneous remissions, as Vierordt and Nothnagel have particularly insisted.

Treatment.—*Simple rest in bed,* good hygiene and *full feeding* very often arrest the process. The tumors, exudate and adhesions may recede and no symptom reappear for months or even years. Spontaneous recovery was first described by Bamberger but was ignored until relatively recent times. In 1884 König proved what Spencer Wells had observed twenty years earlier, that healing may follow laparotomy. Then Lindner, Phillips, Valenta, Rörsch, Hinterberger and others, showed that recovery occurred in 25 per cent. of the cases without medication or laparotomy. Experimental researches have shown that the tubercles heal by simple degeneration, vascularization and development of connective tissue.

Operation gives various results, recovery occurring in 33 per cent. (Frees), 55 per cent. (Chrobak), 64 per cent. (König), 91 per cent. (Dohrn) and 94 per cent. (Mazzoni). In 1300 cases 73 per cent. recovered. Early operation—in the first three or four months—is contraindicated, as Gelpke has shown that the early exudate has strong bactericidal properties. Simple paracentesis is much inferior to laparotomy. Lavage is superfluous. It is thought that the access of air or sunlight, manipulation or lavage, is the potent curative factor in operative treatment. Veit advances the plausible theory that the withdrawal of fluid allows the access of fresh serum with

increased antitoxic properties. Operation, in the author's opinion, is clearly of great value; but it may be mentioned that some conservative writers believe that the tubercles which heal after operation were already in process of healing spontaneously.

VI. Tuberculosis of the Brain and Meninges.—Involvement may result from (*a*) the miliary tubercle, (*b*) the solitary, or, as Virchow prefers to call it, the conglomerate tubercle, (*c*) the tuberculous abscess and (*d*) meningitis. The miliary form and tuberculous meningitis have already been described; the solitary or conglomerate tubercle produces symptoms identical with those of brain tumor (*q. v.*).

Primary cerebral tuberculosis is extremely rare. Warfringe, Fränkel, Alvarez and Dürck have described primary meningitis, but it is extremely probable that small or microscopic foci elsewhere escaped detection. Fränkel found tuberculous meningitis in 4.7 per cent. of adults dying with pulmonary phthisis.

VII. Tuberculosis of the Eye.—Only 50 cases of eye tuberculosis occurred in 141,000 eye patients. The total number of reported cases of conjunctival tuberculosis is 150, of which but 100 were proven tuberculous.

VIII. Tuberculosis of the Alimentary Tract.—1. **Lips.**—The lips are very seldom affected; lupus or tuberculous ulcers may invade them. The latter are very painful and may be confused with cancer or chancre.

2. **Gums.**—The gums are seldom involved, though tuberculosis has followed caries and extraction of the teeth.

3. **Tongue.**—This is involved in 1 to 2 per cent. of cases. Tuberculous tumors in its substance are sometimes mistaken for gumma or cancer, and may break down into cold abscesses. Tuberculous ulcers develop on the dorsum or edges; they have rough caseous bases with uneven or undermined edges; they are almost never primary; they are differentiated from syphilitic ulcers by the inefficiency of mercury and iodides, from cancer by the absence of glands in the neck, and from both by the presence of bacilli and the results of inoculation.

4. **Pharynx.**—Pharyngeal involvement was known to Morgagni but was especially studied by Ricord (1865). Primary disease is most infrequent, and involvement, secondary to pulmonary or laryngeal phthisis, is also infrequent; Heller saw but 4 instances in 8,000 throat cases. The process begins largely in the lymphoid structures, which are infected by tuberculous sputum or food, or it extends by the lymph-vessels from the larynx. Three-fifths of the cases are associated with tuberculous disease of the intestine (Martineaux), and 15 per cent. with oral tuberculosis (Barth). The cervical glands may be affected in acute, but rarely in chronic, cases. The ulcerations are usually superficial; when propagated from the larynx, the most distressing dysphagia results. *Retropharyngeal abscess* usu-

ally results from suppuration of the lymph glands and from spinal or osseous disease.

5. Palate.—The palate is ulcerated in 1 per cent. of cases of phthisis; in coughing, tubercle bacilli from the sputum come in close or forcible contact with the palate. Lubinski observed three instances among 16,000 throat cases.

6. Tonsils.—The tonsils are diseased in 4 per cent. of cases of pulmonary tuberculosis (Warthin); their crypts contain bacilli; the process may stop at the tonsils, for they act as barriers to the infection or a tuberculous cervical adenitis may develop. Their infection may be primary, and Dieulafoy holds that tonsillar infection may be followed by a descending tuberculous adenitis, which in turn infects the lungs. Some maintain that the tonsils are infected by the sputum in half or more of the cases of phthisis. The lesions are more often miliary than caseous.

7. Salivary Glands.—These are fairly, but not absolutely, immune.

8. Œsophagus.—The œsophagus is infected less frequently even than the throat. No clear primary case is recorded, and Cone could collect only 48 secondary cases. Infection by contiguity, from the peribronchial glands, is more common than infection by tuberculous sputum; these glands may occasion œsophageal perforation, hemorrhage, diverticula or stenosis. Extension from the larynx or pharynx along the lymph tracts or from the spine or lung cavities, is possible. Miliary deposits have no clinical importance.

9. Stomach.—There are only four cases on record in which the smallest claim for primary involvement can be made. Secondary disease is estimated at four-tenths of one per cent. Glaubitt found 47 tuberculous ulcers in 2237 tuberculous autopsies. Ricard and Chevrier assert that there are on record sixteen cases of tuberculous stenosis of the pylorus. The HCl is inimical to gastric tubercle. Tuberculous ulceration of the stomach occurs from the sputum, from invasion by contiguity (glands, peritoneum or colon), or in few cases from hæmatogenous infection.

10. Intestinal Tuberculosis.—This was first observed by Bayle (1810). (*a*) It is usually *secondary* to ulcerative lung tuberculosis from infective sputum; its frequency is 30 per cent. (Eichhorst), 51 per cent. (Heinze) or 90 per cent. (Weigert, Orth, Warthin). Tubercle bacilli may be swallowed without causing intestinal tuberculosis. Of all intestinal ulcerations, tuberculous ulcers are the most frequent. Infection from the peritoneum, mesenteric glands or hæmatogenous infection is barely possible. (*b*) *Primary* intestinal tuberculosis is much more rare, occurring in 0.3 per cent. (Eisenhardt), and in only 0.1 per cent. of adult cases. The intestinal mucosa in children, like the bronchial, often allows of the passage of tubercle bacilli to the intestinal lymph glands without disease of the intestine itself; this is due to the loose infantile mucosa and the large lymph vessels. Infection may occur by infected milk. Koch takes an extreme view in denying infection by milk, and Behring takes the opposite extreme in affirming that it is the chief cause of tuberculosis.

The *ulcers* are most frequent in (i) the *ileo-cæcal* region and next in the colon. (ii) They always originate in the *lymphadenoid follicles* or Peyer's patches; they are promoted by erosions or obstipation. Small tubercles fuse into large ones, which caseate and ulcerate; in exceptional cases the caseous nodes do not rupture but cause the peritoneum and mucosa to bulge out. (iii) The ulcers are *irregular* in shape and (iv) their disposition is *ring-like; i. e.,* they are located transversely around the gut, because extension occurs by the lymph vessels (lymphangitis tuberculosa). Occasionally they lie in the long axis of the gut and are ovoid, corresponding, though incompletely, to Peyer's patches. (v) They are single or multiple, and sometimes cover nearly all the surface of both the large and small intestines. (vi) Their bases are rough or caseous and their edges infiltrated; the infiltration also invades the contiguous mucosa and submucosa. (vii) Localized adhesive peritonitis is a common issue when the process reaches the serosa. An early stage of this is seen in the serous and subserous miliary tubercles and in the bead-like tuberculous lymphangitis; perforation occurs in 5 to 10 per cent. of the cases, but is resisted by the muscular coat and by adhesions; it results in pericæcal abscess and in suppurative peritonitis. *Stenosis of the intestine,* single or less often multiple (129 cases collected by Arbuson, 1904), may also result; recovery is very rare. König in two years saw 5 cases recover, and Eisenhardt saw 10 complete and 26 incomplete instances in 567 tuberculous ulcerations. Hepatic suppuration or steatosis may result.

Symptoms.—The symptoms are those of any form of intestinal ulceration (*q. v.*); they usually commence with signs of catarrhal enteritis, diarrhœa, pain and fever. They may for a while resemble typhoid. The *only pathognomonic finding is the tubercle bacillus,* and it is possible that its presence may result only from swallowed sputum. Rosenblatt's method of detecting the *bacilli* in the fæces is to give laudanum until the stools become hard; in the mucus on their surface, the bacilli are usually found on the first examination, for the hard fæces apparently scrape the bacilli from the ulcers. *Pus* is often found and hemorrhage occasionally results. Girode called attention to the black stools, which are usually ominous, as they indicate malnutrition; the picture of dysentery is rarely observed. Fatal hemorrhage is infrequent, though cases have been recorded by Vallin, Hanot, Osler, Chandeze and others. An *appendicitic form* may be noted, with quick or insidious onset and with some induration; recurrence is frequent and fistulæ occasionally develop. In *typhlitis tuberculosa* there is great thickening in the cæcal region, with a hard, slightly movable or totally adherent, vertically disposed, tender tumor. The onset is usually insidious, pain is recurrent, symptoms of increasing intestinal stenosis are present and constipation alternates with diarrhœa. Hemorrhage is infrequent, fever is usually absent and emaciation is sometimes pronounced. Duguet described this condition in 1869, and, since it so fully resembles cancer or sarcoma,

it has attracted the especial attention of the surgeons, Bassini (1887), Bouilly (1889) and Billroth (1891). Campiche (1906) collected 379 cases of tuberculosis of the appendix and cæcum; 25 per cent. died from the operation; 35 per cent. were well for months or years; and 40 per cent. ultimately died of tuberculosis. The process usually begins in the mucosa and is marked by ulceration, villosities in the mucosa, great local infiltration and hypertrophy of the bowel and destruction of the ileocæcal valve. The process may begin, as shown by Conrath, in the serosa, with which tuberculous glands may come in contact. Patients may die of acute obstruction, or only after a chronic course of a decade or more; recovery by fibrosis is possible. Benoit and Dieulafoy regard the process as primary in 80 per cent. of subjects. It sometimes co-exists with cancer.

In the *rectum,* ulcers, either anal or rectal, lupus, tuberculosis cutis verrucosa and periproctal abscess are sometimes seen. *Fistula in ano* occurs in 1 to 5 per cent. of cases of phthisis, and rarely develops in other conditions; the general idea that its excision excites pulmonary disease is a confusion of cause with effect.

11. Liver.—The liver is involved less in adults than in children (38 per cent., Leroux), but at all ages it is of greater pathological than clinical interest. (i) Large tubercles may develop in the liver, sometimes with peritonitis and perihepatitis. (ii) Miliary tubercles, in 80 per cent., are of no clinical significance. (iii) Hanot maintains that there is a primary cirrhosis of the liver, associated with tubercles and fatty degeneration; at the most it is extremely rare; it is said to be associated sometimes with tuberculous peritonitis and perihepatitis (*v. s.* Tuberculosis of the Lungs: Complications).

12. Spleen.—The spleen is involved in 45 per cent. of tuberculous children (Leroux and Müller). Franke (1908) collected 29 cases of primary tuberculosis of the spleen; 19 were not recognized clinically; 10 were operated on, of which 7 permanently recovered. (See Diseases of Spleen: Splenomegaly with Cyanosis.)

IX. Tuberculosis of the Genito-urinary Tract.—Genito-urinary tuberculosis was described by Morgagni and by Bayle in the beginning of the last century, but for the first time fully by Lichtheim, in the cadaver, and by Rosenstein, Babes and Smith, clinically. It is most frequent in the period of greatest sexual activity (twentieth to fortieth year), and 72 per cent. occur in males, in whom the two tracts have more continuity and community of surface.

Modes of Infection.—1. Hæmatogenic Infection.—*This is the most frequent variety.* It occurs in 5 per cent. of cases of chronic pulmonary tuberculosis. The majority (76 per cent.) of cases are *secondary to some lung or peribronchial glandular focus,* which very often has produced no symptoms, so that most seemingly primary foci are really secondary. Jani found tubercle bacilli in perfectly sound testes and prostate glands, but they were clearly an invasion of the blood occurring in the death agony.

2. Urogenic Infection.—The general opinion previously was that a secondary infection mounted from the lower genital to the higher uropoietic structures. However, hæmatogenous *infection of the kidney descends* to the lower urinary tract, not *vice versa*. Infection by infected fingers, instruments, catheters or sexual intercourse is possible but has been grossly overrated. It may follow syphilis and gonorrhœa.

3. Infection from the Peritoneum and Contiguous Structures.—The *Fallopian tubes* are diseased in a third to a half of the cases of tuberculous peritonitis. Jani found tubercle bacilli in the tubes in women dying of phthisis. The bladder, seminal vesicles and vasa deferentia may be invaded *per contiguitatem* from peritoneal, vertebral or rectal foci.

1. Renal Tuberculosis.—The kidney in *miliary* tuberculosis is involved in 90 per cent. (Frerichs), but is marked by no distinguishing symptoms. The common clinical form is the caseous-ulcerative. *Caseous nodes* develop in the cortex, infection reaching it from the blood stream in 90 per cent.; they often break down into cavities, may form cold abscesses, undergo secondary pyogenic infection or the process may remain localized in one pole of the kidney. Tubercles also develop in the apices of the pyramids and pelvis of the kidney and form caseous infiltration and ulcers; in this form involvement was at one time considered the result of an ascending infection, though Caspar, Walker and Israel believe that the process in 41 per cent. of cases descends from the kidney to the bladder. Upward and downward extension in the kidney itself often occurs, so that the entire organ may be more or less diseased. When the process is well developed there is a tuberculous pyonephrosis. The entire kidney may become a large sac of caseous material, detritus and lime salts,—called *dégénérescence massive* by Tuffier. The kidney is often adherent to adjacent structures; its capsule is thickened, its removal strips off particles of renal substance and it is sometimes perforated, causing tuberculous paranephritis. The surface of the kidney may be smooth and normal, lumpy from caseous deposits or softened in many places from diffuse ulceration. In exceptional cases spontaneous healing is possible. Whether one or both kidneys are diseased depends on the stage of the process, and is a most important matter when surgical interference is considered. Israel, in his celebrated observations, stated that he found one kidney involved usually without any other genito-urinary disease. Pallet and Albarran observed unilateral disease in 80 to 85 per cent. of their cases, while post-mortem figures show unilateral renal disease in but 48 per cent.; naturally, in autopsy figures, bilateral disease is greater than in clinical figures. Compensatory hypertrophy of the sound kidney is frequent.

Symptoms.—These may be absent if the renal pelvis remains sound. Otherwise the symptoms are (*a*) urinary, (*b*) local and (*c*) general.

(*a*) *Urinary.*—The urinary symptoms are chiefly those of pyelitis (*q. v.*). The urine is acid, though alkalinity develops in complicating tuberculous or non-tuberculous cystitis, which may be determined by the cystoscope. Urination is often difficult (dysuria) or frequent (pollakiuria), so that the existence of cystitis is at first suspected; it is a safe rule always to *consider the possibility of renal phthisis in every cystitis,* the origin of which is not clear and the treatment of which is unsuccessful. Bladder symptoms are reflex from the kidney lesion, without any vesical involvement, or are due to complicating cystitis, simple or tuberculous. Guyon describes an early *polyuria* in which the urine is clear, and a later polyuria in which it is turbid. *Hæmaturia* (*q. v.*) is frequent; it may be severe or slight and its occurrence is more often early than late; its frequent recurrence is so marked in some cases as to justify the term "hemorrhagic type"; it may last one and a half months to four and a half years (Tuffier). *Pyuria* generally occurs when the pelvis is invaded, but is not observed when cortical foci do not communicate with the pelvis or when the ureter is occluded; the amount of albumin corresponds to the pus cells. Epithelial cells, sometimes caseous matter, elastic fibers and necrotic renal tissue are found. The sediment is often a crumbly, rapidly settling detritus; *tubercle bacilli* are often found, but must not be confused with the smegma bacillus (*v.* General Bacteriology); sometimes bacilli in the urine are found in pulmonary tuberculosis without any disease of the kidneys. Casts are very infrequent. In unilateral renal disease with occlusion of the corresponding ureter, the urine is naturally normal. Urinary segregation or ureteral catheterization determines which kidney is involved.

(*b*) *Local.*—Local symptoms are lumbar pain and tumor. *Lumbar pain* sometimes results from capsular tension; it is severe, colicky and reflected to the groins and testes, which may be retracted as in renal colic; colic is due to the passage of caseous particles or blood clots; differentiation from calculous disease (*q. v.*) may be difficult until tubercle bacilli are found. *Tumor,* in tuberculous pyonephrosis or hydronephrosis, is a small, tender enlargement, though seldom of the size attained in calculous or other obstructive disease.

(*c*) *General.*—General symptoms are hectic fever, malnutrition and other general toxæmic symptoms (see page 310), tuberculosis in the lungs and foci in the testes. The heart is almost never hypertrophied. In 60 per cent. of the cases operated on, the diagnosis was found correct. The course is chronic and remittent, two, three or even five, years being the usual duration; recovery from circumscribed lesions is possible; death results from tuberculous generalization, less often from sepsis, perinephritis or amyloid degeneration, and rarely from nephritis or uræmia. The *treatment* is surgical. In Krönlein's cases, the operative mortality of nephrectomy was 5.8 per cent. and the ultimate mortality was 17.6 per cent.; 70 per cent. recovered. In Albarran's series the operative mortality was 3 and in Caspar's 5 per cent.

2. Ureter and Bladder.—The ureter is thickened, infiltrated, caseous, ulcerated and often stenosed; it is involved in 80 per cent. of cases of renal tuberculosis (Palet). Secondary involvement from the kidney may be limited to the ureter at its vesical ending or may spread more widely over the mucosa of the bladder.

Bladder disease is almost invariably secondary *per contiguitatem* or from hæmatogenous deposits, as seen by means of the cystoscope. Israel (*v. s.*) noted tuberculous cystitis in 41 per cent. of cases of renal tuberculosis, and Vignernon in 50 per cent. Cystitis of long standing should always excite suspicion of tuberculosis, even when it has gonorrhœal or other antecedents. The diagnosis is based on the specific bacteriology and the cystoscope. Rosving cauterizes with carbolic acid. Surgical intervention is indicated.

3. Male Genitalia.—The *epididymis* is more often involved than the testis, except in youth, when the epididymis and tunica albuginea are more frequently affected; in the first years of life testicular disease results from generalized tuberculosis; about one-half of Jullien's cases occurred under two years of age. Sometimes no caseation results, as is the case in hyperplastic lymphadenitis, and even microscopic confusion with sarcoma is possible unless the bacilli are particularly sought. Testicular tuberculosis is most often confused with syphilis (*q. v.*), though in the latter there is less pain and fever and more nodular enlargement. The seminal vesicles, vasa deferentia and urethra may be invaded and a rectal examination should never be neglected. Recently the prostate, "the cross road of the urinary and genital tracts," has attracted especial attention; its primary involvement (Lancereaux) is disputed by some, but the gland is affected in 50 to 80 per cent. of genital tuberculosis in the male. The lungs are invaded in primary genital tuberculosis in 70 per cent. and the urinary tract in 56 per cent. *Treatment* is surgical.

4. Female Genitalia.—The female genitalia become tuberculous in 1.5 to 2.5 per cent. of pulmonary tuberculosis. Infection is secondary in 80 to 100 per cent. *Tuberculous tubes* are found in 1.5 per cent. of abdominal operations; according to W. Williams, 8 per cent. of the tubes removed for inflammatory lesions were found tuberculous. Nearly all cases are due to some small focus in the *bronchial glands.* The tubes show nodules, infiltration, caseation and ulceration; bilateral involvement is usual, and its true nature may be apparent only under the microscope; tuberculous salpingitis may simulate tuberculous peritonitis; tubal disease may be found in very young children. The uterus ranks next and is, as a rule, involved secondarily; systematic microscopic examination would doubtless show higher percentages than those usually given. Ovarian phthisis is almost always secondary. Tuberculosis of the vagina, cervix, vulva and female urethra are most exceptional.

Mammæ.—Schley (1903) collected 65 cases of which 12 were primary. Carcinoma may be closely simulated, for tuberculous

lesions often cause nipple retraction and tumor formation or involve the axillary glands (66 per cent.). When cold abscesses, fistulæ or ulcers form, the diagnosis is naturally more definite. A chronic interstitial non-tuberculous mastitis was described by Bedor.

X. Tuberculosis of the Upper Respiratory Tract.—1. Nose.—The nose is seldom affected, because its irritation excites lachrymation, sneezing and an increased secretion, inimical to the tubercle bacilli. Nasal localization was first described by Willigk (1853). In the rare primary form there are very few bacilli and an exuberant non-caseous growth, which may resemble sarcoma; as in lymphadenopathies and in the Fallopian tubes the absence of caseation is not unique; there is little pain and the chief symptom is a chronic coryza. In the secondary form the bacilli are abundant; multiple caseation and ulceration occur as in other localizations; suppressed coughing, with the mouth closed, is thought to force germs into the nose. Nasal infection may occur by picking the nose, and is promoted by wounds, ulcers and stagnation of secretion by various obstructive processes. Infection may spread by the lymph vessels, especially in children, to the cervical glands or even to the meninges. In 33 per cent. lupus affects the nose (Bender).

2. Nasopharynx.—Its primary involvement is infrequent. Secondary disease, usually late in phthisis, occurs in 20 per cent.; it is promoted by coughing with the lips closed. It may extend by contiguity from the pharynx. The third tonsil may be affected (Lermoyez); adenoids are tuberculous in 20 per cent. (Dieulafoy).

3. Ear.—Tuberculous *otitis media* occurs chiefly in late phthisis (according to Fränkel in 32 per cent.); otitis may occur from mixed infection. It is furthered by measles and scarlatina, is rarely primary, and often causes cervical lymphadenitis.

4. Larynx.—Laryngeal phthisis is very rarely primary, though some well established cases are recorded. Louis, in 1825, held that it was secondary to lung disease, resulting from infection by sputum, which view still holds; hæmatogenous and lymphogenous infection is rare. Laryngeal tuberculosis develops in 20 to 30 per cent. of pulmonary tuberculosis in adults, and in only 3 per cent. of pulmonary phthisis in children (Parrot). It complicates 9 per cent. of lupus cases. Adjuvant factors are inflammation, fissure and syphilis. In 700 cases Krieg observed unilateral involvement in 275 (39 per cent.), of which 92 per cent. occurred on the same side as the lung lesion; Kreig attributed this to lymphatic extension; Cornet refers it to paresis of the vocal cord on the affected side (which is due to compression or neuritis of the recurrent laryngeal nerve), and to stagnation of the sputum in various recesses on the paralyzed and anæsthetic side. Unilateral disease usually becomes bilateral from contact.

Symptoms.—The *cough* varies with the time of onset of laryngeal symptoms; *in those rare and unfavorable cases* in which the trouble

begins with laryngeal symptoms, the cough is often somewhat dry, brassy or metallic; it may come on in the form of acute laryngitis. *In cases clearly following lung disease,* the ordinary bronchitic hack alters and frequently becomes brassy; in the last stages it becomes incomplete, ineffectual or like an eructation (Trusseau), as the vocal cords cannot come in apposition. *Pain and hoarseness* are followed by *dysphonia* (or, later, *aphonia*), *dyspnœa* and the most painful and distressing *dysphagia.*

Forms.—(*a*) The most common form is the *ulcerative;* ulcers are found in the interarytenoid region, vocal cords, posterior wall of the larynx and arytenoids; they are not deep but are broad; their edges are "nibbled," and, on their rather pale and sometimes caseous bases, papillary excrescences are fairly frequent. Particles may be inhaled into the lungs, causing tuberculous or suppurative foci. They are to be distinguished from syphilitic ulcers (*q. v.*), which are more often single and redder, have more "cut out" edges, frequently show cicatrization, involve oftener the parts nearer the epiglottis and posterior surface of the arytenoids, and less often attack the vocal cords; show no tubercle bacilli, do not show local reaction on tuberculin injection and do respond to mercury and iodides. Ninety per cent. of all laryngeal necrosis is tuberculous. Perichondritis may occasion sudden œdema glottidis, urgent dyspnœa and early death unless timely tracheotomy is performed. (*b*) In other cases the *submucous tissues are infiltrated* and the mucosa is thickened or hardened (pachydermous form); the true cords are less involved than other parts; stenosis rarely occurs, and if ulceration develops it is inconspicuous. (*c*) *Caseous tumors* are not frequent; they may precede the ulcerative form or occur independently of it. (*d*) The *vegetative type* occurs with ulceration, and small excrescences develop on the floor of the ulcers, or without ulceration, in which case they appear on the otherwise intact mucosa. (*e*) The miliary form is unimportant.

Treatment.—Treatment is unsatisfactory, as pronounced lung changes already exist and dysphagia occasions rapid emaciation and disinclination to eat. Besides general treatment (*v. i.*), local measures may be employed, but as a rule they do not essentially modify the process. Insufflations of boric acid, iodoform or iodol and painting with 10 per cent. menthol, 10 per cent. carbolic acid or 20 per cent. lactic acid solution (gradually increased to 80 per cent.), may relieve the local pain; gr. $\frac{1}{4}$ of morphine hypodermatically and 10 per cent. solution of cocaine locally, allow the patient to eat, for a time at least, but finally fail to anæsthetize the widespread ulceration. The patient may be able to draw up milk and water by a tube, when the head is turned over the edge of the bed. Caustics, such as silver nitrate, are injurious. In Casselberry's practice, 10 recovered.

XI. Tuberculosis of the Heart and Vessels.—(*a*) Pericardium: (*v.* Serous Membranes). (*b*) Myocardium: Miliary tubercles are occasionally seen; tuberculous fibrous myocarditis is quite rare;

caseous nodes are very uncommon and occur chiefly in the young; Anders in 1902 collected but 72 cases. (*c*) Endocardium: Weichselbaum, Birch-Hirschfeld, Klebs, Kotlar and Chiari have found tubercle bacilli in cardiac thrombi; Heller, Kundrat, Tripier, Londe, Petit and Courmont found them in endocarditic vegetations; Hanot holds that toxins cause the excrescences in which the bacilli are later deposited; Norris found endocarditis in only 1.4 per cent. of over 11,000 phthisical necropsies; tuberculous endocarditis is almost always vegetative and very rarely ulcerative or caseous; secondary infection is the cause of some cases of endocarditis which complicate tuberculosis. (*d*) Involvement of the vessels has been described under miliary tuberculosis. Primary disease of the vessels seems highly improbable, though some few cases are reported; 18 cases of tuberculosis of the aorta were collected from the literature by Simmitsky.

XII. Tuberculosis of the Bones and Joints.—This topic properly belongs to surgery, but its relations to glandular, pulmonary and other lesions demand some consideration. Koch was the first to demonstrate the almost invariably tuberculous nature of caries, fungus joint lesions and tumor albus, and Damsch first determined this relation by inoculations. Schuchart, Krause, Demme and Renke proved that spina ventosa was usually tuberculosis. König and Orth found that about 80 per cent. of bone and joint tuberculosis was clearly secondary to pulmonary, glandular, genito-urinary and other tuberculosis. Probably *all cases are secondary* to tuberculosis elsewhere. Infection is *hæmatogenic* in the majority of cases, far less often lymphogenic. *Trauma* has long been considered an important etiological factor, and experimentation shows that it may determine localization in animals already infected with the Bacillus tuberculosis. This factor has probably been overestimated, and Sprengel even maintains that the joints oftenest involved are those least subject to traumatism. Thirty-three per cent. of the cases occur in the first decade of life, and 50 per cent. in the first two decades. Tuberculosis in children involves the bones and joints in 22 per cent. of cases (Müller).

Localization.—Watson Cheyne, from his own and other cases, estimates that the knee is affected in 16.5 per cent. of cases, the hip in 14.6 per cent., tarsus and ankle in 14.4 per cent., elbow in 6.3 per cent., skull and face in 5.5 per cent., sternum, clavicle and ribs in 5.2 per cent., pelvic bones in 3.5 per cent., femur, fibula and tibia in 3.5 per cent., spine in 2.3 per cent., shoulder in 1.5 per cent. and scapula, ulna and radius in 1 per cent. Jaffe finds involvement of the spine in 20 per cent., foot in 21 per cent., hip in 13 per cent., knee in 10 per cent., hand in 9 per cent. and elbow in 4 per cent.

1. Bones.—Miliary tuberculosis affecting the bones produces no symptoms. In the ordinary type of infection the spongy epiphyses of the long bones are the particular seat of tuberculous osteomyelitis

and periostitis, whence infection easily reaches the joints; the diaphyses and flat bones are seldom involved. Involvement of the short bones of the fingers and toes, and less often of the ulna, may cause bulging,—the *spina ventosa,* which is seen oftenest at about five years of age. In the bone-marrow there develops a grayish-red granuloma which dissolves the bone (*caries sicca*); the trabeculæ necrose and are discharged through fistulæ as sequesters or "bone sand," in a caseous, pus-like fluid. Even large foci may heal by granulating osteitis, in the same manner as tuberculous adenitis may heal. Infection may remain unobserved or semiquiescent until aroused by infections as pertussis or measles or by trauma. *Cold abscesses* in the bone are lined by granulation tissue. There may be local tenderness, stiffness, fever and nocturnal pain, varying in intensity and sometimes radiating. Bone lesions are not incompatible with seemingly perfect nutrition and otherwise robust health.

2. Joints.—Involvement may occur alone or follow contiguous osseous disease. Very often there is synovial inflammation and proliferation; far less frequently there are isolated tubercles in a non-inflamed synovial sac. Serous or serofibrinous synovitis (or the rice bodies, *corpora oryzoidea*) may result from toxins in a neighboring focus in the bone, without the actual presence of tubercle bacilli in the joint, exactly as toxic pleurisy or pericarditis may result from contiguous pulmonary or glandular foci. The *symptoms,* or onset, may be acute or insidious. Pain, especially at night, contractures and fever may be noted. In the later stages of joint disease, granulation tissue forms with large caseous deposits, hydrops, empyema articulorum or the tumor albus (so named because of the thickening of the connective tissue, the œdema and glistening appearance of the periarticular structures). The joint is sometimes quite disorganized. Seventy-five per cent. recover under conservative treatment, as immobilization, iodoform or formalin injections, soap inunctions, cod-liver oil and sea air.

Within a few years Poncet first, and then Leriche, Patel, Trebeneau, Bezancon and Griffon have called attention to a *tuberculous pseudorheumatism,* in which the incipient arthritis closely resembles genuine rheumatism (*rheumatisme tuberculeuse ankylosante*). It is thought by these French writers that the exuded leukocytes are strictly of the lymphocyte type; inoculations have proven positive in a number of cases, but toxæmia is the most probable cause (Mohr). It is most frequent in children. Lejars found one case of primary tuberculosis of the muscles.

XIII. Tuberculosis of the Skin.—1. Lupus.—Lupus is the most frequent form. In 66 per cent. of the cases it is located on the nose, lips and angle of the eye. Besnier found it with pulmonary tuberculosis in 21 per cent.; Sachs and Bender found other tuberculosis in 62 per cent. and Block in 79 per cent. It is typical granulation tissue with true tuberculous tumors, which contain few bacilli be-

cause of the low temperature of the skin, and it often advances on one border while it cicatrizes on another. Its various forms, such as lupus maculosus, exulcerans or hypertrophicans need not be described in detail.

2. **Scrofuloderma.**—Scrofuloderma, the subcutaneous granuloma of Neisser, appears as movable, painless, subcutaneous nodes, which later become cold abscesses (the so-called scrofulous gumma, *gomme scrofuleuse* of Despres and Besnier); they discharge a milky fluid and leave a sharply marked granulating ulcer.

3. **Lichen Scrofulosorum.**—Lichen scrofulosorum is often but not always tuberculous; the eruption originates from the follicles, is constituted of thick, oily, epidermal cells which can be scratched off without bleeding, and is usually distributed in sharply marked groups of isolated yellow or yellow-brown nodules as large as a pinhead.

4. **Tuberculosis Cutis Verrucosa.**—Tuberculosis cutis verrucosa of Riehl and Pautauf is observed on the backs of the hands and fingers and in the interdigital folds, chiefly in butchers, cooks and horsemen.

5. **Postmortem Tubercles.**—Postmortem tubercles were described by Verneuil, Verchère, Pick and Pfeiffer. Laennec referred his pulmonary phthisis, though probably incorrectly, to a postmortem tubercle. Gerber suffered from tuberculosis of the axillary glands following a tubercle. They are observed in butchers, veterinary surgeons and pathologists, especially in those beyond middle life.

6. **Tuberculous Ulcers.**—Tuberculous ulcers may develop from cuts by broken sputum cups; from bites of tuberculous subjects, ants or flies; from burns, tattooing, skin grafting or leeches; and from circumcision when the wound is sucked by tuberculous rabbis; lupus has followed vaccination.

Scrofula is tuberculosis; however, in descriptions dealing with scrofula as a separate clinical picture, affections of the mucous membranes are often associated with the skin lesions above described, with adenopathies (*q. v.*) and with bone or joint disease. Among these may be mentioned chronic ozæna, rhinitis, adenoids, friable carious teeth, otitis media, blepharitis, conjunctivitis or phlyctænular keratitis, bronchitis, intestinal catarrh and leukorrhœa.

Treatment of Tuberculosis.—(*A*). **Prophylaxis.** 1. Antibacillary. Antibacillary prevention concerns the etiological factors in the dissemination of the bacillus. Cleanliness of the skin; disinfection of contaminated fæces, urine or pus; confiscation of diseased meat; and regulation of dairies and inspection of cows are important; but the *destruction of infective sputum* is the prime indication. It is well to regard all sputum as dangerous, and to teach the public and patients this doctrine. Tuberculous, as well as pneumonic, grippal and other sputa must be destroyed. An appeal must be made to the conscience or selfishness of tuberculous patients, setting forth the risks of autoreinfection. Of all educators, Biggs, of New York, and Flick, of Philadelphia, have rendered the greatest service. Patients should

carry small spit cups, of which Dettweiler's and Major Appel's are the best; the sputum should be destroyed by burning or boiling before it dries. Spittoons are dangerous. The patient should sleep alone, and kissing must be interdicted. The bedclothes, linen and eating utensils should be carefully steamed or boiled. Rooms should be cleaned with moist cloths and should be swept with the windows and doors open. The danger of swallowing sputum must be clearly set forth, and the mouth should be washed out with some antiseptic solution before eating, though a few bacilli with the food are not very dangerous, as the gastric juice probably destroys them. The more minute details are as impossible to describe as those of consistent surgical asepsis, though they are equally logical and necessary. In Germany the reduction of mortality by 33 per cent. is clearly due to an awakened public intelligence.

2. Governmental.—Governmental prophylaxis, somewhat less promising in republican than in autocratic states, is important. It comprises: Education of the public by publication of the dangers and preventive measures in tuberculosis, as in the recent excellent and plain pamphlets of the Illinois State Board; the establishment of sanatoria for the poor, among whom the mortality is four times, and the morbidity many times greater than in the rich; the building of separate hospitals, or at least wards, for the hopelessly diseased; and the regulation of the air capacity of dwelling rooms in tenements, factories and stores. Aside from great suffering and sorrow of thousands in every large community, there is an enormous economical waste in work and wages, an enormous cost in hospital care and a great loss in live stock,—over 62,000 cattle and 56,000 hogs were condemned in Prussia alone in one year. The comparative sociological damage is apparent, as the ravages of tuberculosis involve the working years of life (15 to 60), while 94 per cent. of deaths from diphtheria occur under ten years.

3. Individual.—Individual prophylaxis includes the increasing of physiological resistance, the maintenance of general health and the aiding of sound development in children. Weakly children should be brought up in the open air, judiciously fed, watched during acute infections, kept but few hours in school, sent into the country during vacation, taught moderation and later, should be informed as to the danger of alcoholism, sexual excesses and infections.

(*B*). **Hygienic Treatment.**—The *three great factors are fresh air, proper food* and *rest*. Bodington (1839), in England, treated cases by fresh air, but to Brehmer (1850), in Germany, is due the credit of soundly establishing the fresh-air treatment and the curability of phthisis. A few of his views are untenable, but many of them are the basis of the modern methods practiced by McCormack, Driver, Dettweiler, Trudeau, Knopf and others. Patients were treated in sanatoria, and those at Nordrach and Saranac and many others, have given such excellent results that institutions of this class are growing in number. Results depend on (*a*) the extent of the disease;

(*b*) the condition of organs other than the lungs, as the pleura, intestines, larynx and heart; and (*c*) the social and financial status of the case. Dettweiler states that 30 per cent. of his cases recovered and 40 per cent. improved; Trudeau brought about recovery in 31 per cent. of all, and in 66 per cent. of incipient, cases; Turban considers that 88 per cent. recover; Rumpf followed 97 per cent. of his cases: 70 per cent. of incipient cases were able to work—"the best test of a cure"; 55 per cent. of those in the second stage, and 23 per cent. of those in the third, recovered. One of the chief values of sanatorium treatment is that the patient learns the lesson of living properly, which too often in general practice is not sufficiently impressed upon him.

1. Fresh Air.—This is the chief hygienic factor in treatment, at home, in sanatoria or in change of climate. (*a*) *Treatment at home,* for financial reasons, is especially important, as but 5 or 10 per cent. of tuberculous people are able to leave home. The patient should be carefully clothed and should recline in the sun with the windows of his room open, or should lie on a veranda. He should be outdoors for one-quarter to one-half the sunny hours, according to the season of year. At night the windows should be kept widely open; the patient's bed can be brought close to an open window, from which "a window tent" of canvas encloses the head and leaves him out-of-doors. Rain, snow, dampness and extreme cold are no contra-indications, nor are fever, cough and hæmoptysis; but wind, dust and sudden variations in temperature are to be avoided. Very thick clothing, so frequently observed among the poor, is unhygienic. Acute caseous and advanced types should generally be kept at home. Extreme care in disinfection is imperative lest the home itself become a menace to the family. (*b*) *Sanatorium treatment* offers the advantage of strict discipline, systematic living and the constant presence of a physician who regulates the details of every-day life, gives explicit directions and cheers the patient. Sanatoria should be located near, but well outside of, every large centre of population. (*c*) Change of *climate* was once considered absolutely indispensable; now it is said "to be not without influence." No climate is specific. *High altitudes* offer the advantage of purer air, stimulation of breathing, increase of vital lung capacity, increase of the chest dimensions, growth of the muscles of inspiration, increase of metabolism and stimulation of the appetite and blood-making organs. Colorado, Arizona and New Mexico (four to seven thousand feet elevation) are excellent for cure and for opportunities for entering some permanent occupation after recovery; because of the emphysema induced by altitudes, a return to the sea level should be avoided. St. Moritz, Les Avants and Davos (5,200 feet elevation) offer a dry, clear, cold, still atmosphere, where patients may reside in summer and winter; it resembles the climate of Colorado, Montana and northern Wyoming. There are also the Peruvian Andes and numerous other altitudes, for details of which see *Solly's*

Medical Climatology. High altitudes are best adapted to suspected cases, to those with limited or incipient lesions, to those with slight cavity formation and little emaciation, or to more advanced but slowly progressing, or torpid, cases. Slight fever or slight hæmoptysis are not contra-indications to such climates. Emphysema, weak heart, constant fever, great nervousness or repeated hæmoptysis are contra-indications. *Moderate altitudes* (2,000 to 2,500 feet) probably best serve the majority of cases, as Asheville (2,250 feet), the Adirondacks (2,000 feet), Aikin (2,250 feet), Summerville, Thomasville, etc. The climate of Minnesota, Nebraska and Dakota (1,000 feet elevation) is cool and dry; dryness is usually more desired than lack of variability. Moderate elevations do not induce emphysema, whence return to the sea level is easier. The *sea level* is the best location for the tuberculous aged, for quiescent cases with great cicatrization or extensive damage, for those with diabetes, albuminuria, nephritis, scrofula, bone disease, marked anæmia, repeated hæmoptysis, emphysema or rapid softening, for those with incurable forms, for thin, nervous subjects and for those with exophthalmic goitre. Sea voyages are always somewhat experimental, and Robin holds that but 8 per cent. of cases are benefited by them; those with incipient forms, or those with genito-urinary, glandular or osseous disease or with open wounds, are often helped. Moist warm climates (at the sea level) are especially adapted to laryngeal disease,—as the Bermudas, Florida, the Madeira or Canary Islands; patients with dyspepsia and diarrhœa do well in Corsica, Palermo and Capri. Warm, dry climates, as southern California, Egypt, Algiers or the Riviera are good for catarrhal types. Climate alone, without proper rest, diet and reasonable hygiene, rarely benefits.

2. Nutrition.—Practice differs; most physicians recommend 5 or 6 meals daily, but some achieve better results by giving but three meals, whereby the stomach is allowed some rest. Feeding varies with individuals as to the amount and as to the kind of food. The *staple diet* is *meat, eggs and milk,* including cream and butter. A liberal mixed diet should be given of meat, fatty meats and fish; well-cooked leguminous vegetables (which are advantageously given in soups), butter, oil on salads, eggs, milk, cream, bread, pastries and cakes. Anorexia is a common obstacle to full feeding. If it is of the nervous type, patients must be compelled to eat, but long hours in the fresh air usually stimulate the appetite. With habitually light eaters caution is necessary; beef juice, milk and koumyss should be given; the amount should be gradually increased and other foods should be added. Faradization of the epigastrium, cold compresses over the stomach, wine, vermouth, the fluid extract of condurango ♏xv–xxx (Immermann), or strychnia, gr. $\frac{1}{30}$ in solution, half an hour before meals, are valuable appetizers. Raw beef (zomotherapy), especially recommended by Richet and Hericourt, has lately come into use; fresh lean meat is cut, soaked for two hours in one-fifth its weight of water, is then pressed to extract the blood and fluid, and

administered, 1 to 2 pounds daily for moderately sick cases to twice that amount for severe cases; it must be freshly prepared each time, as it soon becomes tainted. Raw eggs are often given between meals, beginning with 1 and increasing to 4, three times daily, the taste being disguised by orange juice. Fever does not contra-indicate full- or overfeeding. Milk should be given, up to 1 or 1½ quarts daily; more than this amount induces gastric atony; it is given to advantage with bread, crackers or toast, in order to divide the curds; it may be peptonized, disguised with cocoa or cognac or given by the rectum (*v.* GASTRIC ULCER); cream and butter are excellent substitutes for cod-liver oil and buttermilk is indicated when the stomach is weak. Fats and carbohydrates are necessary to offset nitrogenous waste; the dark form of cod-liver oil (*v. i.*), lipanin (94 parts olive oil and 6 parts oleic acid), malt, honey, cocoa, nutrose, eucasin and somatose are valuable foods. In all forms of overfeeding (Debove's *suralimentation*) the tolerance of the stomach must be considered, for this is more important than the use of drugs (creosote, cough syrups or cod-liver oil). Debove's method consists of gastric lavage followed by the introduction through the tube of three ounces of powdered meat, a quart of milk and one or more eggs. Alcohol at the present time is administered less often and in smaller amounts than formerly, but is frequently indicated symptomatically (*v. i.*); small amounts of red wine or cognac and whiskey in egg-nogs are very valuable as a food and tonic. Tobacco should be interdicted; in special cases a cigar or two daily may be allowed, but the smoke must never be inhaled.

3. REST.—Rest is one of the essentials of Dettweiler's therapy. Exercise destroys tissue, often induces anæmia and irritates the heart; it is distinctly contra-indicated by fever, emaciation, hæmoptysis, rapid pulse, nausea and coughing. It has been well said that "too many cases of phthisis *walk* into their graves." Late in the treatment graduated exercise may be taken. Pulmonary gymnastics and pneumatic differentiation are clearly injurious. Patients should lie in the bright sunshine and fresh air, and active mental occupation should be carefully avoided. Hardening and exercise should be obtained by quick, dry rubs with a large coarse towel, after which rapid partial, and then general, wet rubs may be instituted; cold rubs and hydrotherapy are to be shunned when there is any tendency to hæmoptysis.

(*C*). **Specifics.**—As yet there are no specifics for tuberculosis and drugs are only of symptomatic value.

TUBERCULIN THERAPY.—(i) The *indications* are: Incipient subjects who are well-nourished, without fever and come to a stand-still after some improvement. (ii) The *contra-indications* embrace malnutrition, rapid wasting, acute phthisis, rapid heart action, nephritis or great nervousness. (iii) *Dosage.* Carelessly used, tuberculin is dangerous. One mg. of the "old tuberculin" (O. T.) is too large; the initial dose should be 0.00001 gm.; the initial dosage for the "bacillus emulsion" (B. E.) is 0.000001 gm., increasing gradually

towards the decimal point. The maximum dose of O. T. is 1 mg. and B. E. 5 mg. and the course lasts 5 months but is interrupted at once when coryza, cough, indigestion, fever, emaciation or hæmoptysis intervene. (iv) *Mode of action:* It is thought that the injections stimulate the formation of agglutinins and opsonins; the latter are bodies which, combining with bacteria, make them an easier prey for the phagocytes. (v) *Results.* Trudeau conservatively states his belief that "tuberculin immunizes, prolongs life, causes the bacilli to disappear from the sputum, aborts commencing infection and also the smouldering fires of chronic infection." The subject is still *sub judice* and perhaps as one writer puts it, "a chaos of personal impressions."

(*D*). **Expectant or Symptomatic Treatment.**—1. Fever.—Rest, quiet, digestible food, fresh air or change of climate are indicated in great preference to drugs. Brehmer administered strong wine at the time the fever began to rise; arsenic is recommended by Pollack and Powell, but no remedy in safe doses controls fever. Quinine disturbs the digestion. Sponging is fairly efficacious.

2. Cough.—A slight morning cough serves to remove secretion and needs no restraint, but, if excessive, it begets coughing by congesting the lungs and tubes; the dangers of excessive coughing are exhaustion, depression, insomnia, aspiration and vomiting, which necessitate interference. Fresh air and avoidance of dirt, dust and smoke are most beneficial. Rest in general, and especially after eating, should be enforced. The cough can be suppressed, for it is somewhat under the control of the will, as Galen knew; the comment on the absence of coughing, especially at the table, is universal among observers of sanatorium methods; slow breathing, with the head slightly thrown back, moist compresses or the sipping of warm water, with 10 grains of salt, before meals and resting often aid in the suppression of the emetic cough. *Creosote* was discovered by Reichenbach, in 1832, and was used by Addison, Sonnenberg and Jaccoud; it is not specific, but often increases the appetite, modifies abundant secretion from the bronchi (by which the drug is in part eliminated), and relieves the cough. Administration in the fluid form is better than in capsules. Beginning with one drop (well diluted) after meals, the dose may be increased to ten times that amount, but full dosage often irritates the stomach and kidneys and necessitates constant supervision of the urine; it should not be given in febrile cases or in hæmoptysis. Creosote may be administered in milk, in water with the compound tincture of gentian (ʒiij) or in sherry wine.

℞ Creosoti ..... ʒiss.
Glycerini ..... ʒss.
Tr. gentianæ co. ..... ʒiv.

M. et S.—One teaspoonful after meals.

Creosote may be given in pill form as follows:

℞ Creosoti ..... ℥iss.
Mucilag. acaciæ ..... ℥iij.
Pulv. althææ .....q.s.
M. et ft. pil. no. 100.

S.—One after meals.

Guaiacol carbonate (gr. v, *p. c.*) is often better tolerated by the stomach than creosote. Jacobi recommends the following formulæ as efficient and very cheap.

℞ Guaiacolis ..... 50 parts.
Tr. nucis vomicæ ..... 40 parts.
Tr. strophanthi ..... 30 parts.
Liq. potassii arsenitis ..... 10 parts.

Sig.—A half teaspoonful three times a day in hot milk.

℞ Guaiacolis carbonatis ..... ℥iij.
Strychninæ nitratis,
Arseni trioxidi .....āā gr. iss.
Sparteinæ sulphatis ..... gr. xlv.

M. et div. in chartulas no. 40.
Sig.—One powder after meals.

Cod-liver oil, first used by Hughes-Bennett, is usually given in doses so large as to disturb gastric and intestinal digestion; the first dose should be 10 drops and the amount should be gradually increased. It is more valuable in tuberculosis of the glands and bones than in the other types. Eructations can be avoided by administering it one or two hours after meals, when the food begins to leave the stomach. The oil is readily absorbed, because of the bile which it contains, and is possibly somewhat alterative; but it is not very superior to butter or cream, and is really more a food than a medicine. Cod-liver oil may be floated on whiskey, beer or milk, and gulped down. Its taste may be partly disguised by a "chaser" of whiskey. Syrup of orange disguises its taste fairly well. It is seldom given when there is fever or indigestion. It is prepared in large capsules, which obviate its unpleasant taste. *Opiates* may be used, such as paregoric; codeine gr. ¼ with aq. laurocerasi and glycerine āā ♏xv and syr. tolutani ℥j q.i.d.; dionin gr. ¼ q.i.d.; heroin gr. $\frac{1}{12}$ q.i.d.; even morphine sulph. gr. ¼ with ac. hydrocyanici dil. ♏ii. Other remedies are myrtol gr. ii. A.M. and P.M.; terpin hydrate gr. ii to v, t.i.d.; ol. terebinthinæ, ♏ij and for unproductive coughs, spts. chloroformi or spts. ætheris co. ℥ss in whiskey, p.r.n. (Various formulæ are given under BRONCHITIS.)

℞ Tr. belladonnæ ..... ℥ss.
Spts. chloroformi ..... ℥iss.
Mucilag. acaciæ ..... ℥j.
Aquæ .....q.s. ad ℥iij.

M. et S.—One teaspoonful after meals, or as indicated.

Expectorants are to be avoided because of their nauseating ten-

dencies. Tr. benzoin co., 1 per cent. phenol, and creosote may be vaporized. Menthol and guaiacol āā may be taken internally in olive oil. If the cough is nasopharyngeal, the mucosa should be painted with iodine, potas. iodide and glycerine, in proportions of 1, 10 and 100; if laryngeal, 20 per cent. menthol should be applied locally (*v.* LARYNGEAL TUBERCULOSIS); if due to enlarged bronchial glands, inunctions of green soap and potas. iodide internally, should be given.

3. SWEATS.—The room should be cool at night, the windows open, the bed not too heavily covered, and the body protected by a flannel night-gown. A glass of cold milk, with ℥ij of cognac, given at bedtime, is often beneficial. Morphine on retiring is indicated when the sweats are associated with cough and insufficient oxygenation. Atropine gr. $\frac{1}{100}$ to $\frac{1}{80}$, and camphoric acid gr. xv to xxx, at bedtime, are the most reliable remedies, and are superior to aromatic sulphuric acid ♏x to xx, agaricin gr. $\frac{1}{16}$ to $\frac{1}{12}$, tr. nucis vomicæ ♏xxx, picrotoxin gr. $\frac{1}{60}$ and muscarin ♏v of a 1 per cent. solution.

℞ Quininæ sulph. ..... ℥ss.
Calcii hypophosphitis ..... ℥j.
Extr. nucis vomicæ ..... gr. v.

M. et ft. pilulæ 20.
S.—One after meals.

4. HÆMOPTYSIS.—Exertion, loud talking or singing and excesses in alcohol, venery, coffee or tobacco, must be shunned in all cases. It is well to warn patients of the possibility of hæmoptysis, in order to prevent undue excitement, if it appears. Effusive pleurisy should not be tapped in a subject who has recently bled from the lungs, as thoracocentesis has produced fatal hæmoptysis. The chief indication is absolute rest, physical and mental; the patient should be kept in the semirecumbent posture; silence on his part should be enforced; massage and stimulants are contra-indicated; no physical examination should be made except by auscultation, as percussion is obviously dangerous; and a hypodermic injection of morphine should be given to lessen cough, to quiet the heart, to allay the excitement attending most hemorrhages and also possibly for its slight hæmostatic effect. As the tonus of the pulmonary vessels is beyond regulation, ergotin, tannic acid and other hæmostatics are useless, though digitalis and fresh suprarenal extract gr. ii to v sometimes seem efficacious. Salt ℥j is a popular remedy and is supposed to act by stimulating the vagus. An ice-bag should be placed over the heart and one over the lung, if rales are heard. Deep breathing seems to lessen bleeding. *Calcium lactate* gr. xv t.i.d. for 2 days is valuable. *Amyl nitrite,* recommended by Francis Hare, is given about midnight, when the vasomotor centre is regaining its tone lost during sleeping. Ol. terebinthinæ ♏ij to iij is considered the best drug by Fräntzel. In severe or obstinately recurrent hemorrhage, bandaging of the extremities (Hippocrates), to lessen the return flow of blood, injections of gelatin, induction of pneumothorax by gas injection or puncture

(Murphy, Cayley), or even emetics to stimulate the vagus (Fränkel) may possibly be indicated. Rest and the administration of morphine, however, are usually effective.

5. Pain.—Pleuritic pain is treated by adhesive straps or by painting with iodine; if muscular, it should be treated by codeine to relieve coughing, by liniments, massage, fomentations or phenacetin.

6. Dyspnœa.—If due to stagnant secretion, moderate movement or change of posture aids in its evacuation; if asthmatic, iodide gr. iij after meals is indicated but it must be given with care lest it congest the lungs; if dyspnœa results from compression of the vagus by bronchial glands, iodide and green-soap inunctions should be given; if cardiac, digitalis or strychnia is indicated; if caused by pleuritis or pneumothorax, these affections should be treated.

7. Cardiac Weakness.—To relieve cardiac weakness, rest, an ice-bag over the precordia, cognac, champagne, camphor, strychnia, digitalis or strophanthus are in order.

8. Insomnia.—The patient should have fresh air night and day, a light, early evening meal, a glass of beer, porter or some whiskey on retiring. Bromides, codeine and morphine are beneficial.

9. Gastro-intestinal Symptoms.—Nervous anorexia is relieved by fresh air, forced feeding and guaiacol (*v. s.*). Dyspeptic symptoms necessitate a scant diet for a few days, feeding by rectum and poulticing of the epigastrium. In gastric atony, frequent meals of small volume, limitation of fluids and strychnia are indicated. For gas formation or pain the following are indicated; carbolic acid gr. j; resorcin gr. iij; fluidextract of gelsemium ♏ij; bismuth gr. x; extr. belladonnæ or spts. chloroformi ♏xx. Pyrosis may be corrected by sod. bicarb. and magnesia carb. āā gr. xv to xxx. Hyperacidity should be treated by milk diet, belladonna and Carlsbad water or other alkalies; anacidity is relieved by easily digested foods, as somatose or eucasin and hydrochloric acid. *Vomiting* is caused by the cough, overfeeding, overmedication or irritation of the pharynx (Lugol's solution locally, or gargles [1 to 10] of pot. bromide); small doses of carbolic acid (*v.* Typhoid) are most successful. *Constipation* (*q. v.*) is treated by the usual measures. Diarrhœa may be dyspeptic, catarrhal, toxæmic, ulcerative or amyloid; the following is indicated: Tannic acid gr. v; opium gr. j; lead acetate gr. ij; camphor gr. ij; tannigen gr. vij to xv, or bismuth ʒj (see Typhoid).

10. Anæmia.—This is benefited by fresh air, change to a high altitude, full diet, iron in small doses and arsenic.

11. Sexual Symptoms.—Intercourse must be forbidden in incipient as well as in advanced cases. Two or three years after recovery marriage may sometimes be allowed; it is generally less injurious to men than to women. Tuberculous parents may rear healthy children, but tuberculosis and scrofula are rather common in such families. In 66 per cent. of pregnant tuberculous women the prognosis is bad, and laryngeal tuberculosis is almost invariably fatal within a short time. In severe cases abortion may be con-

templated; opinions vary as to its indication, for some contend that the mother runs the greater risk. Kaminer found that in 70 per cent. of the cases abortion does not alter the clinical course.

12. Treatment of Other Complications.—The treatment of laryngeal, pleural, peritoneal, intestinal, glandular and meningeal complications (*v. s.*) is considered elsewhere.

LEPROSY.

Definition.—A chronic incurable contagion, caused by the Bacillus lepræ and characterized clinically by nodules, which belong to the infective granulomata (Lepra tuberosa), and are observed externally on the skin and nasal and oral mucosæ, and internally in many organs; or by a perineuritis leprosa, which causes sensory, motor and other symptoms (Lepra maculoanæsthetica). The nodular and anæsthetic forms usually co-exist in varying degree.

Distribution.—The home of leprosy is Egypt, where it existed 2,400 years B. C. (Brugsch) or even 4,620 B. C. (Engel-Bey). The leprosy of Leviticus probably includes other diseases. It prevailed in India 700, and in China 400 years B. C. Just before the Christian era, Pompey's soldiers brought leprosy to Italy, whence it spread over Europe and the Crusaders at the end of the thirteenth century further disseminated the disease. In Europe there were 20,000 asylums, but some cases were probably syphilis; the disease has steadily decreased since the sixteenth century. It is estimated that at present there are 3,000,000 cases in the world. In Europe cases are found in Sweden, Norway, Finland, Russia, Dalmatia, Bosnia, Montenegro, Germany, France, Italy, Spain, Portugal, Turkey, Greece and in some Mediterranean islands; Arning estimates that there are 3,000 cases in the west half of Europe. In Asia numerous cases are found in India (5 cases to each 10,000 of population), China, Malay Archipelago, Formosa, Corea, Persia, Turkestan, Syria and Arabia. In Africa it prevails in Egypt along the Nile, in Tripoli, Tunis, Algiers, Mozambique, Zanzibar, Madagascar and in the Canaries and Azores. In Japan, Australia, Oceanica and Sandwich Islands it is also found. In America foci exist in Canada (New Brunswick, Nova Scotia and British Columbia), in the United States (California, Louisiana, Minnesota, North Dakota, Florida; in 1902 there were 278 lepers in this country, of whom half were native born); in Mexico, West Indies, Honduras, Colombia (5 per cent. of the population), Brazil and Paraguay.

Bacteriology.—The *Bacillus lepræ* was found by Hansen (1871) and was fully described by Neisser (1879). It is a fine, narrow rod, measuring ½ to ¾ of a red cell, and closely resembles the tubercle bacillus (*q. v.*) morphologically and in its staining properties; both bacilli contain fat. Inoculation is the surest differential criterion between the two, for the lepra bacillus is not inoculable. In the internal viscera it may exceed the dimensions which it attains in

the skin and mucosæ five or six times (Cornil). It is motile and cannot be cultivated. It is found in the typical nodes and infiltrations of the skin and superior mucosæ, in the fresh maculæ and recent nerve disease, in the sweat, and sebaceous and hair-glands. It also exists in the nerves and Pacinian bodies of the skin; in the lymphatic glands, bloodvessels, blood during fever (metastatic distribution), and in practically all the viscera and secretions and excretions. It is not known why the bacillus is found so much more frequently and persistently in the nodular and mixed types than in the nervous forms; Stricker found it in the nasal secretion in 96 per cent. of the nodular, 96 per cent. of the mixed, and in 66 per cent. of the nervous, type. It has never been identified outside of the human body. It corresponds to but one of Koch's criteria; *i. e.,* it is always present in leprosy; it cannot be cultivated, and the value of successful inoculations on criminals under sentence of death has been justly criticized, for these cases developed in a leprous community. That *leprosy is contagious* can no longer be doubted. Less communicable than syphilis or tuberculosis, contagion occurs only by long and intimate contact with lepers. The nodular is far more dangerous than the anæsthetic type. Infection may be direct or indirect (by infected linen, utensils, etc.). It occurs chiefly through the skin and nose, possibly through the genitalia. Extension occurs more by the lymphatics than by the bloodvessels. A predisposition toward leprosy may possibly be inherited, but not the disease, and only 4 per cent. of children of leprous parents acquire it. More cases occur in colored than in white races, more in men than in women, and more in the poorer than in the upper and middle classes. It most frequently occurs between the ages of fifteen and thirty and rarely before the third year.

The *incubation* is long, at least three to five, and sometimes twenty, or even thirty-two, years.

Symptoms.—The chief clinical groups do not include the visceral involvement which is seen in every type; the main types are nearly always somewhat blended.

1. Nodular Type (*Lepra tuberosa seu tuberculosa*).—The prodromal toxæmic symptoms indicate an infection which has already been established, but has escaped recognition; they are depression, coldness, formication, sweats, pains, vertigo, digestive disturbance, temperature and sometimes nasal irritation. After months or years, *maculæ* appear on the face and extremities, which may itch or may be hyperæsthetic; they may disappear, leave pigmentation or metamorphose into *nodules,* which are present in 75 per cent. of all cases. The nodule or leproma, is a granuloma very similar to the tubercle or gumma; the nodes are highly vascular and are formed of a scanty fibrillar reticulum, in which lie emigrated leukocytes, small round epithelioid cells and the larger multinucleated lepra cells of Virchow, which contain close clumps of bacilli. The nodes by fusion form *infiltrations* which are gelatinous, glistening and yellow-red. The

nodes vary in size from that of a millet seed, to that of a hazelnut or walnut; they lie in the skin, are dark red and later become yellow-brown; they are glistening, vascular, slightly desquamating and at first soft but later become harder. *Sensation* is always *disturbed* in their vicinity. They are most conspicuous on the face and hands, but also appear on the extensor surface of the knee, elbow and fingers and very seldom on the soles of the feet, the palms of the hands, the scalp or penis. When they develop on the face, the forehead, nose, cheeks, chin, lips and ears are thickened, the nose becomes flatter, the eyebrows, lashes and the beard are shed. The eyes are involved in over 90 per cent. of the cases; the expression is leonine (*leontiasis*), or, as the ancients called it, faun-like (*satyriasis*). The nodes usually grow and extend slowly, and the lymphatic vessels and glands, of the neck and groin especially, become enlarged and tender. Sometimes the progression is interruptedly acute, with intermittent fever and an eruption resembling erysipelas. They may shrink, but more often *necrose,* soften and ulcerate. At the same time or later, nodules which are prone to early ulceration and infiltration, occlude the *nose,* roughen the voice (*raucedo leprosa*), distort the larynx and throat, impair mastication, swallowing or respiration, and even provoke suffocation. Cicatrization of the face causes added *deformity.*

2. Nervous Type (*Lepra nervorum, anæsthetica, maculo-anæsthetica, seu mutilans*).—The most common prodromes are fever, neuralgia, paræsthesia, hyperæsthesia and vasomotor cerebral congestion. *Maculæ* develop on the face, neck, trunk or limbs; they are pale or dark-red, flat or slightly raised, growing peripherally and paling centrally and are glistening or present a powdered aspect. Histologically they are identical with the nodules, but contain fewer bacilli, are flatter, contain no large cells and tend more toward fibrous change. Their distribution is asymmetrical, like that of the underlying nerve lesions; they are often oversensitive, sometimes pigmented (*lepra nigra*) or leukoderma-like (*lepra alba*). Early vesicles sometimes develop. If the skin is powdered with dry methyl violet and pilocarpine is then given, the sound tissue sweats and stains, while the maculæ remain uncolored because they do not sweat.

Sensory disturbances are due to *lesions* in the *nerve trunks,* which are the seat of a perineuritis and interstitial neuritis which slowly compress the nerve fibers. The connective tissue development and the presence of the bacilli differentiates this from the parenchymatous neuritides. Unlike the ordinary neuritis, the leprous neuritis affects the *sensory more than* the *motor filaments.* There is an irregular dissociated anæsthesia in which the perception of pain and temperature is usually more dulled than other forms of sensation, and this occurs mostly in the peripheral branches in the arms and legs. Sensation may be perverted or retarded and anæsthesia may come on suddenly, which is considered rather characteristic. Pain may be slight, or agonizing as in a case of ten years duration which was personally observed. The *nerve trunks are palpable* and tender in

many cases; the ulnar nerve is most markedly enlarged and in the case cited was thicker than a lead pencil, but the auricular nerve is more often felt (in 90 per cent., Baelz). Motor weakness and atrophy are less common and are rarely extreme; the eyelids, muscles of the mouth, and sometimes the masticatory and ocular muscles and also the hands and feet are paretic. The ulnar and peroneal nerves are sometimes paralyzed, causing the "claw-hand" and "claw-foot." Facial paralysis occurs in 9 per cent. of cases. The tendon reflexes are often increased, sometimes normal or decreased; there is often fibrillation and increased myotatic irritability. Trophic changes include œdema, glossy skin, "*mal perforant*" (26 per cent.), loss of hair or nails, gangrene, bone resorption, rheumatoid swellings, ankylosis and pigmentation. Great *trophic* changes mark the *lepra mutilans.*

3. Mixed Type.—This is a combination of the nodose and anæsthetic types. *In all developed forms the internal tissues are invaded:* The liver, spleen and lymph glands are enlarged; the lungs show peribronchial foci, cavities or induration; and the intestine is infiltrated. In both sexes the sexual functions are decreased and later lost. Infantilism results in cases developing before puberty. Cachexia develops, the patient becomes very sensitive to cold, the temperature, except for intermittent rises, becomes depressed and heart weakness develops. Abortive or long localized forms are described.

Course and Prognosis.—The course is slow and progressive, the outcome invariably fatal. The nodular type covers eight to twelve years, the anæsthetic form fifteen to twenty years, but either variety may endure six or seven decades. According to Hillis, 38 per cent. of cases die of leprosy (exhaustion, diarrhœa, cardiac weakness), 22 per cent. of chronic nephritis, 17 per cent. of lung disease, which is more often leprous than tuberculous and 10 per cent. of diarrhœa (leprous infiltration of the gut). The remaining 13 per cent. die of erysipelas, sepsis, amyloidosis and intercurrent diseases, for lepers exhibit a decidedly reduced resistance.

Diagnosis.—The diagnosis is based on the history of residence and exposure which is often suppressed by the patient, on the macules or leukoderma which are found in 90 per cent., on the anæsthesia, amyotrophy and mutilation, and on the finding of the lepra bacillus necessary for exactness, for which the nasal secretion, nodes and macules should be examined (*v.* Syringomyelia).

Treatment.—1. Prophylaxis.—All suspicious cases should be registered and all clear cases should be sequestrated.

2. Therapy.—Drugs are useless, though a few are helpful. (*a*) Gurjun oil 3ss to 3ij in water or lime water; (*b*) chaulmoogra oil, in milk, beginning with a few minims and increasing to 3j–iij; (*c*) salves of 5 to 10 per cent. pyrogallol, 10 per cent. chrysarobin, 10 per cent. ichthyol; (*d*) Calamette's antivenin, 20 to 30 c.c., which was suggested by cases which improved after snake bite; (*e*) arsenic,

salicylic acid and corrosive sublimate; these are the most extensively used remedies. Symptomatic and *x*-ray treatment of pain and ulcers is also indicated.

INFECTIONS OF UNCERTAIN ORIGIN.

FEBRICULA.

Definition.—A fever of short duration, due to a number of causes. If only of twenty-four hours duration it is called *ephemeral fever;* when it lasts a few days, it is called *febricula;* when attended by herpes, *febris herpetica;* or when accompanied by gastric disorder, *febris gastrica.*

Febricula is no morbid entity, and its varied *causes* are not exactly known. It embraces (*a*) benign or abortive forms of various acute infections, such as rheumatism, typhoid, malaria, scarlatina, tonsillitis and pneumonia; infections with the bacillus coli, proteus or pyocyaneus; and (*b*) febrile dyspepsias, especially those in children. Gastric fever is a term which was formerly applied to febrile gastritis, which is possibly produced by toxins in the food. Sewer-gas is popularly held as the cause of fugitive fever with gastro-intestinal disturbance, but Abbott and others have justly contested its etiological significance. Exposure to cold and heat is also a possible factor.

Symptoms.—The three common symptoms are (*a*) chill or chilliness; (*b*) sudden rise of temperature; (*c*) a critical resolution, more often prolonged than acute. The onset is abrupt, usually without prodromes. The fever rises quickly to 101°–103°, or higher in the young, with the usual signs of toxæmia, headache, coated tongue, anorexia and febrile urine. Herpes is frequent. Bronchitis, acute splenic tumor, albuminuria and in children, cerebral toxæmia, are sometimes present. The term ephemeral is inappropriate, as the average duration is three days, with a slow crisis.

Diagnosis and Treatment.—The diagnosis is solely by exclusion, and the treatment is expectant. Rest in bed is advisable as a precaution against some longer and insidious malady, such as typhoid; the bowels should be unloaded, the diet restricted and a drop or two of tincture of aconite and spts. ætheris nitrosi ℥ss should be given every three or four hours.

GLANDULAR FEVER.

Definition.—An acute infection observed in children and characterized by pharyngeal infection, a short high fever and a painful intumescence of the cervical lymph glands. It was described as *Drüsen fieber* by Pfeiffer, in 1889. Its *etiology* is unknown but it is evidently an infection, as suggested by the clinical signs and by its frequent occurrence in epidemics. It is usually seen in children

between five and eight years of age, and during the colder months. Park West observed an epidemic of 96 cases in Bellaire, Ohio. Hochsinger and Kretz maintain that the disease is a sequel of various nasopharyngeal infections rather than an independent infection, the primary trouble being overlooked and the secondary adenopathy being the first obvious sign.

Symptoms.—The symptoms begin without prodromes, with fever of 101° to 103°, pain in the head and neck, redness of the throat, some dysphagia, often pain in the limbs, nausea, vomiting and abdominal pain. On the second or third day the cervical lymph glands become swollen and tender bilaterally, but seldom cause redness of the skin. The glands back of the upper part of the sternomastoid are most involved. The inguinal and axillary glands are involved in 75 per cent. of cases and the mesenteric in about 40 per cent. The glands may reach the size of an egg. After ten to twenty days the swelling subsides, having lasted longer than the fever. In some instances a persistent dry cough suggests involvement of the bronchial glands, and umbilical pain, of mesenteric localization. The spleen is enlarged in 60 and the liver in 90 per cent. The fever, after a few days, falls by crisis. Complications are rare. Suppuration in the glands is exceptional. Acute nephritis, endocarditis, herpes, erythema and otitis media are occasionally observed.

Prognosis and Treatment.—The prognosis is good, though a relapse may occur. The absence of a rash excludes the adenopathies of varicella, scarlatina and measles, and the generalized glandular swelling excludes the local swelling due to simple pharyngitis. The treatment is expectant.

MILIARY FEVER.

Definition.—An acute infection, marked by fever, profuse sweating, a miliary eruption, nervous symptoms, palpitation and precordial anxiety. It first appeared in England after the Battle of Bosworth (1486) and broke out several times in the following century. It was known as the "English sweats" (*sudor Anglicus*), and its mortality reached even 50 to 80 per cent. In 1529 it caused 3,000 deaths in Dantzig. There have been about 200 epidemics between the epidemic in Picardy, in 1718, and that in Poitou, in 1887. It is highly contagious (Brouardel). Epidemics have also occurred in Italy, Switzerland, Germany and Austria, with a death-rate of 5 to 25 per cent. Fortunately they last only a week or month. In 1906 Chantemesse reported an epidemic of over 6,256 cases in northern France.

Symptoms and Treatment.—In mild outbreaks the chief symptoms are fever, profuse periodic sweating and miliary eruptions. The eruption may be erythematous or morbilliform (miliaria rubra), or there may be no redness of the skin (miliaria alba s. crystallina); the spleen may be enlarged. In severe cases there are marked cerebral toxæmia, rapid pulse, precordial distress, palpitation, extreme prostration and hemorrhages into the skin; death results in a few

hours or days. Its average duration is one to two weeks. Desquamation is frequent and convalescence rather tardy. The treatment is supportive and expectant.

WEIL'S DISEASE.—(ACUTE FEBRILE JAUNDICE.)

Definition.—An acute infection, characterized by fever, jaundice, herpes, enlarged spleen and liver, sore throat, muscle pain and albuminuria. It was especially described by Weil (1886), whose name it most often bears, though previously mentioned by Mathieu, Bouchard, Lancereaux and Coynes. Nicolaysen describes 63 epidemics of icterus in Norway and Sweden in the last fourteen years; 3 to 28 per cent. of the inhabitants in certain communities acquired the disease.

Etiology.—Its etiology is unknown, whence our difficulty in stating whether it is a morbid entity or whether it includes several diseases. It occurs in 90 per cent. in men between twenty-five and forty, especially in butchers and alcoholics; it also occurs in the warmer seasons and sometimes in epidemics. It is either microbic or toxæmic. Bordoni, Uffreduzzi and Jäger found the Proteus fluorescens, and Freund a diplococcus, but the *intravitam* findings are usually negative. Some cases have occurred in this country (Lanphear, Musser, Guiteras); the author has seen eight cases in Chicago.

Symptoms.—The symptoms begin suddenly with fever, chill, headache, insomnia and severe *pains* in the limbs and extremities. The *fever* reaches 103°; it afterwards shows remissions and falls either by crisis or lysis after five to eight days. The nervous symptoms may resemble typhoid. *Jaundice* develops on the third day, probably due to catarrhal radicular angiocholitis; bile appears in the urine and the stools are often acholic, though the author has twice seen deep jaundice without obstruction of the bile-duct. *Muscular pains* are very common and intense, especially in the calves, which are usually tender. Articular pains are not uncommon. *Herpes* is very common. The *rapid pulse* is very marked compared with the usual slow pulse of simple catarrhal icterus. In one case the author observed blood-stained bronchitic sputum. Digestive symptoms are common, as furred tongue, vomiting, epigastric pain, abdominal distention and diarrhœa. The *liver* is enlarged and tender in more than 50 per cent. of the cases, the *spleen* in over 66 per cent., and the *lymph-nodes* are frequently affected. The urine is albuminous in over 50 per cent.; acute nephritis is frequent; hæmaturia occurs occasionally and uræmia exceptionally; the urea and the amount of urine are decreased during, and increased after, the seizure (Chauffard's nitrogenous and polyuric crises).

Diagnosis.—Weil's disease must be differentiated from recurrent fever by its spirillum, and from sepsis and typhoid,—in regard to which it may be noted that icterus may produce a weak Widal reaction. Differentiation is more difficult from suppurative angiocholitis (*q. v.*) and acute yellow atrophy (*q. v.*).

Prognosis.—This is usually good and convalescence is generally established in two weeks; in one epidemic, 11 per cent. of the cases died. Recurrence in 40 per cent. may be anticipated if the splenic tumor has not subsided (Mathieu); renal or hepatic insufficiency, pregnancy, hæmolysis with epistaxis and hæmatemesis, render the prognosis less favorable.

Treatment.—The treatment is directed to (*a*) the intoxication, for which calomel and salines are indicated; cold water enemata (Krull and Chauffard) increase the renal flow, and, with it, the excretion of toxins; (*b*) the jaundice, for which salines should be given to lessen duodenal catarrh; (*c*) the albuminuria, for which a milk diet is indicated; (*d*) muscular pains, for which heat and massage with chloroform liniment, are most beneficial.

MILK SICKNESS.

Definition.—A disease acquired by man from the milk or meat of cattle suffering from the "trembles." Cows with the disease, after being driven too rapidly, suffer from anorexia, injection of the eyes, vertigo, trembling and convulsions ending in death. Young horses and sheep may also acquire the disease. Milk sickness occurs chiefly in newly settled countries, as in the Southwestern states, and it is still seen in North Carolina. Its origin and pathology are unknown. Jordan and Harris describe a Bacillus lactimorbi.

Symptoms.—In man the nervous symptoms are frequently conspicuous, as restlessness, convulsions or coma, which appear after a few days of malaise. The tongue is swollen and heavily coated, the breath fœtid, the stomach painful and irritable, the fever low, high or sometimes typhoidal. The issue is either fatal, in acute forms, within a few days, or recovery may result after weeks. Flesh, butter, cheese and milk, from infected animals may, even when boiled, cause death when given to dogs.

MOUNTAIN FEVER.

Wilson and Chowning think that the disease is caused by a red-corpuscle protozoön like that of Texas fever and malaria, though not pigmented; Stiles and Craig could not confirm their findings. Howard Ricketts found the disease was inoculable and he and King found the medium of infection was the wood-tick. Ricketts (1909) discovered a bacterium, of which details are lacking at the time of writing. Cases develop in the early spring, and are marked by chill, fever lasting one or two weeks, rapid pulse, epistaxis, albuminuria, rapid breathing, bronchitis, splenic tumor, rapid anæmia, moderate leukocytosis, slight icterus, muscular pains and widely diffused macular rash,—which develops on the third day, disappears on pressure at first, but afterwards persists on pressure. The liver and spleen are enlarged. Quinine may be beneficial in some cases. The death rate may be 70 per cent.

FOOT AND MOUTH DISEASE.

Definition.—Epidemic stomatitis, Aphthous fever, or Aphthæ epizooticæ is one of the zoönoses and its nature is unknown. Loeffler and Frosch have shown that the virus is ultramicroscopic, as it passes in full virulence through porcelain filters. The disease, first described by von Valentini, in 1695, and known, since the middle of the fifteenth century, chiefly affects cattle, sheep and pigs, but sometimes also goats, horses, dogs and even fowl. In *cattle* there is fever, swelling of the mouth, salivation and the formation of small, clear vesicles on the tongue, gums, lips, udders, hoofs and between the toes. These vesicles soon become opaque, and bursting, form small ulcers; emaciation is rapid, and the milk becomes yellowish and mucoid. Embolism or fatal apoplexy may result. Recovery is usual in one to two weeks. The disease spreads very rapidly. It is observed in Hungary, Germany, France, Switzerland, England and occasionally in this country.

Etiology and Symptoms.—*In man* infection occurs from diseased milk (as proven on himself by Hertwig), butter and cheese, but not by diseased meat. Open wounds may be directly infected. Cattle have been inoculated from man. The incubation is three to five days. There are fever, salivation, aphthous ulcers, angina, nausea, vomiting and vesicles on the skin—which have caused confusion with vaccinia. Hemorrhages occur in severe epidemics. Ebstein has suggested that Foot and Mouth Disease may be kindred with the ordinary aphthous stomatitis of nurslings or pregnant women. Children are more endangered than adults. The death-rate is 2.3 per cent. (Hulin) to 8 per cent. (Siegel). Recovery is usual in three weeks.

Treatment.—Prophylaxis concerns the boiling of suspected milk, the segregation of diseased animals or human subjects, and vaccination, as discovered by Loeffler. A dram of 5 per cent. solution of potas. chlorate, t. i. d., should be given, and the aphthæ should be touched with borax or silver.

SECTION II.

DISEASES OF THE CIRCULATION.

DISEASES OF THE HEART MUSCLE.

DILATATION OF THE HEART.

Definition.—Dilatation means distention and diminished tonicity of the chambers of the heart; it may be partial or complete. Hypertrophy is thickening of the walls of one or more chambers. The two conditions are quite separate in themselves, though they are frequently combined; some clinicians consider that dilatation is the incentive to increased nutrition *i.e.* hypertrophy. Lancisi (1706–1728) described hypertrophy and dilatation.

Etiology.—1. Mechanical Causes.—The normal heart may yield to increased blood-pressure or a weak heart may yield to normal pressure. Rarely, if ever, is dilatation an independent condition. (*a*) *Valvular disease, e. g.,* dilatation of the left ventricle in aortic insufficiency, or of the left auricle and right ventricle in mitral stenosis. Hypertrophy is usually inversely proportional to the dilatation. In combined valvular lesions, as aortic and mitral insufficiency, the heart is enormous—the ox heart, *cor bovinum, bucardia.* (*b*) *Causes raising arterial blood pressure,* as stenosis of the aorta, arteriosclerosis and nephritis produce hypertrophy and dilatation of the left ventricle. Workers in caissons may suffer from dilatation. (*c*) *Causes raising blood tension in the lesser circulation,* as spinal deformity, chronic disease of the lungs and pleura, which produce hypertrophy of the right ventricle; of these emphysema is the most important. Abdominal lesions are thought by French writers to reflexly raise the pulmonary blood pressure and induce right-sided hypertrophy and dilatation. Lichtheim's classic on the lesser circulation proved that the lung vascular area must be reduced to 25 per cent. in order to affect the right heart. The size of the heart varies much physiologically; rapid heart action allows less complete physiological dilatation than does slow action. When there is pulmonary obstruction with its consequent heart changes, the patient stands pneumonia, bronchitis and other infections very poorly. When severe exercise is indulged in, the systole is incomplete and right-heart dilatation—even tricuspid regurgitation and marked venous stasis—may follow. Physical training increases the power and reserve strength of the right heart; *i. e.,* it increases the

"wind" and endurance. An equal effort in an individual out of training, or with poor nutrition, "breaks his wind" and produces transient or permanent cardiac exhaustion. Air embolism also produces dilatation.

2. Nutritive Disturbances.—Dilatation may be induced (*a*) by acute infections, leading to cardiac degeneration or acute myocarditis; (*b*) by anæmia, hemorrhages or loss of fluids; (*c*) by chronic myocarditis, fatty heart or coronary atheroma, possibly following sudden heart strain as in athletics; by endo- or pericarditis invading the heart muscle by contiguity, pericardial adhesions, etc.; (*d*) by poisons, alcohol, tobacco or chloroform anæsthesia; (*e*) it very frequently follows hypertrophy or is associated with it.

Pathology.—Dilatation occurs (*a*) with thinning of the walls, *simple dilatation,* or (*b*) with eccentric hypertrophy (*q. v.*). It is most common in the thin-walled auricles and less resistant right ventricle. The muscle is lax and the heart, when held inverted by the basal vessels, may in extreme instances fall flabbily like a cap over the hand. In extreme dilatation the endo- and pericardium almost touch.

The papillary muscles and trabeculæ are flattened (pressure atrophy) and frequently thickened (pressure sclerosis). Microscopically the heart muscle may be normal, or may show fatty or cloudy degeneration or myocarditis. The precise role of degeneration in the ganglia of the heart has not been determined. There is much blood in the heart's chambers, in the cavæ and other veins. Long-standing dilatation is not to be confused with dilatation occurring during the death agony from acute lung disease, suffocation, etc., in which the right heart is chiefly affected and in which on removal of the post-mortem clots the chambers seem more nearly of normal size.

Symptoms.—The symptoms are those of cardiac insufficiency (*v.* Uncompensated Valvular Disease), as dyspnœa—which is especially great in pericardial adhesions—cardiac asthma, cough, syncopal attacks and precordial anxiety. Cardiac failure begins when the systole is unable to expel all the blood from the dilated chambers.

Physical Signs.—It is not always easy to separate the signs of hypertrophy and those of dilatation.

1. Dilatation of the Left Ventricle.—(*a*) *On inspection,* the *apex beat* is dislocated downward, to the left and is diffuse, undulating and weak, if indeed it is not absent. (*b*) *On palpation,* the apex may not be felt even though visible (Walshe). In other cases the apex and heart shock are snappy and may feel strong to the inexperienced hand; the sharp snappy action contrasts sharply with the weak, rapid and often irregular radial pulse. (A palpable impulse may be due to active diastole of the auricle, to passive distention of the ventricle by blood from the auricle or to closure of the auriculoventricular valves during the presystole.) Albertini (1761) distinguished hypertrophy and dilatation by palpation. (*c*) *On auscultation,* the *heart tones* are weak, though sometimes snappy. The

first apex tone is more valvular. The second aortic sound is weak because of poor arterial tension. In insufficient hearts, the heart tones sound alike, a condition termed embryocardia from its resemblance to the fœtal heart tones. *An apical systolic murmur* is frequent, especially in arteriosclerotic dilatation. It is sometimes due to relative insufficiency of the mitral valve whose normal circumference of three and one-half inches may be stretched to five. Von Jürgensen holds that it is often due to irregular muscular contraction. Rest and the administration of digitalis frequently cause it to disappear. *Gallop-rhythm,* in which three tones are heard, like the rhythm of a galloping horse, consists of either doubling of the first (Potain) or second tone (Fräntzel), or of the addition of a presystolic tone caused by the *auricular contraction* (Rosenbach). It is heard in the failing hypertrophied hearts of nephritics more than in all other conditions combined. It is also found in valvular lesions, atheroma, emphysema and in acute diseases such as pneumonia, typhoid and diphtheria. The auricle cannot long compensate. Whatever its cause, it is a sign of great danger although the heart's tonicity may temporarily improve with rest and heart tonics. (*d*) *On percussion,* the heart extends to the left and downward, giving a more oval area of dulness.

2. Dilatation of the Right Ventricle.—This is more common. (*a*) On *inspection,* a diffuse beat is seen to the right of the sternum or in the epigastrium; and (*b*) it may be *palpated.* (*c*) *Auscultation* shows weakness of the heart tones, and (*d*) *percussion* outlines dulness to the right of the sternum. Extreme dilatation increases the tricuspid orifice from four and one-half to five or six and one-half inches, and the physical findings of relative tricuspid insufficiency obtain.

3. Dilatation of the Auricles.—Because of the deep location of the left auricle physical signs of dilatation are seldom obtained. A presystolic wave may be seen in the second left interspace. The changes usually ascribed to the left auricle are due to enlargement of the conus of the right ventricle. The distended right auricle is recognized by undulation to the right of the sternum above the right ventricle, by dulness and sometimes by a presystolic wave.

Dilatation of all the chambers, right and left, combines the physical findings of each type, giving the heart a triangular or a trapezoid conformation. The cardiac symptoms and physical findings of dilatation may develop gradually, or abruptly after violent exercise, especially when the heart is congenitally weak or its nutrition is altered.

Diagnosis.—The diagnosis depends on the etiological factors, the *weak tones,* the *increased dulness* and cardiac and somatic evidences of *cardiac failure.* The greater the dilatation, the less marked is the difference between the systole and diastole. The *absolute heart dulness* in middle life is bounded by the left border of the sternum, the fourth or fifth rib and the left parasternal line (or a little more to the left). The *relative dulness* reaches from the third or fourth

rib above to the apex beat on the left and to a line drawn obliquely from the third left costal cartilage across the sternum to the fifth right costal articulation. Percussion is difficult in distorted, fatty or œdematous chests. Differentiation concerns the same group of conditions discussed under the diagnosis of *effusive pericarditis* (*q. v.*). Rest and digitalis greatly benefit dilatation and hydropericardium, sometimes within a very short time, while pericardial effusion is unaffected by this treatment. *Consolidation of the adjacent lung* margin can usually be recognized by the altered breathing and rales. *Mediastinal tumor* and *aneurysm* are suggested by pressure symptoms. If the margins of the *lung are adherent* from pleural adhesions, especial care in percussion is imperative, and dilatation is diagnosticated by the relative, more than by the absolute, heart dulness. Palpatory percussion, very lightly made, is most valuable and the *feel* of the increased dulness has greater diagnostic value than the sound elicited. *Emphysema* may greatly obscure the outlines of the heart, but the heart's outlines are usually disclosed by careful palpatory percussion for the relative dulness. *Encapsulated pleurisy* is differentiated by careful puncture, if other signs fail. The fluoroscope is frequently of aid. In short chests, the apex is seen normally at or even beyond the nipple. This occurs in 12 per cent. of women. Though *spinal curvature* frequently overtaxes the right heart and thus induces dilatation, the diagnosis must be made with care, for the topographical relations are much disturbed by visceral luxation, and the irregularity of the sternum and ribs makes percussion uncertain. In *fat individuals,* dulness may result from adipose deposit in the mediastinum, but differentiation is often easy by means of the normal, though perhaps distant, heart tones and by the character of the pulse. Dilatation must not be mistaken for dislocation due to gaseous, tumor or fluid distention of the abdomen.

Prognosis.—The prognosis largely depends on the etiology.

Treatment.—(*a*) The cause must be treated. (*b*) The *dietetic, special* and *general indications* of *valvular disease* (*q. v.*) must be carried out. (*c*) *Oertel's treatment* consists of (i) *limitation* of the amount of *water ingested,* and stimulation of its excretion by sweats and deep inspirations; beer must be interdicted. Many nervous subjects tolerate limitation of water very poorly. Water should be given in small quantities at frequent intervals. (ii) *Reduction of the body-weight* (see OBESITY). (iii) *Stimulation of the circulation by exercise.* Mountain climbing is said to stimulate the heart, develop the respiratory muscles, increase the lung capacity and widen the vessels. The objections to active exercise are that rest in bed gives better results, and that exercise causes harm when the heart muscle or coronaries are diseased, or when endocarditis, pericardial adhesions or Bright's disease exists. (*d*) The so-called *Schott method* was originated by Beneke and Sätherburg, and was elaborated by Nauheim physicians. It consists of (i) *baths* in springs containing sodium and calcium chloride, iron carbonate and carbon dioxide. The tem-

perature should be a few degrees below body-heat, and little carbon dioxide should be used in the baths at first. They should be given once daily lasting from five to twenty minutes, for two or three days, and then intermitted. They may be given artificially with good results, using 40 gallons of water, with 3 to 10 pounds of salt, 10 ounces of pure calcium chloride and carbon dioxide generated in a vessel from sodium bicarbonate and hydrochloric acid. The temperature should be reduced from body-temperature to 90°, and the amount of carbon dioxide increased. The bloodvessels are first contracted and later dilated. The pulse becomes slower and fuller, dilatation decreases, stasis is lessened, diuresis is increased and the metabolism of the muscles and heart is improved. (ii) *Resisted movements* should be carefully and gradually employed. Though active exercise constricts the vessels, gentle movements by the patient, resisted by the operator, dilate the vessels.

Yeo summarizes the resistance movements as follows:

Each exercise is made against slight resistance applied by the physician or a trained assistant.

1. The arms are extended in front of the body at the level of the shoulder, with the palms of the hands touching. The two arms are then moved slowly outward till they are in a line with each other; they are then brought back to their original position.

2. The arm and hand hanging down with the palm turned forward, the forearm is flexed upon the arm (which is kept still) until the fingers touch the shoulder. The forearm is then extended to its original position. This is first done with one arm and then with the other.

3. The arms, hanging down as in No. 2, are raised outward until the thumbs meet over the head; they are then returned to their original position.

4. With arms dependent, the fingers, at the first phalangeal joints, are pressed together, and the arms are then raised until the hands are above the head, after which they are brought back to their original position.

5. The arms, hanging in the position of "attention," are advanced forward parallel to each other until they are elevated to a vertical position; they are then brought back to where they were before.

6. Same as No. 1, but with fists clenched.

7. Same as No. 2, but with fists firmly clenched.

8. The arms, starting from the position of "attention," describe a circle by moving forward and upward until they are raised vertically; then each palm is turned outward, and the arms descend backward to their former position.

9. The body is bent forward, and then brought back to the erect position, the knees not being moved.

10. The body is rotated, without any movement of the feet, first to the right and then to the left, and then back to its original position.

11. The body is flexed laterally, as far as possible, first to the one side and then to the other, and afterward restored to its original erect position.

12. The patient, standing with the feet side by side and supporting himself by leaning with one hand upon any object, flexes the opposite thigh as far as it is possible, and afterward extends it until the feet are again side by side; then, leaning on the other hand, he carries out a similar movement with the other thigh.

13. The patient, supporting himself by one hand, as in 12, and the knee being kept straight, each leg in turn is raised as high as possible in front of the body, and then in the same way behind.

14. Supporting himself by placing both hands in front on the back of a chair, the patient first flexes one leg and then the other upon the thigh as far as he can.

15. Each leg in turn is abducted as far as possible, the knees being kept straight, the patient resting on one or other hand the while.

16. The arms, held horizontally outward, are rotated forward and backward at the shoulder joint.

17 and 18. Flexion and extension, first of the wrists and second of the ankles. In resisting these movements the operator places the palm of his hand on that side of the patient's limb or body toward which the movement is to be made. In the movements of the wrist the operator closes his thumb and forefinger round that joint.

(iii) *Passive movements and massage.* Contraindications to Schott's method are aneurysm, angina pectoris, acute cardiac insufficiency, tendency to hemorrhage and embolism and possibly nervousness.

HYPERTROPHY OF THE HEART.

Etiology.—The causes of *hypertrophy* of the *left ventricle* are usually mechanical resulting from (*a*) *valvular lesions* (aortic stenosis and insufficiency, mitral insufficiency) wherein dilatation precedes, and incites, hypertrophy; (*b*) *renal disease;* usually from a chronic interstitial nephritis but occasionally from hypoplasia of one kidney, hydronephrosis, etc.; it is caused by mechanical obstruction to the flow of blood or by retention within the blood of waste substances which excite arterial spasm; (*c*) *arterio-sclerosis* (*q. v.*), developed by Traube; (*d*) *congenital narrowness* (*hypoplasia*) *of the arterial system,* or of the *aorta at the isthmus;* (*e*) *dilatation of the aorta,* diffuse or aneurysmatic, or occurring rarely from general arterial dilatation (Nothnagel and Fräntzel); (*f*) *pericardial adhesions q. v.;* (*g*) *myocarditis* (*q. v.*); (*h*) *pregnancy;* it is claimed that the placental circulation or increased intraäbdominal pressure increases the heart's work. The high diaphragm apposes the heart to the chest wall and tips the apex outward so as to simulate hypertrophy; (*i*) *over-exertion;* the "irritable heart of soldiers" (Da Costa, Myers, Fräntzel) is caused by forced marches, cold and mental excitement; the so-called *idiopathic* hypertrophy occurs in hard workers, and especially in Munich from excessive use of beer and from the resulting plethora (Bauer and Bollinger); hypertrophy results from continued hard work while dilatation follows sudden cardiac strain; (*j*) *nervous causes;* these are exophthalmic goitre and tobacco, especially strong Havana cigars, which cause rapid irregular heart action; goitre and tobacco are also probably toxic causes; (*k*) *reflex* hypertrophy is said to result from abdominal growths, brachial neuritis, etc. (Potain). Putjatin, Ott and others have found degenerative and sclerotic changes in the cardiac ganglia.

Hypertrophy of the right ventricle depends on (*a*) *hypertrophy consecutive* to *left heart disease;* the entire heart usually hypertrophies from the intimate connection between the muscular fibres of both sides; (*b*) *right-heart valvular lesions* and *congenital heart disease;* (*c*) *increased pulmonary pressure* from disease of the pulmonary artery or pressure upon it; (*d*) *emphysema, chronic bronchitis, spinal deformity,* pulmonary induration, bronchiectasis and extensive *pleural adhesions.*

Hypertrophy of the auricles always coëxists with dilatation (as in stenosis of the auriculoventricular orifices).

Pathology.—The muscle fibers increase even tenfold in size (*hypertrophy*) and increase in number (*hyperplasia*). Hypertrophy may exist alone—*simple hypertrophy.* It more frequently occurs with dilatation—*eccentric hypertrophy. Concentric hypertrophy, i. e.,* with decrease in the size of the ventricle, is rarer and some regard it as merely a postmortem condition; the ventricle may be firmly contracted in death from violence, cholera or hemorrhage, but it can be distended by the finger. The size of the normal heart is that of the fist (Laennec), and the normal weight is about 9½ ounces (300 gm.). The heart is sometimes increased four times in weight, even to 59 ounces (1,980 gm., Stokes)—the *cor bovinum.* Its consistence is increased and its color is normal or darker. In total hypertrophy the heart is round; in partial, the right or left ventricle is larger. The left ventricle is normally nine to ten millimeters in thickness, but this may be more than doubled in hypertrophy; the right ventricle measures three or four millimeters; the left auricle measures three, and the right two, millimeters. In the fœtus the ventricles are equally thick. Up to the eighth year, the left ventricle is relatively thicker than in the adult, because of narrowing of the aorta at its isthmus.

Symptoms.—Hypertrophy of the left heart may exist without any subjective sensations, or there may be palpitation, cough, cardiac oppression or pain or cerebral congestion,—tinnitus, flashes before the eyes, headache or vertigo. There is frequently a tendency to hemorrhage—epistaxis, cerebral hemorrhage, excessive menstruation. Polyuria results from high arterial tension. The causal nephritis, valvular disease, arteriosclerosis or emphysema presents its own symptoms. Righ-heart hypertrophy is attended by brown induration of the lungs, hæmoptysis, cyanosis or bronchial catarrh.

Physical Signs.—Much confusion in the clinical description prevails because the signs of hypertrophy are very often confounded with those of dilatation; both conditions frequently coëxist.

Hypertrophy of the Left Ventricle.—1. *Inspection.*—This discloses (*a*) a *strong,* sometimes heaving, diffuse *apex;* essential dislocation to the left or downward indicates coincident dilatation, because thickening of the heart muscle alone gives no appreciable increase in the size of the heart. Rotatory retraction of the apex beat is sometimes seen. (*b*) *Precordial prominence;* and (*c*) *forcible pulsation* of the *vessels.*

2. *Palpation.*—Palpation confirms (*a*) the strong *apex beat,* detects (*b*) the *palpable second aortic tone* due to high tension (or atheroma), and sometimes (*c*) a *systolic apical thrill* (transmission of the strong first tone). (*d*) The *pulse* is regular, full and hard in simple hypertrophy, but in the eccentric form it is softer and more rapid. It is sometimes weak or almost absent in the arms; this is due to loss of vessel tonus. (*e*) *Thrills over the peripheral vessels* from high tension vibration may be felt.

3. *Percussion.*—It is usually stated that the left heart is dislocated

downward to the left but enlargement means dilatation or an atheromatous relaxed aorta which allows sinking of the heart.

4. *Auscultation.*—This brings out (*a*) the *loud first tone,* over the apex (but not in aortic leakage); it discloses (*b*) the *accentuated* and frequently metallic *second aortic tone;* (*c*) *systolic arterial bruits* from tension and vibration of the walls; (*d*) the *gallop-rhythm* in cases of failing hypertrophy, especially in nephritics; (*e*) a *peculiar* clink, which is sometimes heard to the right of the apex; (*f*) an *apical systolic murmur* is sometimes heard and is frequently of the cardiopulmonary type. Seitz, Gendrin and others have recorded murmurs resembling the pericardial rub, due to the violent beating of the heart or to muscular contraction (Jürgensen).

Hypertrophy of the Right Ventricle.—(1) *Inspection* shows a *strong, systolic impulse under the lower part of the sternum* which is (2) *palpable,* as is the *strong second pulmonic tone.* (3) *Auscultation* brings out the accentuated second pulmonic; and (4) *percussion* is negative, unless there is coincident dilatation.

Diagnosis.—In *left-sided hypertrophy,* the cardinal signs are (*a*) the tense pulse, (*b*) the strong, hard apex beat and (*c*) the *accentuated* second aortic tone; in *right-sided hypertrophy* they are (1) the strong second pulmonic tone and (2) the substernal pulsation. Percussion is useless, for it discloses dilatation only.

Hypertrophy of the left heart must not be confused with nervous overaction of the heart, in which the apex is not heaving. Retraction of the left lung merely exposes more of the left ventricle, but the heart in this case enlarges upward and to the left. In narrow chests with poor lungs, the heart's dulness is wide, but the cardinal symptoms of hypertrophy are lacking. The same caution in the diagnosis of hypertrophy at puberty is necessary because at this period the heart is nervously unstable, the chest is thin and the heart is normally wider than in the adult. Perplexity may be caused when abundant casts and blood are found in the urine; these are usually indicative of *nephritis,* but, if there are other evidences of stasis, such as congested liver, they are compatible, in the minds of some writers, with stasis alone. Nephritis may cause hypertrophy; nephritis and hypertrophy may be due to a common cause; or a failing heart may produce renal stasis (see Nephritis, Differential Table).

Prognosis.—The immediate outlook is good because hypertrophy is usually a compensatory process; the ultimate outlook is bad, because cardiac insufficiency is the ultimate issue. Symptoms usually appear first either gradually or suddenly in the stage of broken compensation from intercurrent fevers, malnutrition or mental and physical strain. The best outlook is in hypertrophy caused by tobacco or athletics, for this may regress. Gallop-rhythm is ominous, as is the occurrence of the first and second apical tones close together. Hypertrophy leads to fatty and other change in the heart, to sclerosis of the vessels, and in the pulmonary circuit to pulmonary atheroma, acute congestion and œdema.

Treatment.—The therapy is eminently causal and dietetic; alcohol, tea and coffee, tobacco and cold baths are to be avoided and constipation must be prevented. The symptomatic treatment and the use of cardiants in the uncompensated stage are identical with the indications in valvular disease.

ATROPHY OF THE HEART.

Atrophy of the heart presents more pathological than clinical interest.

Etiology.—The heart is at times congenitally weak and small. It may then be associated with small vessels, chlorosis (Virchow) or with hypoplasia of the genitalia (Rokitansky). Pressure atrophy, coronary sclerosis, pericarditic adhesions, the senile involution and cachectic diseases are the most frequent causes. *Anatomically,* the heart is reduced in size and weight (even to 1½ ounces), the subpericardial fat disappears, the pericardium wrinkles, the coronary arteries are tortuous and the heart muscle is pigmented. The valves, especially the aortic, may waste. The atrophy may be partial, as that of the left ventricle in some cases of mitral stenosis.

The *symptoms* are indeterminate, and the *diagnosis* impossible.

FATTY HEART.

Etiology and Pathology.—Fatty heart is no distinct pathological nor clinical entity. Two forms are recognized, (1) fatty infiltration and (2) fatty degeneration.

1. Fatty Infiltration (*Lipoma cordis capsulare, Virchow*).—This form consists of invasion of the heart muscle by the subpericardial fat, which is normally found about the heart, in furrows, at the tips of the ventricles and around the basal vessels. The fat extends in parallel lines between the muscle fibers, which it may wholly replace. The fatty infiltration was considered primary by von Leyden; Strümpell believes muscle atrophy is primary and fatty infiltration secondary; Rosenbach holds that fatty infiltration and muscular atrophy are secondary to a common nutritive disturbance. The fat, which develops most in the outer layers of the myocardium, may reach to the endocardium. Fatty infiltration is most common in obese males over forty years of age. It may occur after the menopause or in sterile women. Plethora, high living, excessive use of alcohol and heredity are factors. The heart is reduced 25 per cent. in weight compared with the body-weight (Hirsch).

2. Fatty Degeneration.—This form is caused by deficient oxygenation which is either (*a*) *general,* resulting from acute infections, old age, cachexia, marasmus, anæmia, poisons as phosphorus, chloroform or mercury and from loss of fluids; or (*b*) *local or cardiac,* from pericarditis; coronary disease, which more frequently produces myofibrosis; and from cardiac failure following hypertrophy. Most

degeneration occurs in the left heart. The fatty heart is large and flabby; it is yellow-brown—the "faded leaf" color of Laennec; the splashes and streaks of fatty degeneration contrast with the normal areas, giving the variegated "tiger" or "tabby-cat" appearance; oil globules are found in rows in the muscle fibers and may replace them in part or in whole. Frequently the muscle fibers appear dry and granular ("albuminoid" degeneration), probably the first step in fatty degeneration. The striations of the muscle disappear later, its consistence is decreased and its substance is friable.

The extent of the process is in direct ratio to the intensity of nutritive alteration; parenchymatous and fatty degeneration are observed in those segments of the heart where the greatest work and the most nutrition are demanded, as in the papillary muscles of the mitral valves and where pressure is greatest, *viz.,* the interventricular septum and left conus arteriosus. In embryonal life, these conditions obtain in the right heart. The early investigations of von Leyden and those of more recent date by Göbel have determined that these changes are first focal and later diffuse.

Symptoms and Signs.—No symptoms are characteristic. Extensive fatty changes may occur without clinical symptoms, as in pernicious anæmia. Leube details a case without symptoms in which the apex was completely fatty. In some obese subjects, cardiac embarrassment develops with dyspnœa, palpitation, a somewhat characteristic wheezing, etc. The autopsy may reveal fatty heart, but far more frequently myocarditis, idiopathic hypertrophy or coronary disease is found; *i. e.,* there is no uniformity in the pathological findings, nor parallelism between them and the clinical signs. Though percussion of obese individuals is difficult, the heart is found dilated. In fatty heart, the tones are weak and distant though pure, the apex beat is weak, diffuse or absent, and sometimes an apical systolic murmur is heard, due to irregular systolic vibration. Gallop-rhythm is heard when the heart is fatigued. The pulse may be rapid from brain anæmia; it may be somewhat dicrotic, small and arrhythmic. Low temperature, sweating and bronchitis are common.

Much stress was formerly laid on the *Adams-Stokes' syndrome,* a triad of symptoms including (*a*) a *slow pulse,* even 14 to the minute, due to ischæmic stimulation of the vasomotor centre or to heart changes, and followed by (*b*) *pseudo-apoplectic seizures,* unconsciousness, temporary paralysis or convulsions (pseudo-epilepsy), which are clearly dependent on cerebral anæmia, for one of Stokes' cases warded off these attacks by assuming the genupectoral position; and (*c*) *Cheyne-Stokes' respiration,* in which respiration becomes slower, and is followed by a period of apnœa lasting a half minute or more, with respiration gradually returning to the normal. Narcotics increase or induce the condition. The pupils narrow during the apnœic period and muscular twitching is common. This triad is observed in uræmia, cholæmia and diseases of the vagus and medulla, as well as in heart disease. It seems likely that in Stokes' case the brain

was also diseased. The syndrome is based on the bradycardia which appears to be most frequent in disease of the bundle of His (see Bradycardia, Cardiac Neuroses).

Diagnosis.—The diagnosis is uncertain. Fatty heart may cause no symptoms, or the symptoms observed in the obese may be due to other lesions. Apparently increased dulness is often due to mediastinal obesity. The main point is to exclude valvular lesion and confine the diagnosis to a myocardial affection (*v.* Myocarditis).

Prognosis.—The prognosis depends upon the cause. It is grave in diabetic or arteriosclerotic subjects, or when coronary disease, angina, lung œdema or an irregular or very slow pulse intervenes. Sudden death is not uncommon from acute dilatation, pulmonary embolism, brain anæmia or hemorrhage, "heart block" (described under bradycardia), or rarely from heart rupture.

Treatment.—The treatment is that of obesity in fatty infiltration, but distinction must be made between (*a*) *obesity with anæmia,* in which the subject is obese and pale, with cold extremities, much sweating, scanty urine, weak and easily fatigued muscles and dyspnœa; and (*b*) *obesity with plethora,* in which the color is red, the urine increased and uratic, the heart stronger, the muscles fairly developed and exercise possible with little dyspnœa. Schott's treatment is beneficial in the first type, and Oertel's method in the latter (see Dilatation of Heart). Saline cathartics, total abstinence from alcohol and the use of potassium iodide for general or coronary sclerosis are indicated. The brain anæmia and the sudden syncope are met by the æthereal alcoholics. Cardiac insufficiency is treated as in uncompensated valvular lesions and dilatation. Morphine should be used with great circumspection.

ACUTE MYOCARDITIS.

Etiology and Pathology.—Acute myocarditis is of the greatest clinical and prognostic importance.

Acute diffuse parenchymatous myocarditis corresponds to granular and fatty degeneration (*v. i.*); to the "mushroom" heart of Stokes (described under Typhus) which, when held inverted by the base, would fall like a cap over the hand; and also corresponds to the "softened heart" of Laennec and Louis.

Acute diffuse interstitial myocarditis has been most studied of late years, while formerly the degenerations attracted most attention. Round cells are found in the interstitial tissue, with or without muscular alteration, such as vacuolization, nuclear multiplication, pigmentary deposit, blood extravasations and waxy changes. It was first discovered by Leyden in scarlatina; it is found in a very large percentage of cases of diphtheria and typhoid by Rhomberg; it also occurs in acute endocarditis and rheumatism (10 to 20 per cent.), less frequently in other infections. Though the typhoid bacillus, the gonococcus and Bacillus pyocyaneus have been found in the myocardium, they are chiefly important as causing toxæmia.

H. Martin found inflammatory thickening of the intima in the coronary radicles and Rhomberg a perineuritis in one-half of typhoid and diphtheria autopsies. Virchow, Hayem and Renault hold that inflammation is primary and degeneration secondary; Weber, Blind and others regard the process as degenerative.

Symptoms and Signs.—During the progress of an acute infection the heart's strength decreases; its tones, especially the first mitral, become fainter. There is often a marked presystolic impulse; the apex is less distinct and is dislocated somewhat to the left by dilatation; and the pulse grows weaker and more irregular. Vasomotor relaxation is a very important element in the heart failure. Gallop-rhythm and embryocardia develop, and there is usually a relative, muscular, mitral leakage. Cardiac pain may be severe. These changes may develop during the infection, as is the case in typhoid, scarlatina and rheumatism; or they may occur two to ten weeks after the infection, as in 15 per cent. of diphtheria cases. Sudden death without symptoms may occur during convalescence. Rheumatic myocarditis often leads to chronic fibrous myocarditis.

Diagnosis.—The etiology is important; acute myocarditis is distinguished with great difficulty from acute endocarditis (*q. v.*).

Treatment.—*Prophylaxis.*—Hydrotherapy is indicated in typhoid and antitoxin in diphtheria; the patient should have absolute rest well into convalescence, because sudden heart insufficiency or unexpected death may follow effort, difficult defecation or other exertions. Sexual intercourse should be forbidden. The heart must be carefully watched in every convalescence. See therapy of TYPHOID, DIPHTHERIA and PNEUMONIA. *Cardiants* are indicated as in valvular incompetency.

I. Acute Circumscript Myocarditis.—Acute circumscript myocarditis (the disease of gladiators, Galen) is usually a form of sepsis, and its embolic heart foci are usually multiple. It is usually caused by the pyogenic organisms. The issues are (*a*) resorption with fibrosis, (*b*) caseation or calcification and (*c*) rupture into the pericardium or into the heart cavity, thus producing "heart ulcers" and pulmonary or arterial embolism, heart aneurysm or abnormal communications between the chambers. Sudden valvular insufficiency is sometimes suggestive, though it is usually impossible to make a diagnosis. The treatment is that of the fundamental septicopyæmia and the resulting cardiac insufficiency.

II. Fragmentation of the Heart Muscle.—Fragmentation of the heart muscle (Zenker, Virchow, Rindfleisch, Coats, Renault and Landouzy) occurs in two forms (*a*) segmentation or separation from each other of the heart fibers, and (*b*) fragmentation or rupture across the normal or degenerated muscle fibers. It has also been called segmentary myocarditis, and though it most frequently occurs in death from violence, it may have some clinical significance in sudden or gradual heart weakness (see MYOLYSIS under DIPHTHERIA).

CHRONIC MYOCARDITIS (CHRONIC FIBROUS OR INTERSTITIAL MYOCARDITIS, MYOFIBROSIS CORDIS).

Etiology and Pathology.—Chronic myocarditis is rarely a disease *sui generis,* and the term is somewhat ill-chosen. It is a sequel of some previous retrogressive lesion—a secondary process. It compensates for destruction of heart tissue, and is in itself a measure of that loss. It is more often a *fibroid degeneration* than an inflammation. The longitudinal incision of the heart muscle discloses the scar-like tissue which is often unseen in the ordinary transverse sections (Köster and Rühle). This tissue is seen most frequently in the lower two-thirds of the anterior wall of the left ventricle and the upper two-thirds of its posterior wall near the auricle, as glistening sunken areas under the pericardium or endocardium. It also occurs in the papillary muscles or interventricular septum. The scars appear as streaks, flecks or stellate plaques, sometimes measuring three or four centimeters, or more. The foci may be macro- or microscopic, circumscribed or general. *Gross changes in the heart* include the following:

1. Hypertrophy.— This is vicarious and occurs when the fibrosis is not great. The relation is two-fold; (*a*) hypertrophy often compensates for moderate muscular destruction and maintains the heart; or (*b*) fibrosis develops in tired hypertrophied hearts. Hypertrophy is inversely proportional to the number of scars.

2. Dilatation.—This follows extensive fibrosis and decreased power to resist pressure. Fibrosis of the tissue can neutralize dilatation to some extent, and is therefore a conservative agent.

3. Atrophy of the Myocardium.

4. Valvular Disease.—Valvular disease and myocarditis are indirectly associated. A relative mitral insufficiency may follow myocarditis in the mitral papillary muscles (Hamernjk). Dittrich first described the genuine heart-stenosis, a cicatricial contraction of the left conus arteriosus in the adult, or of the right conus in the fœtus, following myocarditis at those sites. Relative aortic insufficiency may follow enormous dilatation and myocarditis of the left ventricle (personal observation).

5. Partial Heart Aneurysm and Heart Rupture.—Partial *heart aneurysm,* a localized bulging of the heart wall, is seen largely near the apex (left ventricle, 68 per cent. of cases, Pelvet), where acute myomalacia cordis or chronic fibrosis occurs most frequently; it is seen more rarely at the base or in the interventricular septum which may bulge into the right heart; and it occurs most infrequently in the right ventricle or auricle. The heart aneurysm is usually small, but may equal the size of the heart; it is usually single (though four were found by Thurnam), and may communicate with the heart cavity by a neck-like constriction; its walls consist of connective tissue which is rarely ossified, as in Corvisart's case; it often contains thrombi and is adherent to the pericardium. In unfavor-

able cases it ruptures or tears loose a valvular attachment. During twenty years ending in 1903, D. G. Hall found 112 cases recorded (26 had aneurysm in the valves, and 25 in the coronary vessels); Völker alone made a diagnosis. *Rupture* of the heart occurs through the left ventricle (80 per cent.).

Pathogenesis.—The *most frequent cause is disease of the coronary arteries,* which are incessantly active and are end-arteries, and anastomose with each other only through their capillaries. The vessels of Thebesius, entering the muscle from the auricles and ventricles may carry on some collateral circulation when the coronary arteries are occluded. *Coronary narrowing* may be *gradual,* by obliterative endarteritis or by the occlusion of the vessel from an atheromatous plaque in the aorta; or it may be sudden and fatal by embolism or thrombosis in an already narrowed vessel. The most common location for coronary obstruction is in the left ventricle (anterior coronary artery which is relatively small) and in the interventricular septum. In acute coronary obstruction, the heart muscle becomes ischæmic, pale and opaque and the necrotic area becomes swollen and prominent (coagulation necrosis); a wedge-shaped anæmic *infarct* is less common. The muscle fibers become fragmented, lose their nuclei and disappear; this process may lead to rupture of the heart or sudden death. Ziegler named it *myomalacia cordis.* If the subject lives, scar-tissue develops—a *dystrophic sclerosis.* Such "myocarditis" is not inflammatory. Huchard employs the terms, arteriosclerosis of heart and arterial cardiopathy, to emphasize the causal role of arterial disease. *Coronary arteritis* most frequently runs a subacute or chronic course. Its causes are those of arteriosclerosis (*q. v.*). A less frequent cause is *actual chronic myocarditis* following *acute myocarditis* due to rheumatism, typhoid or diphtheria. French writers describe an *acute endarteritis* following acute infections and leading to myofibrosis. Köster, Rühle and others have found myocarditis in valvular disease. Dehio holds that *myocarditis or myofibrosis, is a result of dilatation,* that it occurs where the muscle is destroyed and is therefore a necessary and protective process, conforming with Thoma's conception of arteriosclerosis (*q. v.*).

Symptoms.—(*a*) In the *latent type* there are no symptoms and sudden apoplectiform death occurs, while the subject is apparently in perfect health. (*b*) Most cases present chronic symptoms of *cardiac insufficiency.* The cervical veins are enlarged and there is general venous congestion. The *pulmonary circulation suffers* especially, as is shown by cyanosis, duskiness of the skin, infarcts which are often large (Rühle), hydrothorax, œdema of the lungs, bronchitis and by dyspnœa which is often nocturnal or develops on exertion. The heart symptoms are precordial oppression or dull pain, palpitation, cardiac asthma or genuine angina pectoris. Examination of the heart sometimes gives negative results, but *dilatation* and hypertrophy are usually present (cardiomegalia). The *heart tones* at first are often clear, but later become *faint and irregular,* so that in the

resulting delirium cordis, the tones are disorderly and the phase of the murmur, due to relative muscular insufficiency or to arteriosclerotic dilatation, cannot be distinguished. The second pulmonic sound is accentuated or split and the second aortic tone is weak, because the arterial tension is low. The *pulse* is *usually irregular* (Rühle, Riegel, Bard, Phillippe), either from the beginning or becoming so later, with paroxysms of arrhythmia which may be an extension of the degeneration and sclerosis. Irregularity usually precedes the cardiac insufficiency and persists during restoration of compensation. A regular pulse may be present throughout (Ebstein and Renoy), or a number of regular strong beats is followed by a wave of rapid tumbling heart action. Simple intermission and the pulsus bigeminus are also seen. The pulse rate is often slow at first, and gradually increases to 90 or 100, with ominous crises of tachycardia. In some instances the pulse is persistently slow and is associated with syncope from brain anæmia (*v. i.* BRADYCARDIA). It is seen, then, that the rate and rhythm of the pulse vary, and though irregularity is of important diagnostic value there is no constancy in the pulse findings.

Œdema is present, but as a rule is less common and intense than it is in valvular disease (*q. v.*). Embolism may occur. Digestive disturbances are almost constant (Rühle), and Huchard maintains that they have an important bearing on the heart's action; alimentary autotoxæmia from carelessness in diet, may cause paroxysmal cardiac symptoms. Potain and Barie hold that digestive disorders induce a reflex contraction of the lung vessels and thus lead to dyspnœa. The urine is that of stasis in the later stages, but early in the disease polyuria may prevail from high blood pressure. Renal insufficiency may lead to renal toxæmia (Houchard).

Diagnosis.—There are five cardinal points: (*a*) In the majority of cases *arteriosclerosis* is most important. Its causes should be sought in the anamnesis and the peripheral arteries and aorta should be examined for its usual signs (*q. v.*). Arteriosclerosis is usually general, but may exist in the heart without being seen in the radial arteries, and the converse is also true. (*b*) The *heart is usually dilated* and hypertrophied; *i. e.,* its transverse measurement is increased. (*c*) *No valvular murmur exists,* in uncomplicated cases. The systolic apical murmur of arteriosclerotic dilatation may be heard or the soft variable bruit of relative mitral leakage, which often disappears with rest and catharsis. (*d*) *The pulse is fast and irregular.* A regular pulse does not absolutely exclude myocarditis. Arrhythmia, whether congenital, neurasthenic or caused by tobacco, must be excluded. (*e*) *Dyspnœa* and pulmonary stasis are suggestive.

Differentiation is often so difficult that many clinicians limit the diagnosis to *muscular insufficiency;* for fatty heart, idiopathic hypertrophy and dilatation, or pericardial adhesions may be found at autopsy instead of the anticipated myocarditis. Sometimes when myocarditis is suspected, the heart shows nothing microscopically or

macroscopically, thus demonstrating that the causes of heart failure are often beyond detection by the microscope. A diagnosis is made by exclusion, and then is often only a "probability diagnosis." In distinguishing myocarditis from the "heart of *renal disease,*" long observation, the gallop-rhythm (more common in the *cor renale*) and uræmic manifestations are helpful. Myocarditis and renal disease are frequently associated. From *mitral stenosis:* Both lesions cause irregularity and a weak radial pulse and apex beat; when the presystolic stenotic murmur is absent, the loud second pulmonic sound, the loud snapping apical tone and the wide right heart are most suggestive of mitral stenosis. Digitalis very frequently fails to act in myocarditis. From *cerebral apoplexy:* The sudden "stroke" or "cardiac apoplexy" of coronary disease may be the first sign of myocardial disease and may be mistaken for cerebral apoplexy or embolic processes; the autopsy, in cases of sudden death often discloses coronary closure with myomalacia cordis or fibrous myocarditis.

Prognosis.—The prognosis is that of uncompensated valvular lesions with the extra consideration that the muscle itself and its nutrient arteries are involved. Because of the facts that digitalis is often inefficient in these cases, and that angina pectoris, cerebral anæmia, heart rupture or coronary closure may occur, the outlook is most unpromising. Some patients, especially elderly subjects, may live a decade or more, until intercurrent pneumonia or another secondary affection causes their death. Early digestive symptoms are less favorable than early œdema or congested liver.

Treatment.—The therapy is (*a*) that of the *causal factor,* arteriosclerosis (*q. v.*) being the most common cause. Potassium iodide relieves pain and lessens the atheroma; it should be given in doses of 5 to 10 grains after meals, freely diluted in milk or water—

℞ Potassii iodidi ..... ʒiiss.
Syr. sarsaparillæ co. ..... ʒiv.
M. et S.—One teaspoonful in a glass of water after meals.

(*b*) *Dietetic;* stimulants should be used carefully and overexertion and excitement should be avoided. (*c*) *The Schott treatment* (*v. s.*). (*d*) *Cardiants.* Digitalis is beneficial in the early stages—less so in advanced cases. It is to be avoided when the pulse is slow. Strychnine is frequently valuable. When there are anginal seizures or hard vessels, nitroglycerine should be given (see THERAPY OF VALVULAR DISEASE). Caffeine, camphor, morphine and alcoholics should be administered for cardiac asthma.

RUPTURE OF THE HEART.

Etiology.—(*a*) Spontaneous rupture is practically unknown. (*b*) *Rupture through the altered heart muscle* occurs in myocarditis; heart aneurysm, in which case it may even perforate the chest wall; myomalacia cordis from coronary atheroma and embolic occlusion;

coronary aneurysm; fatty heart, abscess, gumma or neoplasm of the heart, ulcerative endocarditis, obstructive valvular disease and stenosis of the isthmus of the aorta. It occurs largely in men over sixty years of age. It may occur during sleep or may be caused by trauma, vomiting, great effort, excitement, delivery, coitus or by the abuse of digitalis. Kroll collected 332 cases of rupture of the heart.

Pathology and Symptoms.—(*a*) *Partial rupture* of the valves induces sudden valvular insufficiency. (*b*) *Total rupture* of the heart is usually single, though a case having five rents is recorded, and takes place in the left ventricle (80 per cent.) where the pathological factors come most into play. *Sudden death* may occur without prodromes, or death may result after a few hours to eleven or seventeen days. The symptoms are a sense of something giving way in the chest, great precordial pain, incoërcible vomiting or diarrhœa, cerebral anæmia or irritation of the vagus from hæmopericardium, collapse and death from pressure of the blood on the heart or the venæ cavæ (Cohnheim). Hæmopericardium (*q. v.*) may be suspected because of greatly increased triangular dulness, faint heart tones and absent or weak apex beat within the left border of the dulness.

Diagnosis and Treatment.—The diagnosis is rarely made. It depends on (*a*) the *etiology,* (*b*) *increased dulness,* (*c*) *cardiac insufficiency* and (*d*) *symptoms* of *internal hemorrhage.* The *outlook* is bad and the *treatment* wholly symptomatic; the most absolute rest should be enforced.

Heart injury was first operated on by Farina (1896). The first cardiorrhaphy for rupture was by Rehn (1897). Frazier (1906) collated 102 operations for injury, of which 42 per cent. recovered.

Disease of the Coronary Arteries.—This has been sufficiently considered under myocarditis (heart aneurysm) and rupture of the heart. It will be considered again under angina pectoris, which anatomically belongs to disease of the heart muscle though it is usually described under cardiac neuroses.

TUMORS OF THE HEART.

These are most rare because the heart is rather insusceptible even to neoplasms in its immediate vicinity. They cannot be diagnosticated. Carcinoma and sarcoma are the most frequent, and lipoma, myxoma, fibroma and myoma are infrequent. They are usually secondary. Cardiac insufficiency, valvular compression or embolism sometimes develops.

Parasites are most often accidental postmortem findings. In Mosler's monograph echinococcus is the most frequent and occurs twice as often in the right ventricle as in the left. Grulee (1905) collected 55 cases of echinococcus of the heart. Cardiac rupture, sudden death from occlusion of the pulmonary trunks, relative pulmonary insufficiency or cardiac incompetence may occur. The cysticercus, the pentastomum and trichina are of purely anatomical interest.

SYPHILIS OF THE HEART (See Syphilis).

CARDIAC THROMBOSIS—THROMBOSIS CORDIS.

This usually occurs at the time of or after death, and is of little clinical interest.

Symptoms.—(*a*) Entire latency. (*b*) Thrombosis sometimes develops during life and leads to pulmonary or arterial embolism. (*c*) *Sudden cardiac weakness.* (*d*) *Sudden valvular* stenosis, a ball thrombus (*kugelthrombus*) forming in the auricle, and occluding, for instance, the mitral orifice; it is beyond clinical recognition, although according to von Ziemssen, it is frequently associated with gangrene of the lower extremities; in Curschmann's case an ascites resembling chronic peritonitis was the sole finding.

DISEASES OF THE ENDOCARDIUM.

ENDOCARDITIS.

Definition.—Inflammation of the endocardium. Its clinical importance attaches to the fact that chiefly the valves of the heart are involved. Two main varieties are distinguished, the *acute* and the *chronic.* Known to Morgagni and Boerhaave, Bouillaud, in 1840, first placed the disease on a scientific basis.

I. Acute Endocarditis.—This is always a *secondary, general* disease, and is *mycotic* in origin, excepting atheromatous endocarditis. Two main clinical varieties are described: (*a*) Malignant or ulcerative endocarditis, and (*b*) simple or verrucose endocarditis. They *differ only in degree,* and the term benign is only relative, as most endocarditides entail serious sequels. Litten divides endocarditis into (*a*) the *benign* forms produced by rheumatism, chorea, gonorrhœa, scarlatina, measles, variola, typhoid, diphtheria, pneumonia, etc.; and (*b*) *malignant,* (i) non-suppurative and (ii) suppurative or septicopyæmic.

(A). Malignant Endocarditis.—This variety is also known as ulcerative or septic.

Etiology.—The heart lesion is either (1) *secondary* to some clearly causal infection or disease; or (2) *cryptogenetic,* when the causal infection is not apparent, but care in examination and at autopsy usually reveals the cause. The atrium, bacteriology, mode of extension and symptoms are described under Septic Infections.

The disease is more common in women than in men, even excluding puerperal infections; it is most frequent between the years of twenty and forty. Its frequency is 2 per one thousand patients. *Seventy-five per cent.* of cases *develop upon an old valvular lesion* by which the local physiological resistance is lessened. The literature contains 11 cases developing at a patent ductus Botalli (Hart, 1904).

Pathology.—Malignant endocarditis most often involves the *left heart,* because it is far more frequently the seat of previous lesions and its arterial blood favors the growth of microörganisms, although the right heart is involved more frequently than it is in other types of endocarditis. Lenhartz found the left heart affected in 86 per cent., the right in 12 per cent. and both sides in 2 per cent., of his cases.

Though the process may involve the walls (parietal or mural endocarditis), it most frequently attacks *the valves* (valvular endocarditis), which are often already indurated by previous simple endocarditis, are most washed by the circulating blood and are subject to greater mechanical activity. The endocardium becomes opaque, and irregular deficits occur on the under surface of the aortic or the auricular surface of the mitral valves; later there is injection of the endothelial coat and a grayish deposit. Exudation is abundant on the mitral valves, but is late or scanty on the less vascular aortic or pulmonary valves. *Thrombi* and excrescences develop on the erosions, whose removal discloses subjacent *ulceration.* The thrombi, especially in pneumococcic and gonococcic infections, consist of lamellated fibrin and bacteria. The *vegetations* may attain the size of a walnut and often extend from one valve to another by contact infection; some thrombi are purely mechanical deposits of fibrin, but *genuine endocarditis is always mycotic.* Heiberg first found microorganisms (1869) and Weichselbaum first obtained pure cultures. Efforts to classify endocarditis by the bacteriological findings are more or less impracticable (*v. i.*). The following bacteria have been found: Streptococcus, staphylococcus, pneumococcus, less frequently the gonococcus, colon, diphtheria, influenza, tubercle and typhoid bacillus, meningococcus and, very exceptionally, other bacteria. The same organism may produce either the benign or malignant form. *Experimental endocarditis* (Wyssokowitch, Ribbert, Köster) has been produced by inoculations of various organisms, usually after wounding the valves by a sound introduced through the cervical vessels, but also without trauma. The pyogenic organisms are said to attack more frequently the mitral, and the pneumococcus rather selects the aortic valves. The mitral valves in general are more often involved than are the aortic. The process may invade the aorta, the pulmonary artery or the lung. Destructive changes by ulceration through one leaf of the valve may produce acute *valvular aneurysm,* which, when aortic, projects toward the ventricle, and when mitral, projects toward the auricle. If the necrosis destroys both lamellæ of the valve, the perforation leads to valvular insufficiency. Ulceration of the chordæ tendineæ or papillary muscles also produces valvular insufficiency; necrosis of the parietes results in a "heart ulcer," which sometimes leads to communication between the ventricles or to rupture of the heart. Dislodgment of tissue particles, thrombi or bacteria occurs frequently, and usually in the arterial circuit. The effects vary with the location of the *infarct* (see SYMPTOMATOLOGY) and with the character of the microbes; *i. e.,*

emboli act mechanically and infectively. Streptococci are said to produce anæmic infarcts, while staphylococci result in suppuration. Pathological changes *in other organs* are those of (*a*) general *sepsis,* such as parenchymatous degeneration or splenic tumor, (*b*) *embolism,* mechanical or infective, (*c*) *stasis* when the heart fails and (*d*) the primary disease.

Symptoms.—The clinical features vary to such an extent, according to the basic disease, the visceral involvement by embolism and the virulence of the microörganisms, that a general description is impossible. The onset may be violent or most insidious. The autopsy findings in the *latent* cases may be surprising, as, for example, the discovery of a mural gonorrhœal endocarditis in a case of septicopyæmia. In the majority of cases, the septic element prevails. The *various types,* artificial though convenient for description, are:

1. The Typhoid Form.—The toxæmic symptoms, such as apathy, delirium, dry tongue, distended abdomen, diarrhœa and splenic tumor are suggestive of typhoid. The resemblance may be heightened by hemorrhage of the bowels, rose-like spots and the diazo reaction. Dilatation of the right heart and cardiac murmurs, so important in endocarditis, are not uncommon in typhoid. But at this point certain variations arouse doubt; there is no Widal and no typhoid bacillæmia and certain septic symptoms or cardiac signs come to the front. In the *skin, embolism* appears as spots with a white or yellow centre and hemorrhagic periphery, or as pemphigus, gangrene or suppurative foci. Analogous areas may be seen in the upper air-passages. Infarcts sometimes occur in the conjunctiva, but in the *retina,* simple hemorrhage or actual infarcts with yellow centres are frequently detected. The entire eyeball may suppurate. The frequent *splenic infarction* is characterized by sudden pain, tenderness, enlargement and sometimes perisplenitic friction. Embolism is often accompanied by fever, chill and vomiting. *Renal infarction* is very frequent, and is attended by pain in the loins, hæmaturia, albuminuria and sometimes anuria or dysuria. Severe nephritis is common. *Cerebral embolism* usually occurs in the left Sylvian artery and results in the usual stroke and hemiplegia with aphasia. *Pulmonary infarcts* are caused by lesions of the right heart or by auricular thrombi, and occasion sudden pleuritic pain, dyspnœa, hæmoptoë, temperature and possibly a small area of crepitant rales, pleural friction or consolidation. Embolism also occurs in the liver, in the mesenteric artery (with enterorrhagia and peritonitic symptoms), in the muscles and joints, testes and parotid; in the heart and the various mucosæ; in the meninges, pleuræ, pericardium, peritoneum and in the peripheral arteries, sometimes causing gangrene or embolic aneurysm. The pneumococcus is often the cause of this type.

The *course* may be peracute (two to four days), acute (a few weeks) or chronic (several months). Death occurs from the septic typhoid state, from embolism, or from cardiac failure.

2. The Septic or Intermittent Type.—This form has an ir-

regular fever-curve, with chills and drenching sweats, and the spleen is enlarged. Heart symptoms may be absent or indeterminate, the moderate relaxation of the right heart and the murmur often being called functional. The paroxysms are more irregular than in malaria and no response to quinine is obtained. Sepsis is considered but the localization is not immediately determined. The streptococcus is often causal. Cardiac findings, embolism or clearly septic manifestations may establish the diagnosis. The course is usually several weeks but may cover several months to even a year.

3. The Visceral Forms.—These forms embrace the cases in which some one organ is especially involved. In the *cardiac form,* anæmia exists and the heart murmur seems accidental, until a typical valvular lesion develops, or, if one already be present from older valvular disease, until another appears, as a diastolic following a systolic murmur. The diagnosis may be difficult even when chronic valvular disease is present. In the *cerebral form,* meningitis, apoplexy, coma or psychoses may dominate the clinical picture. The symptoms referable to any organ which is the seat of embolism (lung, pericardium, etc.) may seem to indicate a primary disease. In this way acute yellow atrophy, pernicious anæmia, tuberculosis, nephritis, rheumatism, cholera, etc., have been incorrectly diagnosticated.

Special Symptoms.—(1) All types of *fever,* from intermittent to continuous, have been noted, although it may be absent for a long time. (2) The *heart* is *always somewhat disturbed* in its action, cardiac unrest being common. Its action is usually more frequent, but is quite variable, now being rapid, again slow and often irregular. The heart seems to beat harder and the pulse may throb, although the irritable overaction is attended by low arterial tension. The heart tones are dull. Palpitation is frequent. Cyanosis is not common. (3) *Respiration* is *always increased,* from toxæmic centric stimulation, pain or respiratory complications (septic pneumonia, infarcts, pleurisy, pneumothorax, etc.). (4) The *digestive tract* may be affected. Nausea, vomiting and diarrhœa may occur from the general sepsis. Embolism has been mentioned. The liver is enlarged, may be tender or may be the seat of friction. Icterus may be marked, is ominous and is referable to duodenal catarrh or to oversecretion of bile (polycholia). (5) The *spleen* is almost always palpably enlarged from sepsis or embolism. (6) The *urine* shows evidences of the sepsis or stasis by albuminuria, with or without cylindruria, and of the "blood dissolution" and embolism by hæmaturia. Malignant endocarditis may run a course similar to that of subacute nephritis, which is present in 33 per cent. of all cases of the ulcerative type. (7) The *nervous system* may be irritated or dulled by the sepsis toxins, or embolic lesions, such as meningitis or encephalitis, may intervene. (8) The *muscles* are frequently tender and actually inflamed and may undergo suppuration. The bones are often tender and the *joints tender or swollen,* although the swelling is more frequently peri-articular than intra-articular. Bacteria are seldom

found, and this may heighten the resemblance to rheumatism. (9) The *skin* may be the seat of embolism (*v. s.*) or of eruptions, such as sudamina, erythema, *purpura* or *polymorphous septic rashes* from bacteria or their products in the circulation. Bullæ, gangrene, phlegmons or bed sores are also observed. (10) The *blood* usually exhibits marked anæmia; when there are no heart findings, the anæmia and splenic tumor have been confused with pseudoleukæmia. The leukocytes are often but not always increased. Bacteria may be cultivated from the blood by modern methods of inoculation from a large quantity of blood in much bouillon; the latter is considered under SEPTICOPYÆMIA (*q. v.*).

BACTERIOLOGICAL FORMS.—(*a*) *Streptococcic Endocarditis.*—The streptococcus is the usual cause of rapidly fatal and of chronic ulcerative endocarditis, both with severe repeated chills and high irregular fever. (*b*) *Staphylococcic endocarditis* is less frequent. The course is nearly always acute and is attended by high fever, pustules in the skin, disseminated hemorrhages and septic infarcts, especially in the kidneys. (*c*) *Pneumococcic endocarditis* (see PNEUMONIA) is usually acute; frequently follows pneumonia, less often cholecystitis; begins with gradual fever rise associated commonly with chills; and is often attended by suppurative meningitis. (*d*) *Gonococcic endocarditis* (Ricord, 1847) may result from Neisser's coccus or from mixed infection. Cases of recovery are instanced.

Diagnosis.—Bamberger's remark of half a century ago still holds: "The diagnosis of acute endocarditis is seldom easy, usually difficult and often impossible." Unless there are physical changes in the heart, the diagnosis is only presumptive. A proper etiology, the presence of an old valvular lesion, fever or other evidences of sepsis, signs of cardiac insufficiency, such as blue nails, cold extremities, irregular weak pulse with apparently strong cardiac shock and dilatation are very suggestive and may be confirmed by continued observation, in the course of which there may develop embolism, change in the valvular murmur and positive blood-cultures. Actual hypertrophy of the heart not only requires time for development but adequate nutrition, of which the disease seldom admits. The misleading visceral forms have been discussed (*v. s.*). In 37 per cent. of cases, the heart is negative, in 44 per cent. the murmur is systolic and in 5 per cent. diastolic. Absolute certainty rests upon our ability to diagnosticate by physical signs, the presence of a mitral regurgitation (*q. v.*) which is the most frequent form, or of an aortic regurgitation (*q. v.*), plus the *signs of sepsis* of which the disease is a part ("arterial pyæmia"). (See TYPHOID, differential table.) The valvular murmur may disappear after embolism.

Diastolic murmurs are very rarely functional and their appearance is therefore especially suggestive. Myocarditis may produce a systolic murmur, but is usually attended by marked early muscular insufficiency, weak-heart shock and irregular pulse, weak systolic murmur and a moderately accentuated second pulmonary tone.

Prognosis.—The prognosis is most unfavorable; Herrick (1902) collected 22 cases of recovery. It depends on the strength of the heart, the bacteriology, the number of infarcts and their location, the variability of the heart murmurs (which may indicate danger of embolism), and the intensity of the sepsis. Recovery from the gonorrhœal form is relatively frequent. Musical murmurs with fever are usually ominous. Apparently mild endocarditis may become severe. The course may be rapid or severe, of a few days duration, or a slower evolution of over a year.

Treatment.—(See BENIGN ENDOCARDITIS and SEPTIC INFECTIONS.)

(B). Acute Verrucose or Benign Endocarditis.—This variety is more frequent, constituting 1.5 per cent. of clinical cases, and is always secondary.

Etiology.—(*a*) *Rheumatism* (*q. v.*) is the most frequent cause and accounts for 60 to 85 per cent. of the cases. Over 20 per cent. of rheumatic patients develop endocarditis, particularly in the severe infections. In children it is very frequent, for in them the heart "resembles an articulation." Muscular rheumatism, angina or erythema nodosum may occasion endocarditis. Chronic rheumatism predisposes to it. (*b*) *Chorea* is frequently complicated by endocarditis (30 per cent. of clinical and 82 per cent. of fatal cases). By some writers chorea is considered rheumatic but the majority hold that it is only correlated. (*c*) Of other *specific infections,* scarlatina ranks first and causes 10 per cent. of the cases. Endocarditis is far more uncommon after typhoid, diphtheria, tonsillitis, measles, erysipelas, gonorrhœa, variola, grippe, tuberculosis, etc. (*d*) Occasionally it is associated with trauma, burns, syphilis, cancer, Bright's disease, gout or diabetes. (*e*) It is most common in males between the years of twenty and thirty.

Pathology.—The vegetations, due to bacteria, are verrucose, wart-like, papular or even pedunculated, are usually small and occur at or above the lines of closure of the valves, whereby their function is interfered with and valvular insufficiency or stenosis results. At times the vegetations are cauliflower-like or resemble condylomata. They are grayish, gelatinous and transparent; they become whiter and firmer with age and consist of two strata, the deeper being granulating endocardium and subendocardial tissue and the superficial layer consisting of thrombi from the coagulable elements of the blood. The signs of active inflammation, such as redness or swelling, are usually lacking, redness being sometimes confused with post-mortem imbibition of hæmoglobin from the blood. Vegetations must not be confused with Albini's nodes, which are seen in infants and children. The vegetations are at an early stage, quite cellular from leukocytic exudation and tissue proliferation and usually contain bacteria (see ULCERATIVE TYPE). The outgrowths becoming detached, *emboli* are released and produce mechanical hemorrhagic or anæmic infarcts, not suppurative infarcts as in the ulcerative type. The fate of the vegetations is (*a*) *embolism;* (*b*) *organization* with the bare possi-

bility of restoration to normal; (*c*) more often, *organization with valvular lesions* resulting from retraction, induration and calcification; (*d*) being a *locus resistentiæ minoris, recurrent endocarditis* may be observed; (*e*) the intensity of the inflammation may increase, leading to malignant endocarditis.

Location.—As in the first type, the vegetations are largely on the same surface of the same valves and in the left heart, because of its greater physiological activity and its arterial blood; these same conditions in fœtal life predispose to endocarditis of the right heart, either on the valves or on fœtal openings or defects. In general, the mitral valves are most affected (50 per cent. of the cases alone or 80 per cent. when combined with other lesions), next the aortic (13 per cent. alone or 40 per cent. when combined with other lesions), and far less frequently, the tricuspid, the pulmonary and the walls of the heart, chordæ or parietes.

Symptoms.—The symptoms are not characteristic and a diagnosis is impossible or only presumptive unless the valves are considerably involved. Simple endocarditis therefore is found far more frequently at autopsy than at the bedside; *i. e.,* it is very often *latent.* With such heart symptoms as increased rate or palpitation, it is difficult or impossible to determine whether they are caused by a valvular lesion or a myocarditis, though the latter is the more likely cause. Systematic daily examination of the heart in such diseases as rheumatism may reveal the gradual involvement of the endocardium, with moderate fever and dyspnœa, cardiac unrest, oppression, palpitation or insufficiency. Pain is more often pleuritic or pericarditic. The diagnosis depends on the valvular bruit, which often follows muffling or irregularity of the cardiac sounds, and is still later followed by the signs of mitral or aortic insufficiency (*q. v.*). The infrequent diastolic murmurs are more distinctive than the systolic, for they are rarely functional. *Embolism* is very distinctive, as it is in the first type, and is important mechanically rather than bacteriologically. It also occurs in parietal endocarditis; embolism may be so minute as to escape recognition.

Diagnosis.—Conservatism in making the diagnosis is necessary, because acute benign endocarditis is much too often diagnosticated. The most conspicuous symptom is *fever,* though it is not characteristic and may blend with that of the causal disease; the most important findings, objectively, are the signs of valvular heart disease; for these findings, one must often wait, because in many cases the evolution, alone, is final. More stress should be placed upon the results of percussion than on the murmur itself. The murmurs are mistaken for functional murmurs accompanying febrile relaxation of the heart-chambers or myocarditis, which also always accompanies endocarditis.

Recurrent endocarditis shows not only the fever and other symptoms caused by the fresh lesions, but also the *hypertrophy* of the older valvular lesion. An aortic diastolic or a mitral presystolic murmur is more likely to indicate an *old* lesion. With repeated examinations

fresh murmurs may be heard in addition to the old ones. (See MITRAL INSUFFICIENCY, ULCERATIVE ENDOCARDITIS and PERICARDITIS.)

Prognosis.—(*a*) *As to life,* the outlook is generally good, unless (i) emboli lodge in important structures, such as the basilar or coronary arteries, or (ii) the process becomes malignant. (*b*) *As to complete recovery,* (i) valvular lesions are usual which are anything but "benign"; (ii) fresh recurrent endocarditis is very common; and (iii) complete recovery is uncommon.

Treatment.—The treatment of the two forms of endocarditis is considered together. (*a*) *Prophylaxis* resolves itself into the treatment of the causal acute infection, such as rheumatism or sepsis. Preventive treatment, such as the administration of salicylates and alkalies in rheumatism, is uncertain except that they shorten the rheumatic attack. (*b*) *Rest* spares the heart and probably lessens the percentage of cases involved. Complete rest, psychical, physical and cardiac is the prime essential and cardiac stimulants are to be avoided, at least in the early stages. Rest in bed must be enforced for weeks after temperature and signs of heart weakness have wholly disappeared. Should relative recovery occur, the general care of the heart's strength must be insisted upon as in chronic valvular disease and, with intercurrent affections or with the appearance of temperature, the patient must be confined to bed. Senac (1749–1783) recognized the causal importance of inflammation in heart disease, the necessity of avoiding strain and of regulating the amount of fluid and food ingested, the relations of liver and stomach disturbance and the advantages of mental quietude. (*c*) The *sepsis* is treated by alcohol (see PNEUMONIA); quinine, gr. x to xv, once or twice daily, with three ounces of red wine every hour or two, is recommended by Fräntzel; bichloride of mercury and other antiseptics are futile; for pure or dominating streptococcic infections Marmoreck's antistreptococcus serum is indicated in doses larger than those usually prescribed and should be frequently repeated. (*d*) The *irritable heart* may be quieted and slowed by the use of the ice-bag. Sinapisms sometimes act favorably but blisters are to be avoided. Cardiac stimulants should be employed with caution and only coffee, ammonia or brandy should be given. Digitalis is usually to be avoided, but it is indicated when very tumultuous heart action threatens to induce embolism.

(*e*) *Symptomatic Treatment.*—For nervous symptoms, sodium bromide is indicated. Sulphonal and trional are to be avoided when the heart weakens. For the fever, hydrotherapy, aside from simple sponging, is inadvisable, for the joints and muscles are very tender and movement, on account of the possibility of embolism, is dangerous. Diarrhœa of septic origin should not be checked at once, because it is nature's effort at derivation and elimination. Fluid diet should be given because of the fever. Potassium iodide is usually administered as a resolvent after the acute stage has passed.

II. Chronic Endocarditis. Chronic Valvular Disease.—Etiology.—Valvular heart disease constitutes 5 per cent. of all diseases. It most often develops from (*a*) *acute verrucose endocarditis* and therefore has the same etiology; in 60 per cent. the cause is rheumatism. One-half the cases of valvular disease are in children and over one-third occur between 20 and 30 years of age. Some cases develop without symptoms and are apparently primary or possibly hereditary; they are seen mostly in young or adolescent subjects and in women, but doubtless some minor or forgotten infection explains this group. (*b*) *Atheroma* is also an important cause; it is promoted by overexertion, as in the working classes, by advanced years or by chronic nephritis, gout, diabetes, syphilis, alcoholism and lead poisoning. It is a more common cause in males. Though chronic valvular disease may develop from other causes than chronic endocarditis, both topics can well be considered together. Valvular lesions less often result from (*c*) *rupture of the valves;* this occurs most often in the aortic valves during supreme physical efforts and, of course, is favored by previous valvular lesions, although sometimes observed in perfectly normal valves. Healing is most rare. (*d*) *Relative valvular insufficiency* is a condition in which the normal valves are unable to close the orifices to which they belong because the heart is dilated. It is most commonly tricuspid and mitral, although it rarely occurs in the aortic and pulmonary valves. Relative stenosis is not clearly established. (*e*) *Other rarer causes* are aortic aneurysms, neoplasms, valvular aneurysms and cardiac thrombi.

Pathology.—Small grayish-red swellings are sometimes seen in the early stages; they somewhat resemble atheroma plaques. Foci of endocardial thickening and retraction (*endocarditis retrahens*) are seen, in which calcification and even ossification of the valves may be present. The valves become less translucent, are not infrequently rigid, unable to unfold, adherent to each other or distorted by their own thickening and retraction or that of the chordæ or papillary muscles. Mural changes are unimportant, except when associated with myocarditis. Acute exacerbations may occur and then the ordinary vegetations appear. Embolism occurs in about 30 per cent. of the cases. The myocardial changes are hypertrophy, dilatation and myocarditis. The valves most often involved are the mitral (78 per cent.), especially in adolescence, then the aortic (20 per cent.), which are especially subject to change in arteriosclerotics and finally the tricuspid (1 per cent.) and the pulmonary valves. Combined valvular lesions occur in 33 per cent. of cases. The process often extends by continuity of surface from the mitral to the aortic valves. When one valve is but moderately involved, the others of the same valvular orifice may compensate by stretching in order to cover the defect, especially in aortic disease. In fœtal cases the valves of the right heart are more frequently involved (*v.* CONGENITAL VALVULAR DISEASE).

Valvular insufficiency is usually caused by (*a*) thickening and re-

traction or even calcification of the valves and also of the chordæ tendineæ and of the papillary muscles; (*b*) massive vegetations; (*c*) growing together of the valves, or of the valves with the wall of the heart or aorta; or adhesions between the papillary muscles and tendons; (*d*) valvular aneurysm, perforation, rupture, or tearing of the tendons or muscles. *Valvular stenosis* (stenosis of the orifice) results from (*a*) adhesions between the valves, (*b*) retraction, calcification, fibrosis of valves or ring of insertion and (*c*) vegetations. Combined valvular insufficiency and stenosis in one valvular mechanism, especially the mitral, are very common.

Symptoms.—The symptoms of valvular disease are (*a*) the *cardiac findings* peculiar to each lesion, which will be first considered, and (*b*) the *general symptoms* more or less common to all forms of cardiac insufficiency, the description of which will follow the individual lesions.

III. Aortic Insufficiency. (Corrigan's Disease.)—Aortic regurgitation was described by Cowper (1705), Vieussens (1715) and Hodgkin (1829), but the first full description dates from Corrigan (1832).

Etiology.—1. Endocarditic Form.—The sinuses of the valves may fuse, vegetations are present, or the valves grow together at their margins and shrink; this is more frequent than valvular perforation or ulceration, which is rapidly fatal. If calcification develops, there may be coincident stenosis, which is less common in the atheromatous form. This form usually follows a previous mitral lesion. The endocarditic form occurs at an earlier age and is not attended by the arteriosclerotic changes in the aorta and coronary arteries which characterize the atheromatous form.

2. Atheromatous Form.—Atheroma may be slight in the aorta and located mostly on the valves. It is a slow, degenerative process (see Arteriosclerosis) with marginal valvular retraction; it is caused by excessive use of alcohol, by continued hard work or athletics and by syphilis, which may dilate the aortic ring, even without directly involving the valves (syphilitic aortitis). It is often therefore seen in middle-aged men. A relatively slight anatomical lesion may produce disproportionate symptoms. The valves may become adherent to the aorta. Their edges are rounded and are not the seat of endocarditic masses. The atheromatous process may overlie or actually invade the coronary orifices; this is obviously dangerous. Coincident stenosis is uncommon. The following forms are infrequent:

3. Relative Insufficiency.—This form occurs mostly in cases of aortitis, aneurysm and arteriosclerotic *dilatation of the root of the aorta,* which is a more or less physiological process, as the aorta usually relaxes one centimeter in circumference after the fortieth year. It may also occur in cases of *dilatation of the left ventricle,* resulting from idiopathic hypertrophy, myocarditis or renal disease.

4. Congenital Defects.—These are rare; absence of a valve may be in part compensated for by the others, though anatomical anomalies

predispose to sclerosing endocarditis. Valvular fenestration is unimportant clinically and pathologically.

5. Trauma.—This is a most uncommon cause, only 46 aortic cases in a total of 72 being recorded (Drüyfuss).

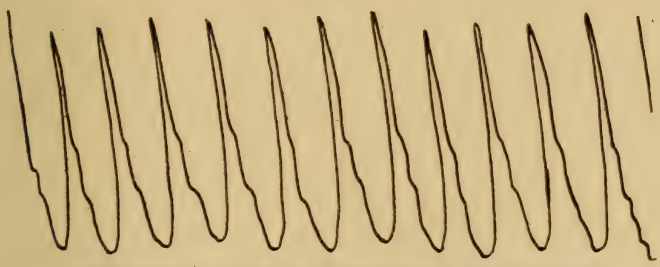
Mechanism.—From aortic valvular incompetence, a diastolic regurgitation of blood into the left ventricle takes place from gravity and contraction of the aorta. Blood currents and vibrations are caused by the blood flowing (normally from the left auricle and pathologically from the aorta) into the left ventricle, thereby producing the *diastolic murmur.* The *left ventricle becomes dilated* to accommodate its added volume and *hypertrophied,* because it must propel the normal plus the regurgitated blood. The left heart becomes enormously increased and may even weigh 50 ounces, whence the name "ox-heart," —*cor bovinum.* The hypertrophied left ventricle suddenly forces an unusually large amount of blood into the arteries, and thus causes the many *arterial phenomena;* H. A. Stewart finds that the amount of regurgitated blood is small.

Physical Signs.—1. Inspection.—(*a*) *Precordial prominence* is observed chiefly in plastic chests and in the endocarditic type and, therefore, is most common in women and children. Mensuration may confirm the prominence, although the right chest normally measures nearly an inch more than the left. (*b*) The heart *shock* is powerful and diffuse. (*c*) The *apex beat* is *dislocated downward* (6th, 7th, 8th interspace) and outward, beyond the nipple or anterior axillary line. The apex also lies low in arteriosclerotics, from relaxation of the aorta. It is *heaving,* pulsating and superficial. The lung is pushed back and some slight rotatory systolic retraction of the chest wall is seen. These findings are referred to the dilated and hypertrophied left heart. (*d*) Pulsation in the first and second right intercostal spaces is usually dynamic, because the blood thrown with extra force into the aorta causes dilatation of the aortic arch. (*e*) The *peripheral vessels pulsate with unusual violence* and the carotids hop; pulsation in the jugular fossa or in the abdominal aorta may simulate aneurysm, and the temporals and radials often attract attention before the patient undresses for examination. The crossed leg may pulsate and the head may throb. These are all caused by ventricular hypertrophy, the extra volume of blood thrown into the vessels or an accommodative dilatation of the peripheral vessels (Stewart). Pulsation may also be seen in the retina, either spontaneously or by pressure on the eyeball, as occurs also in Basedow's disease. The liver may pulsate, though this is rare because a strong heart action with stasis is necessary. The liver also pulsates in cases of tricuspid incompetency, but this throb is due to venous filling of the organ during the systole. Pulsation also may occur in the spleen, palate and penis. (*f*) The *capillary* (*Quincke's*) *pulse* occurs in over 80 per cent. of the cases, but is not pathognomonic, for it is also seen in health, in fevers or anæmias. It is a symptom of overfilling of the arteries and capillary dilatation and is best observed when the

hypertrophy exceeds the dilatation; it disappears when the heart weakens. It is elicited by exerting slight pressure on the tips of the finger nails, on the lips by a glass slide or by rubbing the forehead until it is red; there is a systolic reddening and a diastolic paling in these parts. It is sometimes found in the retina, in the palate or in cases of complicating erysipelas. (*g*) The passing over of the blood from the arterioles into the veins may produce the *centripetal venous pulse,*—most obviously in the forearm, and caused by relaxation of the vessels, as in fevers, some nervous lesions and chlorosis; by delicate skin; and full heart action. (*h*) The *arteries are actually lengthened.* (*i*) Patients often assume the dorsal decubitus, for gravity increases the leakage; subjects with mitral insufficiency usually sit. The findings by inspection are practically pathognomonic.

2. PALPATION.—(*a*) The hand corroborates the results of inspection,—as the heaving, *throbbing apex* and the diastolic impulse. (*b*) *A diastolic thrill* over the base is infrequent, occurring mostly in cases of endocarditic origin, but when present is usually proportionate

FIG. 21.



Sphygmogram of case of uncomplicated aortic leakage.

to the degree of leakage. (*c*) In the jugulum, especially in arteriosclerotics, the widened aorta can be felt. (*d*) The *pulse* findings may seem paradoxical, but it must be noted that the arteries are filled violently (the pulsus durus), though the tone of the vessel wall is less than normal.

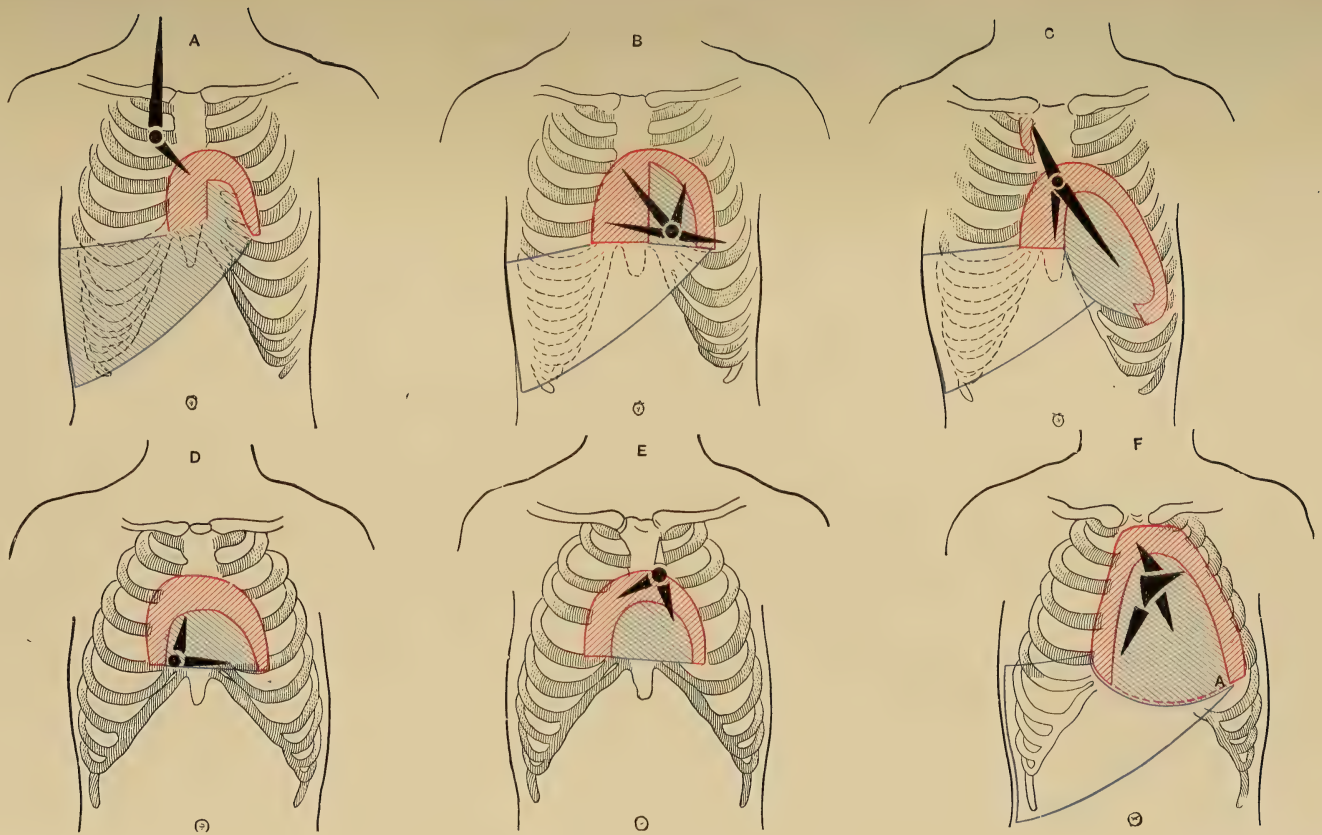
The radial artery is suddenly filled, the "pistol pulse" or *pulsus celer;* this is due to hypertrophy of the left ventricle. The condition of the heart muscle and therefore of the pulse, depends largely on the type or etiology of the case; hypertrophy is usual and marked in the endocarditic form, though in the atheromatous type coronary atheroma and fibrous myocarditis lessen the myocardial activity, render the pulse atypical and make the prognosis less favorable. The sphygmographic tracing shows the high up-stroke with an acute apex (the *pulsus altus*). The dicrotic wave is ill-marked and the pulse rapidly recedes (the "*collapsing pulse*"), due to the inability of the aortic valves to hold the receding blood. (Stewart's explanation is that there is a reflex dilatation of the peripheral vessels and heart stimulation by the increased pressure within the left ventricle.) The falling away of the pulse is sometimes increased by lifting the

arm. The pulse is not absolutely characteristic, because it also occurs in fevers or anæmias, but it is strongly suggestive. The typical findings are altered when atheroma of the aorta or vessels occurs; when other valvular lesions develop, as aortic stenosis or mitral regurgitation; or when the heart muscle is weak or degenerated. When the pulse findings precede the murmur it is claimed that the lesion is due to disease of the aorta. Thrills may be felt over various peripheral vessels. The *peripheral pulse is delayed* in about half of the cases, because the regurgitation continues into the first part of the systole.

3. Percussion.—(*a*) The heart area is *oval* and is increased downward and *to the left,* as could well be anticipated from the mechanism. Dulness reaches the second or third rib to the left of the sternum (see Plate X, Fig. C). The heart is somewhat more horizontal than normal. The dulness extends slightly beyond the apex and is largely due to dilatation of the left ventricle. Because physical laws demand its equal dilatation in all directions, it is not surprising that the right heart may be somewhat crowded to the right, and the left auricle upward, although dulness to the right of the sternum is infrequent. The papillary muscles are found flattened and even thickened at autopsy, from the pressure of the regurgitant blood. (*b*) A small area of *dulness* in the first and *second right interspaces* denotes mechanical dilatation of the arch, which results from the cardiac hypertrophy.

4. Auscultation.—(*a*) *A diastolic murmur* is heard over or to the left of the sternum at the level of the second or third rib. It is caused by the retrograde blood passing through the incompetent valves into the left ventricle. When heard over the so-called "pulmonic area," it is increased downward along the sternum, for the regurgitant column follows this direction (Plate X, Fig. C). It has a *blowing, deep* and less often sawing, musical or whistling sound (which latter suggests a relative aortic leakage). When slight, it may easily escape recognition, but when well developed it is very characteristic, sounding like the word *"who."* It may best be heard with the bare ear against the chest. It sometimes disappears before death when the heart is weak and the lesion is great, but generally corresponds to the degree of leakage. It rarely reaches the neck or abdomen. Cases of valvular rupture have produced a murmur which could be heard at a distance. (*b*) *A slight systolic murmur* over the valves or aorta is common, but the too frequent diagnosis of aortic stenosis (*q. v.*) is not justified. It is soft in the atheromatous and often harsh in the endocarditic type. Its various explanations are: Rough aorta or valves, physiological stenosis (the rigid valves lying between a wide aorta and a dilated ventricle); vibration of the aorta; or leakage continuing into the beginning of the systole. (*c*) *Weakness or absence of the second aortic tone* is explained by imperfect closure of the valves. As the second tone is normally heard over the carotid artery it should be listened for there, because it is

PLATE X



Showing the Size and Shape of Heart in the various Valvular Lesions and in Effusive Pericarditis, the Location of the Murmurs and the Direction of their Propagation.

The black dots show the location of the murmurs and the black wedges show the direction of propagation, growing thinner as the murmurs grow fainter. Over the heart, the red area designates the relative and the blue area the absolute cardiac dulness. A, aortic stenosis; B, mitral lesions; C, aortic regurgitation (note dulness over aorta); D, tricuspid lesions: E, pulmonary lesions (dark wedges designating pulmonary stenosis and insufficiency, and the light wedge, pulmonary stenosis); F, effusive perica ditis (pyriform dulness; wedges showing where friction is most often heard; A, apex, well within the area of dulness).

removed from the murmur and the second pulmonic tone. If it is present, the usual conclusion is that one or more valves are closing, though probably imperfectly. By lifting the stethoscope a little from the skin over the aortic region, the second sound, which was previously obscured by the bruit, may be heard. (*d*) *The first mitral tone is often weak or lost,* because the papillary muscles are flattened and indurated by the regurgitant column. If the change is pronounced, a mitral systolic murmur may be heard in place of the first tone. (*e*) *An apical presystolic* (*Flint's*) *murmur* is heard in 50 per cent. of the cases, and is due to aortic leakage continued in the systole, or to pressure on the mitral valves, which hinders the flow of blood from the left auricle into the left ventricle. Thayer found it in 60 per cent. of 74 cases, and in less than one-quarter of these was there a mitral stenosis. (*f*) *Arterial tones and murmurs* may result from vibration of the vessel wall; these tones may become murmurs, when there is great difference in tension. The systolic vessel tone is increased by the larger volume of blood and increased ventricular action, and is most plain when the vessels are not sclerotic. It is also found in anæmia. Over the femoral artery, a systolic tone is heard which is convertible into a murmur on pressure. *Traube's double femoral sounds* consist of two spontaneous tones (produced without pressure) which are variously explained, probably most satisfactorily by sudden change in tension. They are heard best when the heart is strong and the vessels soft. They occur in other conditions and other valvular lesions, and may be confused with the double venous (Friedreich's) tones, which are heard over the vein in tricuspid leakage. *Duroziez's double murmur* is analogous, but consists of two murmurs, which may be elicited by slight pressure and are due to the progress and recession of blood in the artery. It also occurs in other conditions.

Diagnosis.—Physical examination by inspection, palpation, percussion and auscultation, in the order named, prevents many errors in the interpretation of murmurs. The cardinal findings are: (*a*) Dilatation and hypertrophy of the left ventricle; (*b*) the *pulsus celer* and *altus;* (*c*) the arterial phenomena; and (*d*) the diastolic murmur.

Comparing the two main types, *endocarditic aortic insufficiency* is generally of rheumatic origin and occurs in younger subjects; precordial prominence and diastolic thrill are more common; the heart muscle hypertrophies and the pulsating vessels are more conspicuous; the pulse is more typically "pistol-like" and collapsing, as the vessels are not sclerosed; the capillary pulse and vessel-tones are more frequent; on auscultation, the diastolic murmur is rather harsher and nearer the valves, Flint's murmur is more common and if a systolic aortic bruit is heard, it is often harsher and coincident aortic stenosis is more frequent.

In the *atheromatous form,* sexual excesses, manual work and alcoholism are more evident and syphilis may be declared by the immobile pupil, lightning pains of tabes, etc. As arteriosclerosis readily

involves the heart, coronary symptoms, myocarditis, dyspnœa and dilatation may stand forth. The dilated aorta is more frequently palpable in the jugulum and allows the heart to sag lower in the chest. The diastolic murmur is more characteristically deep and blowing down along the sternum and the typical vascular phenomena are modified by the rigid arteries and arterioles or are not apparent at all.

Relative aortic regurgitation (see ETIOLOGY) is difficult to differentiate from the valvular; the murmur is not the typical protracted diastolic bruit, but rather a short presystolic or postdiastolic murmur (Rosenbach) which produces a short "huff" after the second aortic tone. This character is said to be almost pathognomonic. The pulse in relative regurgitation is frequently small, tardy and tense, but not quick. Hypertrophy of the left ventricle is not very great. The murmur varies from time to time; it is sometimes short, then longer, sometimes blowing, and again coarse, or it may be difficult to fix as to systole or diastole.

Diastolic murmurs are rarely *anæmic* (*cardiopulmonary or functional*). Clear instances exist, however, of such bruits in the cava and other veins. These murmurs are uneven and are stronger in the beginning of the diastole. *Aortic aneurysm* (*q. v.*) may cause confusion. In *mitral lesions,* the capillaries are poorly filled and the skin is cyanotic; in aortic regurgitation the arterioles are overfilled and the skin is red. In mitral disease, congestion of the lung, brown induration and dilatation of the right heart obtain; these are absent in aortic leakage; a hemorrhagic nephritis takes the place of the analogous brown induration of the lungs of mitral lesions.

Relative healing by vicarious stretching of sound valves, by increasing stenosis and by calcification is possible but infrequent. Mercurials may work a cure in cases due to syphilitic aortitis.

IV. Aortic Stenosis.—Aortic stenosis is the most infrequent of left-heart lesions. In Rhomberg's figures, its frequency as a solitary lesion was 2.5 per cent. It may occur congenitally, in which case the conus arteriosus is often constricted (Dittrich's genuine heart stenosis). It occurs, however, largely in the aged; the valves become interadherent or fibrously thickened from atheroma, or chronic endocarditic calcification or retraction at the valvular insertion obstructs the orifice. Acute endocarditis, with massive vegetations, may produce some stenosis. It is usually associated with aortic incompetency, although aortic double lesions are far less common than mitral. Stenosis of the aortic orifice is a better term than aortic stenosis.

Mechanism.—The rigid valves narrow the orifice and at the obstruction a systolic murmur originates. The resistance to the onward flow of blood from the left ventricle leads to its hypertrophy. The pulse becomes small and slow because of obstruction.

Physical Signs.—1. INSPECTION.—Some precordial prominence may be seen in yielding chests. The apex beat is sometimes strong, somewhat dislocated to the left, but often no apex is seen, either on

account of coincident emphysema or of diminution of the heart's recoil. The apex sometimes retracts during the systole from lack of tension in the aorta and from lessened systole.

2. Palpation.—(*a*) A most *pronounced systolic thrill* is often felt over the second right interspace, and sometimes is propagated over the heart and into the cervical vessels. (*b*) *The pulse* is infre-

Fig. 22.

Pulse tracing in stenosis of the aortic orifice.

quent (*pulsus rarus*), because the systole is prolonged and the coronary arteries are not well filled; it is small (*p. parvus*) from obstruction to its flow; it is tense (*p. durus*) from hypertrophy of the left ventricle and arteriosclerosis; and it is exceedingly slow (*p. tardus*) with tardy up-and-down stroke and some retardation. A *strong apex beat* and *a tense but small radial pulse* suggest aortic stenosis.

3. Percussion.—This may be wholly negative. Concentric hypertrophy (*i. e.,* hypertrophy, with the left ventricle smaller than normal), is very frequent. Concentric and simple hypertrophy, which are the more common, as the stenosis is purer, naturally give no change on percussion. Dilatation is sometimes present, giving slight dulness to the left, especially with aortic regurgitation, renal contraction or atheroma. In rare instances both hypertrophy and dilatation are absent.

4. Auscultation.—Auscultation reveals (*a*) a *systolic murmur* over the aortic area, which is sometimes soft but more often *long, sawing,* whistling or musical and is transmitted with the blood current into the aorta, the descending aorta in the back and its vessels (Plate X, Fig. A). It is frequently heard over the entire heart and at times is heard at some distance from the chest (Stokes, Ebstein). It is not proportionate to the intensity of the lesion and disappears with broken compensation. (*b*) *The second aortic sound is weak,* from poor filling of the aorta and weak closure of the altered valves, or it is absent when combined with aortic regurgitation; (*c*) when the second aortic sound is absent, the *second mitral tone,* or indeed all the heart tones, are *weak or absent.* (*d*) A systolic tone is heard over the aorta.

Diagnosis.—The lesion may be a favorable sign, because it is sometimes a healing of an aortic regurgitation. Aortic stenosis is a frequent surprise at autopsy, having run a *latent* course. The *cardinal signs* are (*a*) *systolic murmur* with propagation into the large vessels or over the entire heart; (*b*) a *systolic frémissement* over the aortic area; (*c*) the small, tardy, *slow pulse,* in contrast to the strong apex beat. A diagnosis should be reserved unless these signs are present. As the lesion is uncommon, its frequent diagnosis implies *confusion* with (i) *accidental murmurs,* which are variable. (ii) *Acute endo-*

carditic vegetations; these usually give a soft murmur which becomes harsher if actual stenosis ensues. (iii) *Atheroma of the aorta,* or acute aortitis, where the murmur is more localized over the aorta, the second aortic tone is ringing or metallic and the pulse is generally proportionate to the apex beat. Atheroma, the most frequent cause of diagnostic error, is very common, while experienced clinicians see few cases of pure aortic stenosis. (iv) *Aneurysm* (*q. v.*), which is often distinguishable by its pressure symptoms.

V. Mitral Insufficiency.—Mitral insufficiency (incompetency, regurgitation) is the most frequent valvular lesion and is more often of endocarditic than atheromatous origin. It is (*a*) *valvular,* from adhesions between the valves or chordæ and ventricular wall, from vegetations, from thickening with retraction, from valvular aneurysm or perforation, or from shrinkage or rupture of the papillary muscles or chordæ; or it is (*b*) *muscular,* from relative insufficiency due to such relaxation of the ventricular muscle that the valves will not cover the orifice; this form is common in hypertrophy with dilatation, resulting from hard work, atheroma, nephritis, syphilis, alcoholism, myocarditis and adherent pericardium; (*c*) rupture of the mitral cusps is very uncommon. Barié collected 35 cases of valvular rupture, of which 45.7 per cent. were aortic, 45.7 per cent. were mitral and 8.6 per cent. were tricuspid.

Mechanism.—During the systole, a backward flow occurs through the incompetent valves into the left auricle, which, as it also receives blood from the lung veins, first dilates and later hypertrophies as far as its slight musculature can admit. The left ventricle dilates because it receives in the diastole more blood than normal (*i. e.,* the usual inflow plus the already regurgitated blood) and, responsive to the dilatation, it hypertrophies because its work is increased. In aortic lesions, the first and greatest strain is felt in the left ventricle, but mitral lesions, by congesting the lungs, throw the most work on the right heart. The stasis in the left auricle is transmitted to the pulmonary veins, capillaries and arteries, thus leading to increased engorgement and blood pressure in the pulmonary circuit, because (*a*) of this backward pressure, and (*b*) of the onward pressure from the right ventricle, which dilates and then hypertrophies. The lungs are enlarged and engorged, their vessels dilate and may become atheromatous, possibly causing infarcts in the lung and connective tissue development (brown induration). When the final inevitable weakening of the right ventricle occurs from dilatation, *i. e.,* broken compensation, the right auricle is engorged and relative tricuspid leakage follows from dilatation. The venous system is overloaded and this results in stasis and cyanotic induration of the liver and kidneys, and often in œdema.

Physical Signs.—1. Inspection.—(*a*) The apex may be normally located or *displaced to the left*—much dilatation luxating the apex even into the axilla. The heart may then lie lower than normal. A normally strong apex beat in mitral insufficiency is evidence of

hypertrophy. (*b*) Systolic *pulsation in the second left interspace* indicates a wide pulmonary artery from increased tension or stasis, and (*c*) *a diastolic shock* there is due to closure of its valves; (*d*) *precordial prominence* occurs in yielding chests from marked cardiac dilatation; (*e*) *pulsation,* either epigastric or to the right of the sternum, results from dilatation and hypertrophy of the right ventricle. (*f*) The *veins are enlarged* and pulsate during the diastole from stasis, and sometimes during the systole from tricuspid incompetency (*q. v.*).

2. Palpation.—(*a*) The *apex is dislocated to the left;* (*b*) the *pulmonary artery pulsation* is felt; (*c*) *the diastolic shock* of the forcibly closing *pulmonary valves* is very often palpable; by placing one finger over the valves and another over the apex the two shocks alternate; (*d*) *pulsation* is felt to the *right* from right ventricle dilatation; (*e*) a *systolic apical thrill* is often palpable after excitement, or when the patient lies on his left side, but is not pathognomonic. (*f*) In pure cases the *pulse* is about *normal* in *tension,* or becomes lessened with rupture of compensation. Irregularity is not very common and the sphygmographic tracing is not characteristic.

3. Percussion.—(*a*) *Dilatation of the right ventricle* is commonly followed by *hypertrophy,* in compensated cases. Dilatation shows dulness and palpatory resistance to the right of the sternum (see Plate X, Fig. B). Hypertrophy without dilatation is usually found in the early and well compensated lesion. It should be remembered that the right heart bears the stress of this lesion. (*b*) The *left ventricle* usually but not always *dilates* to respond to the increased amount of blood, and *hypertrophies* because of increased work or the raising of arterial pressure by carbon dioxide.

4. Auscultation.—(*a*) The *systolic murmur* is due to abnormal eddies in the left auricle or to the vibrations caused by them in the mitral valves; it most often gives a *blowing* sound, is best heard *over the apex,* which is less covered by the lung, but at times is localized over the pulmonary area or auricle, where it really originates and where it is heard especially in early lesions. It is *transmitted chiefly* to *the left towards* the *left interscapular* region, and more rarely to the aorta, carotids and abdomen (see Plate X, Fig. B); it is at times heard intermittently, in certain postures only, and it may disappear with weakening of the heart. In very rare cases it is entirely absent (latent). (*b*) The *first mitral tone* is usually absent because the valves cannot vibrate; if present it is referable to conduction from the tricuspid valve or to contraction of the ventricle. When obscured by the murmur, it may sometimes be heard by lifting the stethoscope a little from the chest wall. In cases of extreme leakage, the murmur replaces all tones. (*c*) The *second pulmonic sound* is accentuated, "hammerlike," and is a measure and result of the right ventricular hypertrophy (Skoda). It sometimes reaches the vessels of the neck. When the tricuspid valves become relatively insufficient, it usually weakens; accentuation is absent in those rare

cases where the right ventricle does not hypertrophy. The second tone at the base is sometimes split. (*d*) A *systolic murmur* over the pulmonary area is usually propagated from the apex or is due to vibrations in the distended pulmonary artery.

Diagnosis.—The cardinal features are (*a*) the systolic murmur, (*b*) accentuated second pulmonic tone and (*c*) right-heart changes.

Excepting the etiological diagnosis and the clinical evolution, there are no positive criteria by which we can differentiate a *muscular (relative)* from a *valvular insufficiency,* though in the former the murmur is more variable, lessens with digitalis and is not associated with a diastolic stenotic murmur which is common in valvular incompetency. Pure insufficiency is very exceptional, being usually combined, anatomically at least, with some mitral stenosis into which insufficiency may eventually pass. An organic valvular defect is probable when the apex, dislocated to the left, is forcible.

From the *functional,* accidental, hæmic or cardiopulmonary *murmur,* the diagnosis is usually easy. The murmur is inconstant, is often basal, often depends on posture and coughing efforts, is circumscribed, sometimes disappears on pressure, is not typically transmitted, follows fever, anæmia and emphysema, is often associated with the venous hum in the neck, but is never associated with typical mitral changes in the heart tones and right ventricle. The intensity, *timbre* or *frémissement* of the murmur must not alone be depended upon. So-called "functional" murmurs are usually systolic.

In *chlorosis,* we may obtain a hæmic murmur, apparently large heart (because the lungs are poorly expanded), and a displaced apex, since the diaphragm stands high in chlorosis; deep breathing rapidly decreases the heart's dulness.

Pericardial friction (*q. v.*) may simulate any endocardial murmur (Skoda), though little difficulty attends the differentiation.

Fräntzel's general rules are excellent and aid in accuracy: (1) Never make a diagnosis just before death. (2) Remember the great infrequency of right-heart lesions. (3) Avoid complicated diagnoses. (4) Murmurs are subordinate in value to physical findings by inspection, palpation and percussion.

VI. Mitral Stenosis.—Anatomically pure stenosis of the mitral orifice, without associated mitral leakage is rare. In one group, especially in young girls, the stenosis is clearly endocarditic and the valves are irregular, calcareous and deformed. In another group, seen in old subjects of both sexes, the sclerosing process is probably endocarditic and the last group, almost exclusively observed in women, the nature of the lesion is uncertain; the mitral valves are so grown together or the ring so constricted as to well justify the name used by Corrigan, "the button-hole contraction"; the orifice in extreme cases is funnel-shaped.

Mechanism.—The stenosis obstructs the diastolic passage of the blood from the left auricle into the left ventricle, which consequently dilates and later hypertrophies. The enlarged auricle may compress

the adjacent bronchus or recurrent laryngeal nerve. The stasis is propagated backward through the lungs to the right ventricle, whose dilatation and hypertrophy cause the large right heart and the increased pressure in the lesser circulation. The effects on the right heart and lungs are more severe than in mitral insufficiency. The right auricle also dilates when the later venous stasis follows. Thrombi in either auricle may dislodge and produce pulmonary or arterial infarction. The arterial system is naturally filled but poorly.

Physical Signs.—1. INSPECTION.—This reveals (*a*) *precordial distention,* (*b*) *epigastric pulsation* and (*c*) a *diffuse cardiac shock;* (*d*) the *apex beat* may in comparison be *small,* and (*e*) two impulses or shocks may be seen in the second left interspace; one is systolic, from systolic filling of the pulmonary artery, and the other is diastolic, from pulmonary valve closure under high tension. All these findings depend on the right heart hypertrophy and dilatation.

2. PALPATION.—Palpation confirms the above, especially (*a*) the *apex* beat, which is often *weak in comparison* with the diffuse heart impulse, (*b*) the snap of the *second pulmonic sound,* (*c*) the pulsating right heart, and also (*d*) a *presystolic* (more rarely diastolic) *thrill, frémissement cataire,* which is usually limited to the apex, ends with the *snappy first apex-tone,* and is felt sometimes only in the left lateral posture or after exertion. Its mechanism is identical with that of the murmur (*q. v.*). (*e*) The *pulse* is often *irregular* in cases of pure stenosis, because dilatation interferes with the conductivity of the heart's action, and is usually *small* and of *low tension,* because the aorta is poorly filled; its weakness contrasts conspicuously with the active heart impulse. It sometimes seems hard. The pulse is not pathognomonic but merely suggestive; the sphygmograph confirms the palpatory findings.

3. PERCUSSION.—(*a*) The *right ventricle* in the earlier stages may be only hypertrophied, which does not show on percussion. *Dilatation* without hypertrophy exists in few cases fortunately, for the prognosis is then poor. Most commonly hypertrophy and dilatation coëxist, the latter giving increased dulness to the right, as is the case in mitral insufficiency. (*b*) The *left ventricle* in many cases of dominant stenosis shows concentric atrophy, so that at autopsy it looks like a mere appendage to the huge right heart. It is sometimes both hypertrophied and dilated, for which the following reasons have been assigned: Marked concomitant mitral leakage; stasis with increase in the blood of carbon dioxide, which raises the blood pressure; coincident adherent pericardium, nephritis, atheroma or aortic disease, all of which lead to changes in the left ventricle. (*c*) The *auricles* are distended, much of the dulness under or to the right of the sternum being due to the dilated right auricle and that above and to the left, to the left ventricle. (The right conus arteriosus and the pulmonary trunk are found widened at autopsy.) Increased dulness, due apparently to dilatation of the various chambers, is often caused merely by their dislocation by the wide right ventricle,

4. Auscultation.—(*a*) An *apical presystolic murmur* is important. It occurs at the end of the diastole just before the ventricular systole, *i. e.,* during the systole of the hypertrophied auricle which forces the blood through the narrowed ring or rigid adherent valves. Corresponding with the energy of that chamber, it is loudest at the incipiency and ending of the auricular systole. It so closely precedes the ventricular systole that it is often called systolic by physicians and is almost invariably mistaken by students. It is rarely a blowing sound, but usually snoring, *rolling,* sounding like R-R-R, *after which* comes the loud first tone, sounding like P (R-R-R-P). It is poorly propagated as a rule, corresponding to the localized thrill, but may in exceptional cases be heard in the axilla or back. It is most variable, often being absent even with great stenosis and when the heart weakens and before death, whence the importance of Rule 4 of Fräntzel (see Diagnosis of Mitral Insufficiency). It is rarely diastolic. (*b*) *Marked accentuation* of the *second pulmonic sound* is the proof and measure of hypertrophy of the right ventricle from pulmonary congestion. Its absence is a poor prognostic. It usually weakens when relative tricuspid leakage lessens the tension in the lungs. The accentuation rarely reaches the cervical vessels. (*c*) *Splitting or reduplication* of the *second pulmonic sound* is due to difference in tension in the two arterial trunks, the aortic valves closing first and more weakly. The doubling is best heard at the base and is often propagated into the neck or axilla. (*d*) The usually *loud first mitral tone* is explained in various ways; *viz.,* by difference in tension of the valves during the systole and diastole or by a vigorous contraction of the left ventricle upon little blood. It is sometimes heard at a distance of several feet. (*e*) The *second mitral tone* is weak because of poor filling of the aorta or because the right heart comes forward more than usual. This tone may be split at the apex (the "postman's knock"), and may be present early in this lesion; the cause of the splitting is the subject of variant discussion.

Diagnosis.—The cardinal signs are (*a*) the presystolic thrill and bruit; (*b*) the involvement of the right heart; (*c*) the accentuated second pulmonic sound; (*d*) loud first apical tone and (*e*) the small pulse, which is less important.

The thrill and bruit are also found in other conditions, as in aortic insufficiency (Flint's murmur), in which the vascular phenomena and the involvement of the left heart are sufficiently distinctive, though one out of every three cases of aortic leakage is complicated by mitral stenosis. The bruit has also been heard in lesions leading to dilatation of the left ventricle, such as adherent pericardium, whence the rule that the diagnosis of mitral stenosis must be conservative when there is a clear history of pericarditis or where the *left* ventricle is dilated.

When the thrill and murmur are absent, the diagnosis can usually be made by the other cardinal symptoms.

An absence of the murmur indicates a slight lesion; a presystolic murmur argues for a strong left auricle; a diastolic murmur indicates a severe stenosis; when tricuspid leakage intervenes, the murmur of mitral stenosis is either absent or diastolic—never presystolic—because both auricles are atonic. Absence of the murmur with irregularity is indicative of the most serious lesion. The left auricle, in a way, guards the mitral orifice; consequently when the auricle is strong, it may suppress the systolic murmur of a coincident mitral leakage; when the auricle weakens, the systolic murmur appears.

DIFFERENTIAL TABLE OF THE COMMON CHRONIC VALVULAR LESIONS.

	AORTIC INSUFFICIENCY.	AORTIC STENOSIS.	MITRAL INSUFFICIENCY.	MITRAL STENOSIS.
ETIOLOGY.	Arteriosclerosis chiefly.	Often arteriosclerosis.	Endocarditis chiefly.	Ditto.
PULSE.	Pulsus celer. Water-hammer, collapsing.	Pulse small, slow, tense.	No characteristic alteration—often normal.	Small and often irregular.
LEFT VENTRICLE.	Hypertrophied and dilated greatly; heaving apex.	Hypertrophy usually concentric.	Some hypertrophy and dilatation.	No essential or regular change; may be small.
RIGHT VENTRICLE.	No change.	No change.	Hypertrophy and dilatation marked.	Both marked, also in auricles.
MURMUR.	Diastolic; blowing over sternum near second rib; propagated toward apex.	Harsh systolic murmur over aortic area with systolic thrill; propagated into neck.	Systolic at apex, blowing, transmitted into left axilla and behind scapula.	Presystolic (oftener than diastolic), apical, accompanied by presystolic thrill, fairly localized.
FIRST MITRAL TONE.	Usually weak.	Strong usually.	Usually present, perhaps covered by murmur.	Loud and sharp, clearly palpable and following murmur.
SECOND AORTIC TONE.	Weak or absent.	Weak only.	Somewhat weak.	Somewhat weak.
SECOND PULMONARY TONE.	Not accentuated.	Not accentuated.	Loudly accentuated, and often palpable.	Ditto; often split, reduplicated.
PERIPHERAL VESSELS.	Violent pulsation; capillary pulse; arterial tones; skin red.	Absent.	Absent. Skin cyanotic.	Absent. Skin cyanotic.

In conjunction with this table, Plate X should be consulted.

VII. Pulmonary Insufficiency.—Pulmonary insufficiency of which Barié has collected 58 cases, is most uncommon and is often combined with pulmonary stenosis or other rare right-heart anomalies. It is (*a*) *congenital,* resulting from fœtal endocarditis or from supernumerary or rudimentary valves; (*b*) *acquired,* from rheumatism, sepsis, which destroys the valves by ulceration, or from atheroma; or it is very rarely (*c*) *relative* from extreme intrapulmonary blood pressure or from embolism of the pulmonary trunk or its branches.

Mechanism.—The mechanism is analogous to that of aortic insuffi-

ciency except that the right heart suffers instead of the left and the arterial phenomena are manifest in the area of the pulmonary artery.

Physical Signs.—1. Inspection.—The right heart is dilated and hypertrophied, and pulsates under and to the right of the sternum. It may slightly dislocate the apex to the right. A systolic dynamic pulsation may be seen over the dilated pulmonary trunk.

2. Palpation.—This determines the diffuse right-heart impulse in 20 per cent. of cases, and a diastolic thrill over the pulmonic area; the apex beat and radial pulse are normal or weak.

3. Percussion.—Percussion confirms the schematic expectation that the *right ventricle* should be *dilated* because of the backward fall of the regurgitant column of blood and should be *hypertrophied* to maintain the onward flow under this burden. The left ventricle is normal or sometimes atrophic. (See Plate X, Fig. E.)

4. Auscultation.—A *diastolic murmur* is heard, which is propagated down the sternum over the right ventricle. This bruit may be confused with that of aortic leakage, aneurysm, a dislocated aorta, functional venous murmurs, congenital anomalies of the vessels or patent foramen ovale. The *second pulmonic* and the second tricuspid tones are *weak or absent* and are replaced by the murmur. The bruit is but rarely heard in the neck and the normal second arterial tone is heard over the carotids (see Aortic Insufficiency). A systolic murmur, heard in 25 per cent. of cases over the pulmonary trunk, is due to vibrations in its walls or to coincident stenosis of the pulmonary ring. A double tone has been heard over the lung, as well as variation in the respiration during the systole,—a capillary pulse in the pulmonary vessels.

VIII. Pulmonary Stenosis.—The etiology will be considered under diagnosis and under congenital heart lesions.

Mechanism and Physical Signs.—1. Inspection.—Inspection shows precordial bulging over the right heart, a diffuse heart shock from hypertrophy and dilatation of the right heart, and a weak or absent apex beat.

2. Palpation.—Palpation confirms the above and reveals a systolic thrill in the left second interspace. The pulse is weak.

3. Percussion.—Percussion shows an area of increased *dulness* over the *right heart* from dilatation.

4. Auscultation.—On auscultation, a loud and usually whistling or musical *systolic murmur,* caused by the stenosis, is often but not always heard and the first and second pulmonic tones, hence also the second tricuspid, are weak or absent from poor filling of the pulmonary vessel and from valve alteration. (See Plate X, Fig. E.)

Diagnosis.—The most frequent form is (*a*) the *congenital,* which is due to malformation, myocarditis or endocarditis, and is usually associated with kindred lesions (*e. g.,* open ductus Botalli or foramen ovale). The obstruction may be below the valves, from myocarditic scars narrowing the conus arteriosus, in which case the second pulmonic sound is accentuated; or it may be at the valves or in the

trunk of the artery. Congenital cyanosis and acquired pulmonary tuberculosis are common. The murmur may be heard in the neck. (*b*) The *acquired* valvular form is rare. Atheroma or gummata may be causal. The thrill is also, though far less frequently, felt in *accidental murmurs* which are most common over this area because the walls of the artery are so thin and compressible as to vibrate easily. These "functional" murmurs are so common in this locality as well to justify Fräntzel's rule to observe extreme caution in the diagnosis of right-heart lesions in the adult. Functional murmurs may occur (i) in health; (ii) fevers; (iii) anæmia; (iv) as the cardiopulmonary murmur. The *organic murmur* rarely reaches the jugular or cervical vessels. The left heart is frequently atrophic. When the acquired stenosis is beyond the valve in the pulmonary trunk or lungs, the murmur is heard most advantageously along the right border of the sternum or in the back, and the second pulmonic sound is loudly accentuated, from which points a diagnosis occasionally may be made. In atresia of the orifice 80 per cent. of the subjects die in the first year.

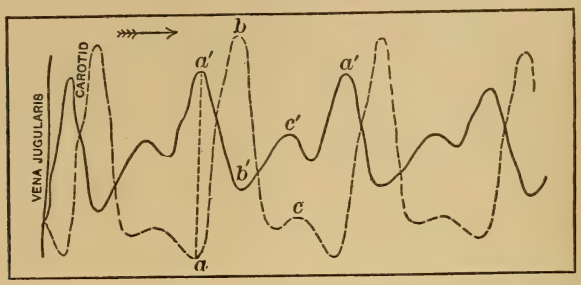
IX. Tricuspid Insufficiency.—Etiology.—(*a*) The *congenital* form is most rare, isolated tricuspid disease occurring in 0.8 per cent. of cases of valvular lesions. The venosity of the blood in the right heart tends to prevent endocarditis, except in fœtal life when the blood is arterial. Myocarditis or ulcerative endocarditis may exceptionally occur. (*b*) Of *acquired* forms, the *relative insufficiency* is very common, resulting from (i) valvular lesions of the left heart, especially mitral stenosis; (ii) obstructive lung lesions, such as induration or emphysema; (iii) possibly abdominal lesions which reflexly contract the pulmonary vessels; and (iv) in severe anæmias (Leube), in which, however, cyanosis and dyspnœa are not observed.

Mechanism.—Whatever the cause of the high pressure in the lesser circulation, it leads to dilatation and hypertrophy of the right ventricle. Extreme dilatation stretches the tricuspid orifice so that the valves cannot cover it, and relative leakage occurs. The leak backward into the right auricle produces its dilatation and hypertrophy, a systolic murmur and a systolic pulse in the veins of the neck and liver.

Physical Signs.—1. Inspection.—(*a*) *A positive* (*systolic*) *pulse in the jugular veins* is absolutely pathognomonic and is due to systolic entrance of blood into the veins from the right ventricle through the insufficient valves. It is presystolic-systolic and is known as positive to differentiate it from the negative or diastolic venous pulse which is observed in many other conditions, and either physiologically, as on deep inspiration, caused by temporary damming back of the blood by the auricular systole, or pathologically, as in stasis. It is seen best on the right side because of the more direct vertical connection with the right innominate vein. A certain degree of heart force is necessary for its production, and it may cease therefore when the heart grows weak. Digitalis may cause it to reappear in cases of relative

and sometimes of organic leakage. It is best seen when the patient is in the dorsal decubitus. To avoid confusion with pulsation transmitted from the carotids, the carotid should be compressed as low down as

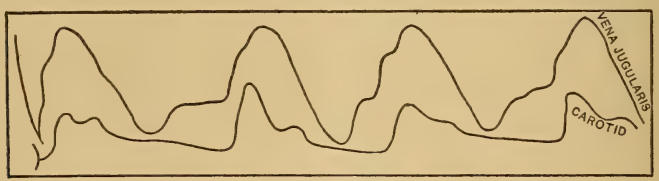
FIG. 23.



Negative (diastolic-presystolic) venous pulse. (After Riegel and Sahli.)

possible, or the pulsating jugular vein should be compressed at the middle; the upper part will then pulsate if the carotids beat against it (transmitted pulsation) and the lower part will beat if the pulsation is in the vein itself. Though it is seen most advantageously in the internal jugular vein, it is sometimes seen in the inferior vena cava or in the veins of the chest and extremities. It is difficult to

FIG. 24.



Positive (presystolic-systolic) venous pulse. (After Riegel.)

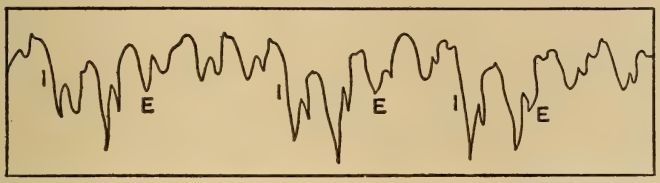
determine when the heart is rapid or irregular. (*b*) A *liver pulse,* systolic in time, venous regurgitant in origin, is also pathognomonic of this disease. (*c*) *The right heart pulsates widely,* while the apex beat is weak.

2. PALPATION.—(*a*) This aids in determining the systolic phase of the venous pulse, and (*b*) it establishes the *systolic venous pulsation of the liver,* which expands in the bimanual examination. It is easier to find, and often earlier to appear than the jugular pulsation. It must not be confused with an arterial liver pulse, which occurs in cases of aortic leakage, exophthalmic goitre and liver inflammation, but is less expansile than throbbing. (*c*) A *systolic thrill* over the tricuspid area; (*d*) tones or thrills over the peripheral veins; and (*e*) a weak apex and radial pulse are also found.

3. PERCUSSION.—This shows increased dulness to the right, from distention of the right auricle and ventricle. Changes in the left heart may indicate an older valvular lesion which causes the tricuspid leakage. (See Plate X, Fig. D.)

4. Auscultation.—(*a*) A faint *systolic murmur* is heard over the tricuspid region; the causal mitral murmur is higher pitched, more superficial and louder. Unlike the murmur caused by mitral lesions,

Fig. 25.



Illustrating the influence of respiration on the positive (presystolic-systolic) venous pulse of the jugular vein. (After Kovacs.)

it is rarely heard in the back but is heard to the right of the sternum. The murmur is absent when the right heart is weak, because it then fails to produce the necessary vibration or eddy. (*b*) The *second pulmonic sound is weak* because the pulmonary circuit is poorly filled. This tone often varies with the changing activity of the right ventricle. It may remain accentuated even with tricuspid leakage when the ventricle is fairly strong. (*c*) Over the *cervical veins* may be heard a systolic tone referable to closure of valves in the veins; the tone may be double, or presystolic-systolic; or venous murmurs may take the place of the single or double tone.

Diagnosis.—An isolated lesion is most rare. The relative insufficiency is diagnosticated by means of (*a*) the etiology, (*b*) the very large right heart, (*c*) the faint systolic murmur, (*d*) the positive venous pulse in the neck, disappearing under digitalis and rest and (*e*) the weak second pulmonic tone. Even when tricuspid incompetency is apparently the only lesion, it should be remembered that rest or digitalis very often brings out a causal mitral lesion. The "safety valve" action of the tricuspid valves is compensatory. Guarding as they do the door between the venous and arterial circulation, their insufficiency indicates most serious cardiac failure.

X. Tricuspid Stenosis.—Tricuspid stenosis is rarer than tricuspid insufficiency; it occurs alone in only 10 per cent. of the cases; it is more often associated with insufficiency, or with aortic or mitral stenosis; the latter is coincident in half the cases. In about half the cases there is a history of rheumatism or chorea; three-quarters of the cases occur in women. Obstruction of this orifice induces hypertrophy and dilatation of the right auricle, and this produces dulness to the right of the sternum. In the single lesion the other heart chambers and the large vessels are small. The pulse is weak. The second pulmonic sound is weak; a diastolic or presystolic murmur and thrill to the right of the sternum are detected if the heart is sufficiently strong, but they are often absent. The lesion is most serious, because it taxes the weak auricle and greatly engorges the venous system. In the jugular vein there is a presystolic wave. The diagnosis is usually impossible. (See Plate X, Fig. D.)

XI. Combined Valvular Lesions.—These occur, clinically, in 33 per cent. of valvular cases. From an anatomical standpoint, a pure lesion is rare, and *double lesions are found oftener than physical signs would indicate.* Pure lesions occur most often on the arterial valves. One lesion may follow another; for instance, stenosis may gradually result from insufficiency, sometimes with good results; or combined lesions may develop simultaneously.

(*a*) *Combined mitral* lesions are most frequent, though one lesion usually predominates; (*b*) *combined mitral and aortic lesions, e. g.,* aortic vegetations reaching the nearest mitral cusp, are next in frequency; (*c*) *double aortic lesions* are less common; (*d*) *combined aortic, mitral and tricuspid lesions, e. g.,* stenosis of each orifice with no signs other than slowing of the circulation, occur in 16 per cent., and (*e*) *combined mitral and tricuspid lesions* (especially relative tricuspid insufficiency) occur still less often (in 9 per cent.).

Diagnosis.—The diagnosis depends upon the character of the pulse, the quality and direction of transmission of the different murmurs, and the hypertrophy and dilatation of the right or left heart. Skoda's rule was that a murmur is usually most intense at its point of origin except in cases of aortic insufficiency; and Oppolzer held that when listening to the mitral and pulmonary valves, murmurs transmitted to them disappear as examination is made more to the left, while murmurs due to lesions on these valves remain; on listening further to the right, the true tricuspid and aortic murmurs remain and those which are transmitted disappear. In diagnosis, the seeking out of the *main lesions* and the avoidance of too complicated diagnoses lead to the most practical results.

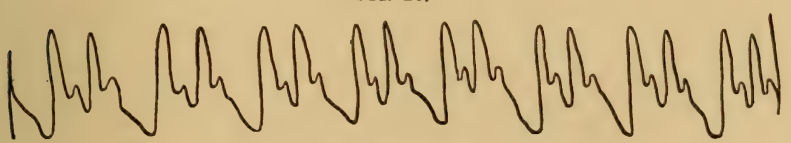
In cases of combined aortic and mitral disease: (*a*) *Aortic insufficiency* and *mitral insufficiency* lead to dilatation and hypertrophy of both ventricles; the combination is frequent in children; the aortic findings as to pulse are frequently modified; the mitral insufficiency may be relative (muscular) or organic (valvular), and this can be determined only by close and continued observation. (*b*) *Aortic stenosis and mitral insufficiency* are next in frequency and are the most common combination in adults; less blood reaches the arteries than in simple aortic stenosis; the right heart suffers disproportionately, whence the severity of this complication. The systolic murmurs usually vary from each other in location, transmission and quality. (*c*) *Aortic insufficiency with mitral stenosis* is a relatively frequent and favorable combination, because the left ventricle is usually less dilated; both may produce presystolic apical murmurs and therefore the state of the right heart and the second pulmonic tone are all-important diagnostically. (*d*) In combined *aortic and mitral stenosis* the greater the aortic stenosis the more is the hypertrophy of the left ventricle; the more dominant the mitral stenosis the less is the hypertrophy of the left ventricle and the greater is the dilatation and hypertrophy of the right ventricle. Differentiation is often difficult.

General Symptoms of Valvular Disease.—The heart possesses not only a certain *reserve power* but in favorable instances its musculature *hypertrophies.* Symptoms may easily be lacking in the early stages, but later compensation to meet the lesion may fail, either temporarily, "disturbed compensation," or permanently, "loss or rupture of compensation." This is the *asystolie* of French writers. Every hypertrophy is more or less temporary in its compensation, and ultimate cardiac failure is inevitable. All lesions, practically speaking, tend to reduce arterial tension, increase venous pressure, retard the capillary flow and promote embolism. Circulatory disturbances may be spontaneous or induced by psychical excitement, physical exertion, intercurrent disease, recurrent endocarditis, malnutrition, excessive use of alcohol or tobacco, or changes in the heart muscle. The symptoms and physical findings are more or less common to all lesions, but some are more frequent in certain forms of valvular disease.

1. Constitutional Symptoms.—Anæmia is frequent (aortic insufficiency); polymorphonuclear leukocytosis is present in half the mitral cases; Andral spoke of a "heart cachexia"; the body temperature is normal or subnormal from slow circulation. Fever is due to recurrent endocarditis, embolism or intercurrent disease. In tricuspid stenosis the red cells may be increased (polycythæmia). The nails are often clubbed in congenital lesions. Skin hemorrhages are due to "blood dissolution" or less frequently to embolism.

2. Cardiac Signs and Symptoms.—Apart from actual change in the chambers, such as dilatation and hypertrophy, the heart itself is not well nourished in "breaking compensation," for it receives less arterial blood and is engorged with venous blood. The *heart muscle* is in some cases fatty, and in others is the seat of myocarditis about the arteries or some cyanotic induration about the veins. These changes are less the cause than the effect of lack of compensation; the autopsy and the microscope may fail to demonstrate the cause of broken compensation. Here and there pressure atrophy occurs, as in the papillary muscles (aortic regurgitation). The *rhythm* is frequently

FIG. 26.



Pulsus bigeminus.

disturbed, especially in mitral lesions (stenosis). The sphygmograph sometimes shows two ventricular contractions occurring very close together (pulsus bigeminus); when the pulse intermits to the finger, the tones of the abortive contraction may be heard over the heart with the stethoscope, the weaker wave failing to reach the periphery. In general, the pulse beats are fewer than the heart

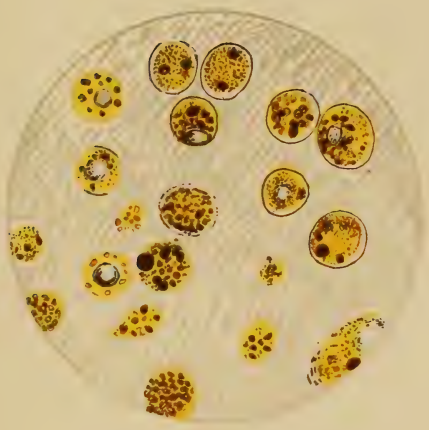
beats. The *gallop-rhythm* is sometimes heard (Bouillaud 1847). In great irregularity tones and murmurs are indistinguishable (*delirium cordis*). The pulse is slow (the *bradycardia* of aortic stenosis) or faster than normal, *tachycardia* (mitral lesions).

Among the complications are pericarditis (aortic lesions) and recurrent endocarditis. *Palpitation* is frequent, either continuously or intermittently, and is sometimes induced by lying on the left side. It may be absent with the greatest heart alteration. A sense of tension over the heart is not infrequent, and *pain* like angina pectoris occurs in aortic oftener than in mitral disease (Nothnagel).

3. Nervous Symptoms.—These are not frequent. Syncope (aortic stenosis especially) occurs from arterial anæmia of the brain. Venous congestion is evidenced by repeated epistaxis, vertigo, tinnitus or *muscæ volitantes*. Brain hemorrhage is infrequent (mostly in aortic insufficiency). Chorea, epileptiform seizures and embolism of the Sylvian arteries are sometimes observed; the psychoses are delirium, mania, melancholia and suicidal tendencies, and are usually of unfavorable import; stasis, inanition, carbon-dioxide narcosis and renal inadequacy are their cause. *Retinitis* hemorrhagica and simple retinal hemorrhages result from stasis and anæmia.

4. Respiratory Symptoms.—Lung stasis is sometimes confused with independent lung disease on account of dyspnœa, cough and expectoration. *Cyanosis* occurs *more often in mitral* than in aortic disease and is most intense in lesions of the pulmonary orifice. *Bronchitis* and *hæmoptysis* are most common in mitral affections. Bronchitis results less from congestion in the lesser circuit than from congestion in the cava superior, into which (and the azygos) the bronchial veins empty. Though usually bilateral, it may predominate on one side from local causes as pleural adhesions. Hæmoptysis results from lung stasis with vessel rupture, from embolism emanating from right-heart clots, thrombosis in the pulmonary and less frequently in the peripheral veins, or from sclerosis in the pulmonary artery. The *sputum* may resemble that of pneumonia, though it is darker, more venous and less viscid. It contains the so-called "heart-disease cells" (E. Wagner). They are large granular delicate cells with oval nuclei, which often contain hæmosiderin or more rarely hæmatoidin; they are probably alveolar epithelial cells and also occur in other lesions than brown induration (as emphysema, asthma, pneumonia and phthisis). (See Plate XI.) *Pulmonary œdema* (*q. v.*) is either (*a*) due to weakening of the left ventricle while the right heart beats more strongly or (*b*) far less frequently is inflammatory. Crepitant rales are frequently heard. In mitral disease the *lungs* frequently become *distended* and rigid from engorgement of the capillaries, and closely resemble emphysema if not identical with it (von Basch's *Lungenschwellung und Lungenstarrheit*). *Brown induration* (cyanotic induration) of the lungs has for its anatomical basis, (*a*) capillary engorgement, often with fatty change in the intima, (*b*) increased inter-alveolar and perivascular connective tissue, (*c*) hem-

PLATE XI



Heart Disease Cells, showing Alveolar Epithelial Cells, Loaded Down with Granules of Hæmatin. (Simon.)

orrhage into the lung from vessel rupture, (*d*) consequent pigmentation by the altered blood-pigment of the lungs and sputum, and (*e*) the "heart-disease cells." *Dyspnœa,* the "cry of distress of the myocardium" (Sansom), constant, intermittent or nocturnal, is caused by unbalanced slow circulation, the bulging of the turgid capillaries into the alveoli, pressure of the heart on the lower left lobe or left bronchus, ascites, hydrothorax, œdema, bronchitis or infarcts. The intimate mechanism of dyspnœa is obscure. F. Kraus has shown that the change of air is the same as in health; he regards the disturbed circulation as at fault; Hofbauer fixes the blame on the decreased elasticity of the lung; François Frank assumes a reflex from the coronaries or aorta which causes a spasm of the pulmonary vessels; Jellinek and Cooper describe a sclerosis of the right coronary artery ("cardiac asthma is a right-sided angina"). It may come on even without exertion when there is constipation or poor digestion; or it may occur especially at night when the voluntary deep and diaphragmatic respiration of the waking hours is not in play, whence the respiratory centre becomes anæmic. The dyspnœa is not expiratory as in asthma but is mixed in type. The gallop-rhythm is rarely absent, and this threatens relapse of the dyspnœa; arteriosclerosis and nephritis are important elements in its production. Exertion not only increases the heart's work but the carbon dioxide and fatigue substances from the muscles irritate the respiratory centre, and the respiratory muscles ultimately become tired. The breathing is more rapid. It may be *Cheyne-Stokes* in type (aortic stenosis); cerebral anæmia, arteriosclerosis and uræmia are its causes. Hypostasis and lobular pneumonia may develop. *Hydrothorax* may hasten death within a few hours (Andral).

5. Gastro-intestinal Changes.—Gastralgia, anorexia, hemorrhagic infiltration and erosion, vomiting or hæmatemesis, result from passive congestion and may be mistaken for independent gastric disease, such as cancer or ulcer. Delayed digestion and constipation are usual. The secretion of hydrochloric acid is normal except in cases of marked weakness of the heart. Intestinal hemorrhage may result from capillary congestion, rarely from embolism of the mesenteric artery, and most infrequently from mesenteric venous thrombosis.

6. Changes in the Liver.—The liver suffers stasis through the congested cava and hepatic vein; this leads to cyanotic distention of the central veins of the liver lobules (appearing dark) and to cloudy and fatty degeneration of their periphery (appearing lighter), the "nutmeg liver." This occurs particularly in mitral disease. The liver is not uniformly affected. Pigmentation occurs and connective tissue develops in the liver and its capsule (perihepatitis, "iced liver"). Small granulations develop on its surface and in some instances a liver shrinkage results from "cyanotic induration" (cardiac cirrhosis). The liver is symmetrically *large* before œdema appears; when disturbed compensation is being restored, it is one of the last symptoms to disappear. Occasionally, from local changes, a tumor-

like enlargement of the liver may simulate cancer. There is a *feeling of tension* and weight in the right hypochondrium and the organ is *tender* and *variable* in size; it may become smaller from rest or digitalis. Its edge is rounded and not hard except in the late stages. (See DIFFERENTIAL TABLE OF HEPATIC DISEASES.) The *icterus viridis* develops gradually and is a combination of mild icterus and cyanosis; icterus is rarely intense but is of the "urobilin" type; it is rarely sudden except in the infrequent embolism, which may cause a clinical picture resembling acute yellow atrophy. *Ascites* is usually late and secondary to the liver congestion; its early appearance is indicative of independent liver disease or peritoneal affections. It is sometimes chyliform.

7. **Changes in the Spleen.**—The spleen is sometimes increased from embolism or liver disease, but is often hard and small.

8. **Changes in the Kidneys.**—The kidneys are enlarged from hyperæmia, the capsule strips readily, and scattered areas of blood extravasation are seen. The *stellulæ Verheynii* are prominent, as also are the glomeruli. Epithelial degeneration follows malnutrition and, in cases of long standing, "cyanotic induration" occurs. Deformity from the stellate scars of healed embolism and fresh infarcts are often encountered.

Renal symptoms are greater, the higher the venous and the lower the arterial blood pressure. The urine is decreased—its daily amount being an accurate gauge of the heart's force; it is of higher specific gravity (1020 to 1030), very acid, and is highly colored from urates and increased uric acid; the urea is decreased and also the chlorides when œdema exists; albumin is frequent, and there may possibly be a few red disks though these are usually due to embolism or acute nephritis. The kidneys are slow to eliminate methyl blue or potassium iodide. Krehl has found granular as well as hyaline casts, contrary to the usual experience. (See DIFFERENTIAL TABLE OF DIFFUSE RENAL DISEASES.) Chronic nephritis may coëxist. Rest and digitalis increase the amount of urine and decrease or remove the albumin. In women, menstruation is frequently increased by passive congestion.

9. **Embolism.**—Embolism occurs as in acute endocarditis but its significance and effects are wholly mechanical. Embolism occurs in about one-third of the cases. Though it is most common in the kidney (77 per cent.) and spleen (54.7 per cent.), it also occurs in the left Sylvian artery of the brain (22.7 per cent.), in the extremities, retina, liver, aorta (with paraplegia) and mesenteric vessels (in 1 per cent.). In the lungs it results from right-heart clots or thrombosis in the large peripheral or pulmonary veins. The special visceral features of embolism will be discussed under Diseases of the Brain, Lung, Kidney, etc.

10. **Œdema.**—This is a result of venous stasis, transmitted to the lymph-vessels and interspaces, and is also an evidence of changes in the vessel walls from actual blood alteration (hydræmia). It ap-

pears first in the connective tissue of the lower extremities (right leg); it disappears over night; and it extends upward to the vulva or scrotum, abdomen, chest and serous sacs (ascites, hydrothorax and hydropericardium) and brain ventricles. The loss of fluid to the system is unfavorable; the anasarca also impedes the arterial circulation, threatens life from involvement of the glottis or lungs, increases the tendency to secondary infections and may lead to hypertrophy of the connective tissue about the genitalia,—cardiac elephantiasis. It is most common in mitral disease. The prognosis is less favorable if the liver becomes very large before œdema appears.

11. Changes in the Joints.—Swollen joints and tabes dorsalis (aortic insufficiency) are occasionally associated with valvular disease.

Course and Prognosis of Valvular Disease.—Patients may live for years with compensated valvular affections; a case of aortic leakage lived for twenty-five years and one of mitral leakage lived for sixty-six years. As a rule the course is chronic and unfavorable, and is influenced by the following conditions:

1. State of the Heart Muscle.—This muscle is the basis of hypertrophy and compensation. Myocarditis is most unfavorable whether associated or not with coronary disease, for digitalis then has less effect on the heart muscle. In cases where digitalis has immediate effect, the prognosis is favorable. The size of the heart is more important than valvular murmurs. Great dilatation or dilatation without hypertrophy is most unfavorable.

2. Sex.—The prognosis is generally better in women, because they are less exposed and less subject to physical strain and coronary disease. Pregnancy as a rule is a rather unfavorable complication. Peter advises, "In girls (with valvular lesions), no marriage; in wives, no pregnancy; in mothers, no nursing."

3. Age.—In the first decade of life the prognosis is poor, even though the heart and coronary vessels are young, because hypertrophy and dilatation are extreme, exercise is difficult to regulate, the valvular lesion tends to recur and the endocarditis tends to develop into pericarditis or pancarditis. After puberty the outlook is better, except, of course, in congenital types.

4. Valve Involved.—Opinions vary widely. In aortic insufficiency, the outlook depends on the type, being fair in the endocarditic and most ominous in the atheromatous type. Clinicians disagree as to the relative outlook in mitral and aortic lesions, although subjects of mitral insufficiency seem to have the more favorable prognosis. Broken compensation is more likely to be restored in mitral insufficiency than in mitral stenosis or in aortic lesions. Mitral stenosis is less favorable, although it is often found in women at an advanced age. Aortic stenosis is also found at an advanced age. Combined or multiple lesions, with some few exceptions, are unfavorable. The writer observed a complete recovery from a double mitral lesion; in eight years after the causal rheumatism the outlines and tones became

perfectly normal. Pulmonary lesions predispose to early tuberculosis.

5. **Mode of Life.**—The social scale, hygiene, necessity of hard work, psychical excitement, alcoholism, exposure and the individual constitution are most important factors.

6. **Unfavorable Symptoms.**—Cardialgia, angina pectoris, embolism, persistent palpitation, dyspnœa and irregular or intermittent pulse are unfavorable symptoms. The "reserve heart power" of Martius is not present in all cases; this is due to failure of the coronary vessels to increase their calibre (Verney).

7. **Favorable Conditions.**—As described by Sir Andrew Clark, these are good general health, just habits of living and absence of special rheumatic tendencies, endocarditic (not atheromatous) origin, no changes in the heart after three years observation, sound ventricles and arteries, and freedom from stasis in the lungs, liver and kidneys.

8. **Sudden Death.**—In most cases, sudden death occurs in aortic affections from coronary disease, but it may also result from cerebral or pulmonary embolism, thrombosis cordis occluding a valvular orifice, heart rupture, sudden ventricular dilatation and glottis œdema.

9. **Intercurrent or Associated Disease.**—Tabes, syphilis and chorea have been mentioned. The acute fevers greatly tax the myocardium. Tuberculosis (*q. v.*) is found in 8 per cent. of autopsies on valvular subjects.

Treatment of Valvular Disease.—(*A*) **In Compensated Lesions.**—In compensated lesions medicinal treatment is unnecessary. With some exceptions, the patient is informed of the nature of his trouble that his coöperation may be enlisted. The *mode of life* is important, *i. e.*, regular orderly living, as free as possible from psychical or sexual excitement, worry or hurry. Especial care should be enforced in aortic cases because of danger of sudden death. Much depends on the *social status* of the patient. The *diet* should be simple but not too schematic. *Tobacco* and *alcohol* should be interdicted as a rule; beer and champagne are poorly tolerated, though some red wine and brandy or cognac are permissible in some adults and in the aged. Coffee may be given in the morning but should not be strong, for cardiac irregularity frequently results. A breakfast of milk may be taken before rising, and the noon meal should be the heartiest of the day. Nitrogenous food is best, with light carbohydrates, as toast; foods which ferment, such as cabbage, beans or brown bread, should be avoided and the amount of fluid ingested should be moderately restricted. Foods which load the stomach heavily are prone to elevate the diaphragm, and every meal somewhat taxes the heart. A light meal at night is the best precaution against gastric and cardiac oppression during the sleeping hours. A milk diet is recommended in cases of stasis, especially when there is much hepatic congestion, but it necessitates the ingestion of too large quantities of fluid.

Exercise.—Exercise in cases not too far advanced is advisable, for the muscles can accommodate all the blood of the body, but their

activity reflexly stimulates the heart muscle. Palpitation, recurrent endocarditis and intercurrent fever necessitate keeping the patient in bed. Moderate *massage* is beneficial, but care must be taken lest it produce dilatation of the abdominal vessels. Deep breathing and singing are beneficial. Plethoric patients should be given an enema of *salines,* which should be allowed to remain in the bowels over night. Tepid are more beneficial than cold or hot baths.

Climate.—High altitudes are to be avoided, although moderate elevation is tonic. The seashore often disagrees with rheumatic or catarrhal subjects. A warm climate should be chosen for the winter.

(*B*). **Broken Compensation.**—Broken compensation may be acute, gradual or terminal, as by coronary occlusion, and is evidenced by dyspnœa which develops on exertion or spontaneously during the night, by anæmia, by irregular pulse (of special coronary significance in aortic cases) and by dilatation. The indications are as follows:

1. Absolute Rest in Bed.—This is frequently curative. Liebermeister's case of severe cardiac disease which recovered while in bed with a broken leg, is an example of compensation restored without digitalis or other drugs. Rest is imperative in intercurrent fevers, bronchitis, recurrent endocarditis and acute cardiac insufficiency. Bed sores can be prevented by the use of water-cushions and by cleanliness. Some patients are obliged to sit up because of dyspnœa. The head may be propped up by back-rests, but they should have side supporters so that falling to sleep will not waken the patient. Rest should be combined with purgation (*v. i.*).

2. Saline Purgation.—Epsom or Glauber salts ℥ss–j in concentrated form should be given in the morning or compound jalap powder 3ss–j at night. These remedies deplete or bleed, as it were, the portal circulation and relieve the heart. Constipation *per se* often increases the size of the left heart.

3. Venesection.—This is necessitated by extreme venous engorgement evidenced by cyanosis, orthopnœa, dilatation or tricuspid leakage. One pint to nearly a quart may be drawn. The effects are often transient but are sometimes wonderful. In two cases apparently dying of valvular disease, where no blood could be obtained from the arms, the external jugular veins were opened, and the patients lived more than a year afterward. Venesection is not to be left until the patient is *in extremis.* It may be necessary to have the patient "actively move the arm so as to bring the blood from the capillaries into the veins" (Liebermeister).

4. The Oertel and Schott Methods.—(See page 366.)

5. Digitalis—Digitalis was first recommended by Wm. Withering (1785) and its physiological action demonstrated by Traube. Its *general indications* are (*a*) *dilatation:* digitalis restores tonicity to the heart-muscle upon which the drug exercises its main influence; (*b*) *functional dissociation:* it is claimed that the ventricles have different innervation and that the drug is most efficacious when the pulmonary and venous circulations are engorged from too weak

systole of the right and too strong systole of the left ventricle. When the *pulse is irregular,* digitalis coördinates the cardiac energy (rhythmicity) by action on the heart muscle and its ganglia or upon the terminal branches of the vagus. (*c*) *Rapid, weak, heart action:* digitalis slows the heart by action on the vagus and by increasing arterial pressure. It tends to abolish abortive cardiac contractions (*v. s.* pulsus bigeminus), and synergizes the systole of the chambers, as is shown by the strong systolic spasm of the heart in cases of digitalis poisoning. The slower and stronger systole empties more completely all the heart-chambers, forces the venous and arterial blood into the main arteries, and squeezes the venous blood out of the heart muscle itself, thereby improving the intrinsic heart circulation. The slower and more complete diastole allows of better filling of the heart and drainage of the engorged veins. Digitalis raises the arterial tension by increasing the working power of the heart and by its centric and local action on the vasomotors.

In the individual lesions: (*a*) in *mitral stenosis,* the longer diastole drains the left auricle and lung vessels and the stronger systole forces the retarded blood through the stenotic orifice; (*b*) in *mitral insufficiency,* the best results are obtained; the leaking cusps are better coapted and the stronger systole fills the aorta and pulmonary artery; (*c*) in *aortic stenosis,* when dilatation occurs or the hypertrophy is inadequate, digitalis is valuable; (*d*) in *aortic regurgitation,* it is theoretically contraindicated since it increases the diastole and hence the leakage; it is, however, valuable in decompensation.

Digitalis is *contra-indicated* (*a*) in balanced compensation; (*b*) in adequate hypertrophy; (*c*) unless rest and purgation have first been tried; (*d*) when the vessels are fragile, as in aneurysm and advanced atheroma (on account of danger, *e. g.,* of brain hemorrhage); (*e*) in fatty degeneration of the heart, except when resulting from stasis and malnutrition; (*f*) in vascular contraction. It is in this case combined with nitroglycerine, strophanthus or potassium iodide, for the purpose of "unlocking the arteries" (Balfour). The drug is not absolutely contra-indicated in the high tension of arteriosclerosis and nephritis, for it may relieve the condition, paradoxical as this may seem, by lessening dyspnœa and carbon-dioxide narcosis and equalizing the circulation. (*g*) In idiosyncrasy to the drug, *e. g.,* when there are gastrointestinal symptoms which, however, are more often due to stasis. Digitalis also causes contraction of all unstriped muscular tissue (gastric, vascular, muscular). It may cause insomnia, delirium, headache, diplopia, blindness to green colors, etc. Digitalis is said to dilate the left and contract the right coronary artery; therefore when the right coronary vessel is diseased, it may dilate and cause excessive contraction of the right ventricle and thus dissociate the heart's action.

Cumulative action is averted (*a*) by intermittent administration, although some patients tolerate digitalis continuously for months or years, as Thomeyer's patient who used digitalis eight and a half

years, taking in all over 15 ounces of the solid digitalis; (*b*) by great care in ambulatory cases; (*c*) by care in cardiac dropsy, on relief of which toxic quantities may be resorbed from the tissues; (*d*) by care in intercurrent febrile affections, for toxic effects often follow the fall of temperature; (*e*) by withdrawal on the appearance of vomiting, syncope, reduced urine or irregular pulse (with twice as many heart as pulse beats); in the latter condition Hewlett combines atropine and digitalis. Twenty-seven cases of pulsus bigeminus from the use of digitalis are reported.

Digitalis may fail to operate unless free purgation, venesection and mechanical removal of the anasarca have been first employed.

Preparations.—Some physicians prefer the powder, gr. j, others the tincture, ♏xv to xx, or the infusion ℥ss–j. The *infusion* is considered more diuretic (containing digitalein and digitonin).

℞ Infusi digitalis ..... ℥iij.
Spts. ætheris nitrosi ..... ℥j.
Potas. bicarbonatis ..... ℥ss.
Aquæ ..... ℥ij.
M. et S.—One tablespoonful after meals.

Larger doses may be given though with risk. It is excellent for rectal use when the stomach is irritable. The *tincture* is thought to be the best cardiant because it contains digitalin and digitoxin. The writer has found digitalin almost inert. *Digitoxin* has seemed of doubtful value, but in the author's hands it has often slowed and strengthened the weak and irregular heart (gr. $\frac{1}{400}$ t. i. d.), when digitalis has failed; it easily disorders the stomach, and often cannot be given for more than six or eight doses. Digitoxin is often advantageously combined with champagne. Cloetta's digalen is a soluble non-irritant digitoxin, and may be given intravenously, 1 c.c. equalling 0.3 mg., or $\frac{1}{180}$ grain. The preparation acts promptly, though it is somewhat irritating when given hypodermatically. For hypodermic use, the fluidextract, ♏ij to iv, is non-irritant.

6. Other Cardiants.—(*a*) *Strophanthus,* ♏v to x of the tincture every eight hours, may be given. *Advantages:* it is more rapid in action than digitalis, has no cumulative effects, perpetuates the effects of digitalis and is indicated in the irregular heart of mitral disease, in children under twelve years, in cases where we are waiting for the digitalis to act, and in dilatation where the blood pressure is high. *Disadvantages:* it is less certain and less energetic; it is inferior in œdema and does not give tone to the vessels (which may be an advantage in atheroma). (*b*) *Strychnine* stimulates the vagus and heart muscle, besides being a splendid vascular tonic (see Pneumonia, Treatment) and tonic to the nervous, gastric and blood-making tissues. Given carelessly, it may irritate the heart, decrease the urine by spasm of the renal vessels, or even induce rupture of the vessels. (*c*) *Sparteinæ sulphas* (gr ¼ to ½ every six or eight hours) is given in capsule because of its bitterness; larger doses may

be given, gr. j to gr. iss; it seems to quiet the nervous system and regulate the heart better than other cardiants (Sée) but is inferior to digitalis. It does not contract the vessels. Convallaria and adonis are very unsatisfactory.

7. Symptomatic Treatment.—(*a*) *Dropsy.*—Saline or hydragogue *catharsis* often relieves moderate œdema. *Digitalis,* alone or combined with calomel or blue mass and squills (Guy's pill) is often efficacious—

℞ Digitalis ..... gr. x.
Massæ hydrargyri ..... gr. vj.
Scillæ ..... gr. x.
M. et ft. capsulæ x.
S.—One after meals for one or two days.

Mercurials, which were recommended by Jendrassik, must be used with the greatest care. The author saw, in consultation, a case complicated by nephritis in which the tongue sloughed off after five grains of calomel had been given. Desperate stomatitis may result in alcoholic, myocarditic or nephritic subjects. Excess of digitalis, it must be recalled, is held to suppress the urine by inducing renal spasm. It is better to use potassium salts, as potassium acetate gr. xxx, t. i. d., which are less abundant in the tissues than the sodium salts; the potassium acetate, *e. g.,* passes through the tissues and kidneys, taking fluid with it (Bunge). Houchard proved that the diuresis following digitalis was associated with increase of sodium chloride elimination. *Caffeine* with digitalis is beneficial in dropsy when the pulse is slow, the mind dull and the digestion sluggish; it may cause nervousness or insomnia (therefore should be given early in the day), irregular pulse (therefore should be combined with rum or brandy), or biliousness; gr. v should be given, but not for more than three days—

℞ Caffeinæ citratæ ..... ʒss.
Sodii benzoatis ..... ʒss.
Aquæ ..... ʒiij.
M. et S.—One hypodermicful (ʒss) as indicated.

Diuretin (daily dosage of 60 to 90 grains) may have marvelous effects or may absolutely fail; chemically it is sodio-salicylate of theobromine; it contains 50 per cent. theobromin and operates on the renal cells directly, and possibly also on the heart and vessels. *Canadian hemp* (fluidextr. apocyni ♏xv) is hard to obtain pure but sometimes justifies Benjamin Rush's term, "a vegetable trochar." *Sweats* and the use of pilocarpine are both dangerous and injurious. Mechanical drainage is recommended, by scarification, by multiple knife or pinpricks, or by Southey's capillary tubes; but the best method is by a single long *deep incision* down to the bone. Sodium chloride should be excluded from the diet. (See Treatment of Chronic Nephritis.) Ascites, from heart disease alone, rarely necessitates puncture; it is indicated chiefly in coincident liver cir-

rhosis or chronic peritonitis. Hydrothorax is far more important, and thoracocentesis should not be delayed when dyspnœa is urgent.

(*b*) *Anæmia.*—The administration of digitalis with iron, arsenic and zinc phosphide often produces remarkable improvement; iron and arsenic may be useful without employing digitalis at all.

℞ Digitalis ..... gr. xx.
Ferri reducti ..... ʒss.
Arseni trioxidi ..... gr. ss.
Zinci phosphidis ..... gr. j.
M. et ft. pilulæ, xx.
S.—One pill after meals; take for a week only.

Cod-liver oil is excellent in children.

(*c*) *Insomnia.*—It is often difficult to relieve insomnia or unrestful sleep, broken by nightmare or sudden "heart starts." Spts. chloroformi, spts. camphoræ and spts. ætheris compos. āā ʒss are often helpful. Sulphonal (gr. xx in hot milk at bed-time) may be tried but is somewhat dangerous in stasis, when used more than three nights in succession. In morphia we place our chief reliance.

(*d*) *Dyspnœa.*—The ice-bag and spiritus glycerylis nitratis ♏j are of value for high tension. *Morphine* may be given in the early stages without hesitation and especially at night, for restlessness and dyspnœa; it is excellent while waiting for digitalis to act. It is almost specific and lessens the irritability of the respiratory centre. It requires care when the heart weakens or Cheyne-Stokes' breathing is present. Dyspnœa is often renal or due to right-sided hydrothorax, chronic bronchitis or excessive dilatation, and appropriate treatment is indicated by catharsis, paracentesis, potassium iodide or digitalis, respectively.

(*e*) *Respiratory Symptoms.*—Cough, cyanosis, pulmonary œdema and hæmoptysis are usually cardiac symptoms and have for their anatomical basis pulmonary engorgement or brown induration; they are relieved by the cardiants. Balfour recommends the following combination for associated bronchial catarrh:

℞ Ammonii carbonatis ..... ʒj.
Tr. hyoscyami ..... ʒiv.
Potassii iodidi ..... ʒj.
Tr. digitalis ..... ʒj.
Infusi calumbæ ..... q.s. ad ʒvj.
M. et S.—One teaspoonful every four hours.

Expectorants usually nauseate. Hæmoptysis is often beneficial; it may be regarded as a species of lung venesection and is seldom urgent or fatal. For cyanosis, alcohol and camphor are indicated. Pulmonary œdema necessitates phlebotomy, a 30-grain dose of lead acetate, gr. $\frac{1}{100}$ of nitroglycerine and gr. $\frac{1}{4}$ of morphine.

(*f*) *Palpitation and Pain.*—For palpitation and throbbing, the ice bag is the best calmative; a precordial blister or a few minims of tr. belladonnæ and tr. aconiti are often helpful.

℞ Fluidextracti belladonnæ ..... gtt. xv.
Tr. digitalis ..... ʒj.
Aq. laurocerasi .....q.s. ad ʒj.
M. et S.—One teaspoonful two or three times daily.

Care regarding the evening meal (see DIET) and the condition of the colon is important. Ten grains of iodide of potash and nitroglycerine (or gr. ss of erythrol tetranitrate which produces a more lasting effect) are valuable for the pain of aortic lesions, especially when accompanied by weakness, pallor, neuralgia, vertigo or headache.

(*g*) *Gastric Symptoms.*—These are troublesome and ominous when the liver is enlarged and there is not much œdema. They are due to portal stasis more often than to a cumulative effect of digitalis, which should then be given by rectum. Small doses of phenol, gr. ¼ every half hour for five doses, and rectal nourishment are indicated.

CONGENITAL HEART-DISEASE.

These cases are infrequent, most complicated and occur largely in the right heart, upon which more work devolves in fœtal life. The causes are (*a*) *developmental errors,* by far the most frequent cause; (*b*) *fœtal endocarditis* is nearly always sclerotic and very rarely verrucose; (*c*) *fœtal myocarditis* or (*d*) *adult endocarditis developing upon developmental anomalies.* They are most often seen in male children, and heredity seems occasionally a factor.

1. Pulmonary Stenosis.—This, by far the most frequent congenital lesion, occurs in various forms; (*a*) stenosis or actual atresia of the orifice itself from endocarditis or myocarditis. In this form the second pulmonic tone is weak or absent. (*b*) Changes in the *conus arteriosus* which obstruct the onward flow of blood. The second pulmonic sound is clearly heard, if the valves are normal. The constriction may make, as it were, a second ventricle. (*c*) The pulmonary vessel beyond the valves is narrowed or converted into an impervious cord; this is a grave type of pulmonary stenosis. The second pulmonic sound is usually heard.

The physical signs are hypertrophy and dilatation of the right ventricle, a thrill over the pulmonic area, and a systolic murmur propagated into the neck when there is a defect in the interventricular septum. These cases are more favorable than those in which such defect does not obtain, because in them the right ventricle is atrophic and the subjects rarely live over a year. In pulmonary stenosis other congenital abnormalities are most common. If the closure or stenosis arises before the end of the second fœtal month, the interventricular septum fails to close in 75 per cent. of cases and is pushed to the left, so that the aorta may partly or entirely originate in the right ventricle. If the closure develops after the second month, the foramen ovale remains patent (6 per cent.). Upon these openings acute endocarditis is prone to develop. The ductus Botalli usually remains open and thereby conducts blood from the aorta to the pulmonary

artery and lungs. Should the ductus Botalli and the pulmonary orifice be closed, collateral circulation is possible through the œsophageal, pericardial and bronchial arteries. The right ventricle usually hypertrophies, especially at the conus, but hypertrophic changes in this chamber are not constant; the left heart is atrophic.

2. Defects of the Interauricular Septum.—The foramen ovale is open to some extent in 44 per cent. of all autopsies, and the membrane is deficient, cribriform or wholly absent. The foramen ovale usually closes in the first week of life, but fails to adhere when low pressure in the left auricle exists. Entire absence of the septum is the most extreme defect possible; this is called the "*reptilian heart*," or *cor biloculare* when the interventricular septum is also absent, or the *cor triloculare* when the ventricular septum is present. Its patency aids compensation in this condition or when the valvular orifices are narrowed or closed or when the great vessels are transposed. Symptoms are absent or are those of the more important associated trouble, for its patency is almost a necessity in some congenital lesions. Rare occurrences are presystolic murmur at the level of the third or fourth ribs; "crossed or paradoxical embolism," which may occur, for instance, in the brain from venous thrombosis in the leg, the clot passing directly from the right to the left auricle; and positive venous pulse in mitral regurgitation. Several cases have been found in living subjects over seventy years of age.

3. Defects of the Interventricular Septum (*Maladie de Roger*). These have been spoken of under pulmonary stenosis. They may be single, associated with other anomalies, or may result from myocarditis. Sometimes a canal-like opening connects the aorta with the right ventricle. The deficit occurs oftenest in the upper "undefended" part of the septum. When it is wholly absent the heart has but three chambers, the "trilocular heart" (*cor triloculare biatriatum*). Symptoms are absent, complicated or ambiguous. At times the signs are those of mitral leakage without the lung symptoms of stasis. A loud systolic murmur is heard over the upper third of the precordium but does not reach the large vessels.

4. Patency of the Ductus Botalli.—The duct is usually closed by proliferation of its coats within the first month of life, because the pulmonary blood-pressure is lowered after birth. Thus premature closure follows pulmonary stenosis, which necessitates poor filling of the lungs with blood. Permanent patency of the duct is favored by other congenital lesions or by atelectasis or pneumonia in the newborn. It leads to increased blood tension in the lung. The second pulmonic sound is accentuated, and the artery is frequently dilated, whence the dulness in the second left interspace, with thrill, pulsation and loud vibratory systolic murmur which may reach the cervical vessels or the fourth dorsal vertebra behind. Sometimes the vessel is so dilated as to compress the recurrent laryngeal nerve and thus cause hoarseness. The right ventricle is hypertrophied and dilated; it is distinguished in clear cases from pulmonary stenosis by the greater

cyanosis of the latter lesion, the less frequent propagation to the neck and the absence of the second pulmonic sound.

5. Persistent Isthmus Aortæ.—The fœtal developmental thrombotic or inflammatory *narrowing* at a point between the left subclavian artery and the ductus Botalli was described by Paris (1789). Maude Abbott finds 198 cases on record. The narrowing is located in 46 per cent. below the duct, 37 per cent. at the duct, and 17 per cent. over it and is characterized (*a*) by hypertrophy of the left ventricle (50 per cent.) to overcome the stenosis, and sometimes by a systolic murmur in the interscapular region; (*b*) by largeness and fulness of the aorta above the isthmus, of arteries of the head, neck and arms (full radials); (*c*) by a small, retarded, abdominal and femoral pulse, corresponding to the anatomical smallness of the vessels below the isthmus; (*d*) by a pronounced collateral circulation between the branches of the aortic arch and those of the thoracic and abdominal aorta, especially the internal mammary, inferior thyroid and transversalis colli arteries. Other anomalies are present in 37 per cent. of cases.

The pathologist makes the diagnosis, as he cuts through the large vessels in the chest and abdomen, and the clinician suspects the lesion when he feels the large vessels (present in 25 per cent. of the cases) and the thrill in them or hears the systolic or less often, diastolic murmur. Valvular lesions, especially aortic insufficiency, may coexist. Broken compensation may intervene, though all cardiac signs may be lacking. Aneurysm and mediastinal tumor are frequently confused with it. The aorta sometimes ruptures. The lesion is nearly twice as common in the new-born as it is in adults.

6. Aortic Atresia or Stenosis.—The lesion is uncommon. The *conus arteriosus* may be greatly constricted. If the lesion is developmental, the interventricular and -auricular septa are open; if endocarditic or myocarditic, they are closed, provided the lesion develops after the third month of fœtal life. The left ventricle hypertrophies in stenosis or atrophies in atresia, while the right ventricle hypertrophies in order to force the blood from the pulmonary artery through the ductus Botalli into the aorta. The lesion may be associated with other cardiac anomalies, with myo- or endocarditis, or with chlorosis, tuberculosis or hæmophilia.

7. Tricuspid Stenosis or Atresia.—This is developmental or inflammatory. In atresia the interventricular and -auricular septa are open and the right heart is atrophic. Tricuspid insufficiency is most rare.

8. Transposition of the Arteries and Veins.—In this lesion the foramen ovale remains open; the aorta originates in the right heart.

9. Valvular Anomalies.—These are largely of anatomical interest. The semilunar valves, especially the pulmonary, may be increased in number from three to five; or they may be decreased to two, which occurs especially in the aortic valves. Whether developmental or inflammatory in origin, accessory or abnormal valves are particularly prone to acute or chronic endocarditis. Mitral anomalies are rare,

10. Anomalies in Location and Development.—These are absence of the heart, *acardia,* double heart or ectopia cordis, where the heart lies in the abdomen, or in the pectoral (with fissured sternum) or cervical regions.

Dextrocardia (dexiocardia) occurs with *situs viscerum inversus;* the mitral valve has three cusps, the tricuspid has but two; the pulmonary veins empty into the right auricle; the pulmonary artery arises from the left, and the aorta from the right, ventricle; the aorta runs to the right of the spine, to the right of the œsophagus and to the left of the cava; the vena azygos is on the left and hemiazygos is on the right; the innominate artery is left-sided; the right lung has two lobes, the left three; the bronchial fremitus is greater on the left side, the left bronchus being larger; the spleen, liver, stomach and colon are transposed; the right kidney and testicle are higher than the left. The heart is rarely transposed alone. In one case complete transposition of all the viscera except the cæcum occurred.

Symptoms of Congenital Heart Disease.—*Cyanosis* is present in 90 per cent. of cases, whence the name "morbus cæruleus"; it is more intense than in acquired lesions. It usually appears early, and is either general or limited to the nose, fingers, etc.; the skin may be actually purple, and this is due to mixing of the arterial and venous currents, to a sluggish circulation in the small veins or to impaired function of the red cells. Sometimes the cyanosis is paroxysmal, with convulsions and dyspnœa; then the anatomy is displaced aorta, septal defect and pulmonary stenosis. Some subjects are anæmic, but a most striking increase of the red cells up to 10 or 12 millions (polycythæmia rubra) is seen in some cases, probably as a compensatory effort. The hæmoglobin may register 230 and the specific gravity 1,077. Dyspnœa, cough, convulsions and marked clubbing of the fingers and toes are frequent. The pulse is variable and the temperature often subnormal. Mental and physical development is incomplete and exophthalmos, swelling of the face, lips and nose and of the optic disk are frequent. Œdema and stasis are not frequent. Coincident maldevelopment occurs (in 10 per cent. of cases) in other organs, such as the stomach, colon, kidney and spleen; imperforate anus, hypospadias, cryptorchismus, polydactylia, hare-lip, spina bifida, acrania, anencephalus and uterus bicornis also occur. The murmurs are usually systolic and rough, although very complicated lesions may exist with normal heart tones. In a few instances the fœtal murmur was heard before birth (Peter). The dulness concerns the right ventricle chiefly.

Prognosis.—The prognosis is poor, for the lesion is incurable and compensation is incomplete. The patient usually succumbs to cardiac failure or to pulmonary tuberculosis (25 to 40 per cent. of cases) in the second decade (81 per cent.), hence Rokitansky's rule, that cyanosis protects the lungs from tuberculosis, is not absolute.

Treatment.—The treatment is wholly symptomatic. The body should be kept warm; baths should be employed to guard against

colds; mental and bodily strain should be avoided; and in general the therapy of adult valvular disease should be followed, except that the heart stimulants must be used with considerable caution.

DISEASES OF THE PERICARDIUM.

PERICARDITIS.

Definition.—An inflammation of the pericardium, secondary to infectious diseases, inflammation in contiguous tissues, other diseases of the pericardium or dyscrasiæ; it is fibrinous or plastic, exudative, or adhesive. Known anatomically to Morgagni, it was first described as a clinical condition by Senac (1749).

Etiology.—The disease is rarely primary, and final analysis usually demonstrates that the so-called primary cases are tuberculous, rheumatic or septic. Cold, exposure and trauma only reduce the physiological resistance of the tissues to bacterial invasion. Pericarditis is mycotic, the organisms of suppuration, pneumonia and tuberculosis being most frequently found; perhaps toxins alone may excite inflammation.

Pericarditis is almost invariably *secondary* (*a*) to *infectious diseases,* of which rheumatism (40 per cent.) is foremost, and it is most frequent when many joints are involved in rapid succession. Pericarditis may be the only manifestation of rheumatism. Many "primary" cases are probably rheumatic. It occurs with recurrent rheumatism, endocarditis and sometimes chorea. It usually develops within half a week to two weeks, but may precede arthritis, especially in children. It is commonly serofibrinous.

It is common in pneumonia, septicopyæmia, puerperal fever, ulcerative endocarditis, osteomyelitis, scarlatina, and more rarely in other exanthemata, and in pulmonary or multiple serous tuberculosis. In children, scarlatina and rheumatism are most important.

(*b*) It is secondary, *by contiguity,* to disease of adjacent organs; to pleurisy, pneumonia and aneurysm; to disease of the ribs, sternum, bronchial glands, spine, œsophagus, stomach, spleen, liver, peritoneum, myocardium and endocardium; and to cervical cellulitis. Trauma of these parts, from without or within, may be included here.

(*c*) It is secondary to *certain diseases of the pericardium,* as tuberculosis, carcinoma and other processes. In 30 per cent. of Matter's "cryptogenetic" cases, tuberculosis was found at autopsy. Scaglios (1904) could find but 8 cases of primary pericardial tuberculosis.

(*d*) It is *secondary to cachexiæ or dyscrasiæ, e. g.,* nephritis (3 to 10 per cent. especially in contracted kidney), gout, diabetes, carcinoma, alcoholism, blood diseases and scurvy; and often, as a terminal infection, to reduced physiological resistance, in which case it may

easily escape clinical recognition. This class is usually found in adults. Though encountered at any age, pericarditis is most frequent between the fifteenth and thirtieth years.

Fibrinous (Plastic) Pericarditis.—A strict division into dry (plastic) and exudative (effusive) pericarditis is impossible, because the forms are usually combined. Dry (plastic, fibrinous) pericarditis is *circumscribed* over the base and large vessels, or is *diffuse,* and consists pathologically of vascular injection, punctate ecchymoses, loss of the smooth, glistening, mirror-like appearance of the serous membrane, and exudation of plastic lymph in small particles or irregular lamellæ, which may appear smeared as with butter, spongy, shaggy or hairy (*cor villosum*). Some fluid is usually found in the meshes of fibrin. Stripping off of the fibrin may reveal tubercles. The subjacent myocardium often suffers infiltration and degeneration in the more intense or protracted cases.

Symptoms.—Symptoms are lacking in most cases; pain is inconstant and when present, is by no means characteristic.

Physical Findings.—Inspection and percussion are usually negative. The writer has seen a strong apex beat where the autopsy revealed a fibrin deposit which was an inch in thickness. Palpation may disclose a friction fremitus, usually over the sternum, which may be obtained as frequently as is friction on *auscultation* (*described under Pericarditis with Effusion, to which refer also for diagnosis, prognosis and treatment*).

Pericarditis with Effusion.—This form has been called the second stage, the fibrinous or dry form being the first stage. The fluid may be *serous* with much or little cellular or fibrinous admixture (serofibrinous); it may be *hemorrhagic,* this type being observed in the aged, in scurvy or cancer, in tuberculosis, purpura, hemorrhagic exanthemata or Bright's disease; the exudation may be tinged with fresh or with darker altered blood; it may be *purulent,* and is usually fibrino-purulent rather than purely purulent, and is often associated with pyæmia or contiguous suppuration; it may be *putrid,* when caused by pyæmia, carcinoma of the œsophagus or stomach, or by lung cavities. Sixty-seven per cent. of pericarditis are sero-fibrinous, 19 per cent. hemorrhagic and 14 per cent. purulent (Breitung). The pathological sequences of pericarditis are *fatty and cloudy degeneration, inflammatory infiltration,* or actual exulceration *of the heart muscle;* rupture of a purulent exudation externally into the tissues of the chest, or possibly into neighboring organs or cavities; inflammatory extension outside of the pericardium (*pericarditis externa*) and thence to other tissues, such as the mediastinum or pleura; formation of polypi, which, becoming detached, may, in rare cases, produce foreign bodies in the pericardium; complete resorption of the exudate and *restitutio ad integrum,* which is a rather uncommon issue; organization of connective tissue, either as local adhesions, which are mostly observed near the fixed base of the heart, because the heart's movements probably detach apical adhesions, or as general

obliteration of the sac (*concretio pericardii*); and, finally, desiccation of the exudate, leaving a cheesy mass which may calcify. Coincident endocarditis is less a sequence than an associate or cause of pericarditis.

Symptoms.—All symptoms are frequently absent (Latham, Graves, Stokes), and no symptom is diagnostic. Leudet found total latency of symptoms in 55 per cent. of his cases. Daily examination of the heart in those diseases which are most likely to cause pericarditis is the only certain method of avoiding diagnostic errors. *Fever* may exist from the disease or from the causal affection, but is irregular (Wunderlich). It is often absent, especially in secondary pericarditis, and the temperature may be subnormal, even in purulent effusions. High fever is infrequent even in rheumatic cases.

Dyspnœa or thoracic oppression occurs in 90 per cent. Oppolzer observed cyanosis without dyspnœa when the pulmonary circuit remained intact. Dyspnœa is due largely to heart compression by the exudate or extension of inflammation to the myocardium, and is less frequently due to vagus irritation, phrenic-nerve inflammation, venous, pulmonary and arterial compression. It is remarkable that patients may sometimes walk into the hospital with enormous pericardial effusions. The respirations are increased.

Pain is variable, is often absent in secondary pericarditis and is said to be more common in small effusions. Sibson found pain in 90 per cent. of his cases. Severe pain oftener indicates pleurisy than pericarditis (Bouillaud). Epigastric pain may precede pain elsewhere (Guéneau de Mussy) and the writer observed initial pain over the appendix. Anginal pain (Andral) radiating to the shoulder, arm and neck is rare. Barlow's patient put on a belt to relieve his pain. Precordial tenderness is at times present, and palpitation is common.

Cerebral Symptoms.—Delirium, anxiety, restlessness, insomnia, mania, convulsions, psychoses or even coma may be due to pericarditis or to complicating uræmia or endocarditis. The symptoms may be suggestive of gastritis, while pericarditis is found on examination. Vomiting, singultus, dysphagia, recurrent laryngeal paralysis and tracheal cough are symptoms of pericardial pressure.

Physical Findings.—The definite diagnosis depends on the physical findings. These are as follows:

1. Inspection.—Precordial prominence, *voussure,* first noted by Louis, may occur when the chest is plastic, as in women and children, and when the intercostal muscles are paretic from inflammatory œdema. The left chest often moves less than the right, since the left lung is usually compressed. The apex-beat is somewhat lower when exudation depresses the diaphragm; it is more horizontal, more to the left and the *dulness transcends the apex to the left.* The greater the effusion, the weaker the apex becomes, and *disappearance of the previously distinct apex beat is all-important.* There may be only an indefinite, diffuse cardiac impulse. On bending forward, the apex

beat or cardiac impulse usually reappears. The interspaces may bulge. Collateral œdema of the chest wall (Rendue) and undulation (Senac) due to the heart itself are most rare. Graves and Stokes described tumor-like extrusion of the compressed lung above the clavicle. According to Ewart, the upper edge of the first rib can be felt as far as its sternal attachment, being separated from the clavicle ("first rib sign"). An epigastric tumor-like bulging of the luxated liver was noted by Auenbrügger and Corvisart. The veins of the thoracic wall are large. During convalescence the *apex may retract* during the systole, from adhesions. The decubitus is usually dorsal or left-sided, rarely right-sided, for this compresses the sound lung; the head and shoulders are usually elevated, and the attitude is fixed and rarely changed. The knee-hand position was noted in Zehetmayer's case.

2. Palpation.—The *friction rub* is often felt. Palpation confirms the location of the apex. *Disappearance of an apex beat previously observed, and the presence of a strong radial pulse, are very important;* mere absence of the apex is observed in many conditions. *Epigastric tenderness* on either side of the ensiform, or tenderness between the attachments of the sterno-mastoid, is due to inflammation of the phrenic nerve (Guéneau de Mussy and Peter). Fluctuation is never felt.

3. Percussion.—Dulness was first described by Auenbrügger. Three to five ounces of fluid are usually necessary to produce distinct physical signs, though Fräntzel has detected the presence of one and one-half ounces. There are two complementary recesses in the pericardium: one over the basal vessels, where the fluid may accumulate, in which case it is early revealed by a triangular dulness, whose apex is directed downward (Skoda, von Dusch, Oppolzer), which may compress the large veins or arteries; and the other in the fifth right intercostal space (Rotch, Ebstein), where the fluid exudate replaces the relative liver dulness, and is most easily recognized by palpatory percussion. Extensive *exudates produce an outline usually* designated as *triangular,* with the blunted apex upward, or as pyriform with the base directed downward. The dulness may even reach the interclavicular notch, the right nipple, or the left axilla, and may impinge on Traube's semilunar space, extend beyond the left lobe of the liver, compress the lungs and markedly depress the diaphragm and liver. The dulness is, at times, trapezoidal rather than triangular. (See Plate X, Fig. F.) Very massive exudates may be mistaken for pericarditis plus pleurisy with effusion. Though the outline is roughly triangular, the right border is more vertical than the oblique *left border,* which *is beyond the apex beat,* if the latter remains visible (Skoda). The apex beat is dislocated abnormally when the patient lies on his side, because the pericardial sac is larger than normal, and permits greater cardiac movement. German writers, following Skoda's teaching, hold that the heart falls back in the fluid, but the heart lies above the bulk of the fluid and

close to the chest wall. The dulness varies with fluctuations in the effusion and with resorption. When the patient is in the erect posture, the dulness may be one-third to one-half as broad again as when he is in the dorsal position; it also is not so high (Gerhardt); this is not invariable, because the lung borders must be normal and the exudate freely movable to allow free shifting of the fluid. Sahli found the same increase in dulness in the sitting posture in enlarged heart and valvular disease. Change of the dulness, especially when the patient lies on the right side (Rosenbach), is important, for this is uncommon physiologically, whereas the shifting of dulness in the left decubitus is of minor value.

Dulness may persist from cardiac dilatation, massive adhesions or pulmonary retraction. The effusion may be enormous (10 quarts, Kyber; or 8 pounds, Corvisart), and may lead to a diagnosis of pleurisy, or pleurisy plus pericarditis. As the normal pericardial sac holds only two-thirds to three-quarters of a quart (650 to 800 c.c.), the pericardium must be greatly relaxed by the inflammation, to allow such large effusions. The pericardial dulness may not be typical when the effusion is largely covered by emphysematous lungs, or when the borders of the lung are adherent and cannot be pushed back by the effusion.

4. Auscultation.—Auscultation reveals the chief and most reliable diagnostic sign, the *pericardial friction* due to attrition of the inflamed pericardial surfaces. It was first fully described by Collin, Laennec's assistant, in 1824. (*a*) It is usually a *"to-and-fro"* rubbing which is not exactly synchronous with either the systole or diastole, which character is most clear when the heart is slow. It is less often single, and then is late in the systole; it may be triple, i. e., presystolic, systolic and diastolic. It is often absent in fibrinous, hemorrhagic or purulent inflammation. In contradistinction to pleurisy, where the effusion of fluid abolishes the primary friction, it usually persists, even with great exudation (Stokes), especially at the base, since here the heart may touch the parietal pericardium. Balfour claims that friction, once heard, is never effaced by even the greatest effusion. A certain consistence of the fibrin seems necessary to friction, for the latter is absent in very soft fibrinous deposits. (*b*) In *quality,* it may be harsh, soft like a gentle and interrupted scratching of the ear with the finger, whistling, creaking, crunching, musical or even metallic (resonance from the stomach or intestines). (*c*) In *location,* it is most often heard over the base or the tricuspid region, where it is especially significant. It may also be heard over the apex. Sears describes a case in which the friction was heard most clearly at the angle of the left scapula. (*d*) It is very *superficial* and close to the ear, not deep as in endocardial murmurs. Superficiality of the friction sound is absent when partial adhesions exist anteriorly, and the pericarditis is most intense behind the heart. (*e*) Its *propagation* is not wide (*il nail et meurt sur place,* Jaccoud), though it has been heard at a distance of nine feet. In children

it may be heard over the whole chest, and the author has once seen the same in an adult. Coincident hydrothorax (Graves) has been thought to favor its propagation, and cardiac hypertrophy, by bringing the heart closer to the chest wall, diffuses the friction more widely (Stokes). The murmur usually ceases abruptly when the stethoscope has been removed a short distance. It does not follow the same lines of propagation as in valvular heart disease. Propagation along the sternum is not uncommon. (*f*) *Inspiration* usually increases the murmur (Traube), due to the closer apposition of the pericardial leaves, caused by the wedge of expanding lung. (Sometimes it is more marked during expiration, particularly when adhesions exist between the heart and lung.) (*g*) *Moderate pressure* usually *increases the friction,* especially in plastic chests. Change of posture *e. g.,* leaning forward, often makes the murmur clearer (Corrigan, Stokes). (*h*) *The murmur* is *variable,* now present, now absent, now systolic, or again systolic and diastolic, varying in acoustic properties, or changing with change of posture; it is often short lived, lasting sometimes but two to six hours. It is not dependent on the intensity of the inflammation, but rather upon the accidental quality of the effused lymph and its location. Disappearance of the rub is explained by regression, adhesions or weak heart. As a rule, the *heart tones* gradually become more or less *weakened* and distant. The *x*-rays outline the pericardial effusion.

Secondary Physical Signs.—There may be evidences of cardiac incompetence, which is due to the mechanical hindrance, by the fluid, of the diastole, especially of the auricles; then the face is pale; it is also due to myocardial degeneration or coincident valvular disease; then the face is cyanotic. The signs of cardiac incompetence are hepatic and renal stasis, ascites, œdema, cyanosis, dyspnœa, cerebral anæmia (syncope), distention and pulsation in the jugular veins and œdema of the lungs. The *pulse* at first undergoes irritative acceleration, but a lasting frequency indicates myocardial change. It may, in exceptional cases, be normal, or even slow (to 36), as a result of compression. There is no characteristic pulse; dicrotism is frequent; there may be the pulsus paradoxus, weakening or intermitting with each inspiration (Traube). A very strong or water-hammer pulse may suggest underlying aortic disease. Irregularity is occasional, as is the pulsus differens and difference in the pupils. *Lung compression* is frequent. Bamberger observed a small area of compression, the size of a dollar, at the angle of the left scapula, where bronchial breathing, dullness (or tympany) and increased fremitus are noticed; the compression clears up when the patient assumes the knee-chest posture (Pins). Pleural puncture excludes the possibility of fluid in doubtful cases, and the absence of crepitant rales excludes pneumonia. Bamberger's sign occurred in cases of hæmopericardium (Allbutt). There may be tympany (or dulness) under the left clavicle and to the left (and sometimes to the right) of the heart in fair-sized effusions. Ewart describes tubular breathing near the right mamma.

Thrombosis of the innominate veins may result from compression. Walshe observed tracheal compression. The second pulmonic sound may be accentuated, which Warthin regards as an early sign. The second tone may be split (Skoda). Metallic heart tones may be due to an adjacent cavity, pneumothorax or the distended stomach and intestines. A systolic non-valvular murmur over the aorta has been explained by pressure on, or inflammatory relaxation of, the vessels, thus interfering with their normal vibration.

Diagnosis.—Differentiation rests upon (1) the *friction* and (2) the *dulness,* or on both, with constant attention to (3) the *apex beat.* Subdivision into types, such as acute or chronic, fibrinous, serofibrinous, seropurulent, purulent, ichorous or hemorrhagic is inadvisable, as puncture alone reveals the character of the exudate. Fibrin formation and serous exudation usually coëxist (Latham).

1. The Pericardial Rub.—*The pericardial rub* is practically *pathognomonic* of pericarditis (von Dusch). (It has occurred on rare occasions in cholera from tissue dryness, in tubercles, cancer, soldier's spots, hemorrhage, hypertrophied heart, and inflammation of the diaphragm or the peritoneum, the heart beating against the inflamed surface.)

(*a*) In distinguishing it from *endocardial murmurs,* the acoustic quality, superficial character, lack of rhythmic precision, variability, slight propagation, palpatory difference in the rub, the basal location and increase of the pericardial friction by pressure or inspiration are most decisive. The distinction is rarely difficult, between an aortic to-and-fro murmur and pericardial friction. Valsalva's experiment—*i. e.,* a long inspiration with closed glottis followed by expiration with tense abdominal muscles—intensifies pericardial friction and decreases endocardial murmurs. The frequency with which pericarditis is a complication or coordinate phenomenon of endocarditis, necessitates reserve in the prognosis, because the valvular disease remains and may first become manifest, after the pericarditis subsides.

(*b*) It must be differentiated from *pleuropericardial friction* (pseudo- or extrapericardial) originating in the inflamed pleura, where friction depends not only on respiration, but also on the heart's action. The usual distinction lies (i) in the close connection of the pericardial rub with the cardiac activity, persisting during expiration, and (ii) in the location of the pleuropericardial friction in the sinus mediastinocostalis (along the lingual lobe), in its intimate relation to respiration and in its usual cessation, either at extreme inspiration or expiration (Stokes, Addison). This method of differentiation is not infallible, for inspiration also augments true pericardial friction, which is, however, increased by bending forward. Valsalva's test (*v. s.*) is also useful. Pericardial friction toward the base or sternum is less often confused with pleural friction. Rosenbach finds differentiation most difficult when the basal vessels come in contact with a tuberculous cavity or pneumonia.

(*c*) Differentiation from *precordial emphysema* is possible on account of the crackling sounds of interstitial emphysema (air) in the anterior mediastinum. The sounds resemble rales, but are closely associated with the heart's action and are often metallic. The condition of the heart tones is decisive.

(*d*) *Pericardial splashing* (see PNEUMOPERICARDIUM).

(*e*) *Crepitant rales* are easily differentiated.

2. THE DULNESS OF PERICARDIAL EXUDATION is typically pyriform or triangular. The markedly increased *relative cardiac dulness* should be considered rather than the *absolute* dulness (which is often little altered in pleural adhesions or pulmonary emphysema).

(*a*) *Hydropericardium* and effusive pericarditis may be confused, because the friction may be absent or disappear, and fever may be lacking (chronic pericarditis). Hydropericardium shows its etiological factors (*q. v.*). It is distinguished by anasarca or hydrothorax which almost always develops first, by lower dulness, by resorption under digitalis and purges, by absence of friction, and paracentesis, which reveals the usual differences between exudates and transudates in their opacity, specific gravity and percentage of albumin (see PLEURISY).

(*b*) *Hæmopericardium,* resulting from trauma, rupture of a coronary artery or ventricular or aneurysmal rupture, is usually sudden, is attended by syncope and blood is removed by puncture.

(*c*) *Pneumopericardium* (*q. v.*). (*d*) From *cardiac dilatation* the differentiation may be most difficult, especially when there is dilatation plus hydrops pericardii, in which case drastics, digitalis, friction, the history and evolution only, may decide. In dilatation the dulness is rarely triangular but rather is increased laterally, the shock-like impulse of the heart is more marked and often contrasts sharply with the weak radial pulse, compression of the lung is much more uncommon, and the heart tones are more clear and "snappy." The very distinctive relation between the apex and the outer dulness to the left is rarely fallible (*v. s.*). If the pulse is strong and the apex weak, pericarditis is present (with, of course, the typical dulness); but compression or weakness of the heart in pericarditis necessitates a weak rapid pulse. In both affections the heart's dulness is increased by leaning forward, but this may clearly demonstrate the apex within the dulness of the exudate. When the patient lies on his right side the apex in pericardial effusion becomes visible (Rosenbach), which does not obtain in cases of enlarged heart. In children the diagnosis of pericardial effusion is often made incorrectly, as dulness oftener signifies dilatation. (*e*) When there is *retraction of the left lung* away from the heart, the diaphragm is high, the apex and outer cardiac dulness coincide, the spleen is high, the pulmonary artery is exposed, and the proper signs of pulmonary retraction are elicited (Leube). (*f*) In cases of *mediastinal tumor* the dulness is irregular; there is no variation of dulness on postural change; the signs of increased conduction and bronchial breathing

are noted with the absence of the cardinal signs of pericarditis. (*g*) From *aneurysm* the differentiation is usually possible, as well as from *marginal infiltration of the lung;* callous pleura, encapsulated pleurisy, redundant fat; and anterior mediastinitis (usually with friction, but with more vertically elongated dulness).

Diagnosis of the Character of the Exudate.—The fluid is serofibrinous in rheumatism; ichorous in perforation from hollow viscera; hemorrhagic in scurvy, carcinoma, tuberculosis, hemorrhagic exanthemata and alcoholism; and purulent in sepsis and empyema. Paracentesis is the only certain test. Tuberculous pericarditis, even in clearly tuberculous cases, can be diagnosticated positively only by detection of tubercle bacilli in the aspirated exudate; a chronic pericarditis is probably tuberculous. In children 30 per cent. of pericarditides are suppurative and 17 per cent. are tuberculous (Baginsky).

Course.—The clinical course is variable; exudation may follow the friction in a short time or only after many days. The exudate may be rapidly absorbed in a few days or may endure for weeks or months. The average duration is one or two weeks. In convalescence the heart may long remain irritable on exertion.

Prognosis.—The prognosis depends largely upon the physiological resistance, the causal disease and the character and quantity of the fluid; it is usually good in rheumatic and serofibrinous cases. Septic, purulent, putrid or hemorrhagic types are serious. The pneumococcic form is fatal in 50 to 75 per cent. Pericarditis is most dangerous in the very young or aged. Death may be rapid in hemorrhagic cases; it usually is due to cardiac exhaustion, especially in chronic cases, and results from myocarditis rather than from simple pressure. The prognosis is more favorable in men than in women and children. Seemingly benign cases may result seriously from *concretio cordis.*

Treatment.—1. The *hygienic treatment* should be managed as in any acute infection with cardiac involvement.

2. *Absolute rest* in bed is indicated, in order to spare the heart, even during convalescence. The patient should never be allowed to sit up or leave the bed, even to urinate or defecate, for severe or fatal syncope may follow the effort.

3. *Laxatives* should be given, for constipation *per se* may increase the heart action, and difficult bowel movements, with the attendant straining, may precipitate syncope. Sod. phosphate ʒss to ij is an excellent laxative.

4. The *diet* should be light or fluid and tea and coffee, which excite the heart, should be avoided. Alcohol may be given when a septic process prevails or when the patient has used it regularly.

5. *Local antiphlogistic measures.* An ample ice-bag upon the precordium quiets and regulates the heart-rate, relieves palpitation and pain and, the author believes, is often a better cardiant and sedative than drugs. Of all drugs, none quiets the heart like opium. Leeches may be applied after drawing some blood by small scratches over

the heart; their action is regulated by applying salt, which causes them to release their hold. A Spanish-fly blister, as large as the hand, often not only relieves pain but stimulates resorption. It must be dressed carefully with weak carbolic salve and gauze.

6. *Causal therapy.* Therapy of the underlying disease is indicated,—such as the use of salicylates, in the rheumatic form.

7. *Heart stimulation* is indicated when the pulse is irregular, fast or small, for this is due to early cardiac irritation or to later mechanical compression of the heart and lungs by the exudate. Alcohol should frequently be given. Strychnia, coffee and camphor operate more quickly than digitalis; they can also be used to follow up its action. Cardiac depressants, such as aconite, are to be carefully avoided.

8. *Treatment of the fever* depends on the basic disease. Hydrotherapy is preferable to the antipyretics.

9. *Compression symptoms,* such as dyspnœa, insomnia and vomiting, when mild in degree or not of long standing, are met by the judicious use of morphine hypodermatically. When the face is pallid, the heart is compressed by the exudate and tapping is indicated. Tardy resorption of large exudates or sudden or severe symptoms therefrom necessitate *tapping of the pericardial sac,* under the general precautions observed in pleural aspiration. The puncture is made in the fifth left (or right) interspace about an inch from the sternum, where the sac is not covered by the pleura. Care and gentleness in passing the trocar enable us to feel the giving way of the parietal pericardium and to avoid cardiac trauma. Removal of but a small quantity frequently provokes absorption by relaxation of the vessels in the tense pericardium, although results are sometimes disappointing. The fluid is withdrawn very gradually by the gravity or siphon method. (See PLEURISY.) Sudden or extreme cardiac embarrassment may necessitate free venesection.

10. *Delayed absorption* is treated by potassium iodide gr. x to xx, by diuretin 60 to 90 gr. *per diem,* and by small doses of calomel followed by salines. Hot packs, alcohol sweats and pilocarpine should be entirely avoided lest collapse occur.

11. *Early pericardiotomy* is indicated in suppurative or putrid forms. About one-half of the cases recover.

Adhesive Pericarditis.—(*Pericardial Concretion* (*Synechia*); *Cicatricial* (*Callous*) *Mediastino-Pericarditis.*)—Though pericarditis may resolve completely, local adhesions within the sac or more extensive bands to the chest wall, pleura, spine or other structures are frequent. They develop in 41 per cent. of cases of pericarditis (Breitung) and are found in 6 per cent. of autopsies (Leudet). In one group of cases, complete obliteration of the sac may occur with absolute clinical latency; the heart may beat itself loose from fresh or lax adhesions, especially when they are located about the apex. In a second group, there are undistinctive evidences of cardiac insuffi-

ciency. In a third series, distinctive physical signs exist. Adhesions most frequently follow serofibrinous pericarditis. A clear history or the actual observation of a previous pericarditis is one of the strongest points in the diagnosis.

Symptoms.—1. Cardiac Insufficiency.—Cardiac insufficiency is caused by pressure on the coronary arteries, by myocardial fibrosis and degeneration, but more often by dilatation or atrophy. While severe heart symptoms without valvular disease in advanced life are indicative of myocardial degeneration, in the young they suggest pericardial adhesions (Wilks). Failure of the right ventricle without valvular, renal or pulmonary disease, or sudden break in compensation in the usual valvular lesions, directs attention to possible pericardial adhesions. Weiss and Pick have called attention to an isolated ascites, associated with large liver and sometimes with fibrous peritonitis; it is probably due to the extension of inflammation along the vessels from the pericardium to the peritoneum; it has been produced experimentally. Sudden death is sometimes due to pericardial synechiæ (Aran).

2. Systolic Retraction of the Interspaces at the Apex.—This may occur even with slight basal adhesions which hinder the systolic descent of the heart, while diffuse adhesions other than basal often produce no symptoms. It is especially significant when the normal systolic protrusion has been gradually replaced after pericarditis by systolic retraction, for retraction of the apex also occurs in other conditions. Systolic retraction is merely an expression of impaired locomotion of the heart and of lack of space at the apex. It is best seen on deep inspiration and disappears when the heart grows weak. Retraction of the apex can be seen and felt.

3. Retraction.—Retraction, not merely of the apex, but of the *sternum, epigastrium* (Oppolzer), *diaphragm* and *lower chest wall* may be observed when adhesions also exist outside of the pericardium in the mediastinum (Skoda); it is indicative of indurative mediastinitis, which attaches the heart anteriorly to the chest wall and posteriorly to the spine, and necessitates retraction until cardiac weakness develops. Broadbent's sign is a systolic retraction of the eleventh and twelfth ribs posteriorly on the left side.

4. The Diastolic Recoil.—Friedreich described a diastolic rebound of the diaphragm and chest wall which were retracted in the systole; it is recognized by palpation and by auscultation as a dull vibration which may cause the second tone to appear doubled.

5. Diastolic Collapse of the Cervical Veins (Skoda).—This is of great diagnostic value, and is due to their aspiration by the heart; it is sometimes dicrotic and may produce diastolic pallor of the face. The cervical veins often swell with the systole.

6. The Pulsus Paradoxus.—The pulsus paradoxus, a pulse intermitting with inspiration, strongly suggests cicatricial mediastinopericarditis, particularly when observed with the signs previously mentioned. It is observed to a moderate extent physiologically and

sometimes in other conditions which lessen the entrance of air into the lungs or render the heart sensitive, especially in myocarditis (Riegel). It is due in mediastinopericarditis to inspiratory stretching of the great vessels by well-attached mediastinal adhesions, which shuts off the blood supply to the extremities. It is a mediastinal symptom, and occurs (*a*) when the arterial system is poorly filled, and hence is more significant when the heart tones are normal

FIG. 27.

Pulsus paradoxus (Kussmaul); E, beginning of expiration, and J, of inspiration.

or stronger than normal, and (*b*) when the return venous flow is retarded. Schreiber holds it pathognomonic when (i) it appears without forced inspiration, (ii) when the radial and all other pulses are completely suspended by inspiration, (iii) when the heart's action is regular and (iv) when it is combined *with inspiratory swelling of the cervical veins* (*v. i.*). Strümpell observed slowing of the pulse during inspiration. This was explained by him as irritation of the vagus by the mediastinal connective tissue.

7. INSPIRATORY SWELLING OF THE NECK VEINS.—Inspiratory swelling of the neck veins (Kussmaul), which normally collapse during inspiration, is due to the same mechanism as the pulsus paradoxus. Inspiration stretches the mediastinal bands, which constrict the large venous trunks and interrupt the return venous flow. Cyanosis may occur with each inspiration.

8. OTHER SIGNS.—The other signs are fixation of the heart by adhesions, which gives no change of dulness by change of posture (von Dusch); absence of respiratory excursion over the heart; decrease of Traube's semilunar space by pleural adhesions (Jaccoud); decreased movement of the left half of the epigastrium (diaphragmatic adhesion to heart); cyanosis and dyspnœa due to adhesions with the diaphragm impeding respiration (Wenckebach); expiratory weakening of the apex by pleural adhesions (Tuczek-Riegel); weak right heart with no accentuation of the second pulmonic tone; loud, even musical, murmurs, especially in the aged; systolic emptying of the veins of the thorax due to systolic dilatation of the internal mammary veins (Broadbent); a rumbling presystolic murmur at the apex, especially in children, without signs of mitral stenosis (Fischer, Hale White); precordial creaking (Perez) when the patient moves the arm; and other mediastinal symptoms, *e. g.*, left recurrent laryngeal paralysis, venous thrombosis in arm, etc.

Diagnosis.—In the diagnosis, two groups of symptoms are observed; first, the myocardial (dilatation, hypertrophy, relative tricuspid insufficiency); and second, the mediastinal (pulsus paradoxus, etc.). (See Pick's PERICARDITIC PSEUDOCIRRHOSIS, under DIAGNOSIS OF HEPATIC CIRRHOSIS.)

Prognosis.—The prognosis depends upon the condition of the myocardium. Cardiac concretion is the usual cause of heart failure in juvenile rheumatic cardiopathies.

Treatment.—The treatment is identical with that of valvular disease (*q. v.*) or of myocardial insufficiency in general. Brauer advocates surgical solution of the adhesions (cardiolysis); about a dozen operations are reported. The heart muscle must, however, be in good condition to warrant surgical interference.

PNEUMOPERICARDIUM.

Gas or air in the pericardium is very infrequent. But 38 cases were collected in 1904 by W. B. James, from whom the figures below were taken.

Etiology.—It results (*a*) from trauma (18 cases), such as perforating wounds; 7 cases resulted from crushing of the ribs or sternum; (*b*) from perforating processes from cavities or viscera containing air or gas (15 cases); pulmonary cavities; gangrene, pneumothorax, œsophageal ulcers or neoplasms; gastric ulcer, liver abscesses or caseated bronchial glands. There is almost always fluid, which may be ichorous, hemorrhagic, purulent (pyopneumopericardium), or more rarely serous (hydropneumopericardium). (*c*) Exceptionally spontaneous gas development without solution of continuity has been observed, due to the Bacillus aërogenes encapsulatus.

Diagnosis.—On *inspection,* there is *precordial bulging* and *absence of the apex beat,* though this may reappear upon leaning forward. On *palpation,* the vocal fremitus about the heart, and the apex beat are gone, friction and occasionally splashing, churning, gurgling sounds may be felt. On *percussion,* when the patient is sitting, a *tympanitic,* cracked pot, or even metallic note is obtained above or indeed over the entire heart, especially when percussion is made with the plessimeter and the hard part of the percussion hammer or the finger-nail. Fluid is usually detected when the patient is in the sitting or lateral decubitus.

The tympanitic note heard above, and the fluid below, is present only when no adhesions are present and varies during the systole and diastole; it becomes higher in the erect posture and varies with the amount of exudation. The change in note on change of posture and the condition of the apex depend upon hydrostatic laws, as is the case in pneumothorax. The lung is compressed. On *auscultation,* the heart *tones* vary with the fluid, being loud if there is no great effusion, and *usually metallic* or musical, like the sound from a zither. The same is true of the quality of the *friction* (for pericarditis is almost invariably present). The tones, the metallic tinkling (in 63 per cent.) or splashing, the falling-drop sound, the mill-wheel churning (in 50 per cent.) and succussion, may be heard at a distance. They may keep the patient awake and may be heard in an adjacent room, as in cases of Stokes, Laennec and Graves.

Differentiation is required from cavities or encapsulated pyopneumothorax near the heart. Consideration of the heart dulness, even though it be dislocated, and of the apex beat renders the diagnosis easy; in James' series all but 6 were recognized. Confusion with distention of the stomach, which imparts a metallic note to the heart tones, is similarly excluded.

Prognosis.—Death occurs in 70 per cent. of the cases and usually results within two weeks, from the inherently fatal causal disease and from cardiac insufficiency. Recovery is most probable in the traumatic cases.

Treatment.—Treatment is the same as in pericarditis. Puncture to void the gas, and operation for putrid or purulent processes, are indicated.

HYDROPERICARDIUM (HYDROPS PERICARDII).

Hydropericardium is always secondary to (*a*) *circulatory disturbances,* such as heart or pulmonary lesions, or, infrequently, local obstruction in the pericardial veins; or (*b*) *cachectic conditions* producing blood alteration and transudation, such as Bright's disease or carcinoma.

Symptoms.—They are much the same as in pericarditis, except that friction is absent. The heart region is sometimes prominent, the apex beat is lost, the heart tones are distant, the fluid changes with change of posture and the dulness is clearly triangular or trapezoidal since no adhesions exist. In most autopsies a small amount of fluid is found which transudes during the death agony or after death. In hydropericardium, the fluid is yellow-green, and is sometimes slightly tinged with blood. Cholesterin crystals are sometimes found. Evidences of inflammation are absent. The fluid is alkaline, contains some albumin, sugar and urea, and is of low specific gravity. Chyle-like effusions (chylopericardium) are very rarely seen.

Diagnosis.—The signs of fluid are the same as in pericarditis (*q. v.* for differentiation), but there is no temperature, tenderness or friction murmur. Dilatation of the heart causes the most frequent diagnostic confusion and is differentiated as in pericarditis. The etiological diagnosis is most important, such as finding a cardiac lesion in the first and renal lesion in the second group.

Prognosis and Treatment.—The prognosis and treatment depend wholly on the cause. Cardiac stimulants, blisters over the heart, laxatives, etc., are indicated.

HÆMOPERICARDIUM.

Blood tinges the exudate in certain forms of pericarditis. A large quantity of blood may escape into the sac in trauma of the heart or of the sac, rupture of the heart (*q. v.*) or of aneurysms (*q. v.*).

The *symptoms* and *physical signs* relate to (*a*) the *acute anæmia,* with pallor, syncope and rapid pulse, which usually ends, in a few

hours or possibly a few days, in death; (*b*) signs of fluid in the pericardial sac. The *prognosis* is favorable only in the traumatic cases; death may be postponed 11 days. The *treatment* is expectant; the heart should be stimulated and salt solution infused.

NEUROSES OF THE HEART.

Palpitation of the Heart.—Palpitation is also known as cardiopalmus, cardiogmus or hyperkinesis cordis, and is purely a symptom. It is closely related to the hyperdiastole caused by vagus stimulation. In health and in many cases of heart disease the patient is not conscious of his heart-action; the most extreme irregularity, or the pounding heart of aortic insufficiency, may escape the patient's notice, while the normal heart action may cause palpitation after overwork, abuse of coffee and other causes.

Etiology.—1. Nervous Causes (*Hyperæsthesia of the Centripetal Fibers from the Heart to the Brain*).—Nervous excitability is common especially in medical students, whose hearts must be examined when they first study physiology or physical diagnosis. Even physicians are not wholly free from heart egotism. Peter Frank had violent palpitation while writing his "Diseases of the Heart," so that he thought he had aneurysm. It is common in school children, from nervous tension, fear of examinations and public exercises; it is more frequent in females at puberty, the climacteric or with menstruation. It occurs constantly in exophthalmic goitre and in 50 per cent. of neurasthenics, so that a neurasthenia cordis has been recognized, which is attended by turgid face, red eyes, contracted temporal arteries and angioneurotic œdema, which symptoms disappear when the seizure is over. Hysteria is a less important cause. Nervous exhaustion, anæmias, acute infections, lactation, poor hygiene, the beginning of tuberculosis and sexual excess or perversion may produce palpitation. The "irritable" heart of soldiers, observed by Da Costa in our Civil War, associated with arrhythmia, diarrhœa, emotional causes and physical strain and often placed under this head, is better classified as muscular insufficiency.

2. Reflex Causes.—This type might be included under nervous palpitation. It follows dyspepsia, especially after a heavy meal, and the products of digestion or mechanical gastric distention have been thought causal. It may also result from constipation, intestinal parasites, gall-stones (Kehr), renal calculi (Potain and Houchard), puncture of ascites, prostatic, uterine or ovarian disease and floating kidney.

3. Toxic Causes.—Palpitation may result from the use of coffee, tea, alcohol, tobacco, narcotics and from gout.

4. Cardiac Causes.—Palpitation may occur in organically incompetent hearts, *viz.*, valvular disease, acute or chronic myocarditis, hypertrophy, dilatation, coronary atheroma and in obesity.

Symptoms.—The patient complains of "feeling the heart beat,"

"goneness," "fluttering," forcible, irregular or intermittent action of the heart, dyspnœa, anxiety, vertigo, tinnitus and spots before the eyes. In some instances the symptoms recur periodically. Intermittent epigastric throbbing is not infrequently present. Vasomotor symptoms, as flushing of the face, pale urine, etc., are not uncommon.

Physical Examination.—Sometimes the heart is absolutely normal. Conceptions of palpitation differ widely, and some authors describe it under tachycardia. The heart's rate is often increased, going up suddenly after exertion or fright to 100 or 150, with violent arterial throbbing. The increased rate may be due to stimulation of the accelerator nerves. The heart is rarely slowed, as in Binswanger's case. The rhythm may be normal or disturbed. A series of rather rapid beats followed by a series of slower beats is possibly somewhat characteristic of nervous palpitation. If the pulse is irregular when the patient is quiet it sometimes becomes regular after exertion. The first apex tone is often accentuated, is even metallic or may be heard by the patient or by the physician at some distance from the patient's chest. In some nervous subjects the systole sounds short (Müller). The second tone at the base is more often strong than weak. A murmur at the base is usually functional.

Diagnosis.—(*a*) The *etiology* is of the greatest importance. It is confirmed by (*b*) *negative results* of examination of the heart during the attacks, but particularly in the intervals. Coronary disease, arteriosclerosis, myocarditis, etc., are usually excluded with ease, but it should always be remembered that nervous palpitation may coexist with organic difficulties. Palpitation is held by some to lead to dilatation or hypertrophy, but this is probably confusion of cause and effect. (*c*) If pain be present in nervous palpitation, it is more often lower (an "*epigastric cramp*") than the pain of organic lesions which radiates to the shoulder or arms.

Prognosis.—Though palpitation is sometimes lifelong, the outlook is generally favorable, but this depends on the etiology. The old belief that fright could cause death is as unfounded as most popular conceptions, and few such instances are recorded (Bollinger, Laache).

Treatment.—Treatment must be *based on the cause.* (*a*) If the cause is *nervous, suggestion* is valuable and repeated examinations are essential, to assure the patient and avoid error. Regulated exercise, tepid baths with active friction, light meals, regular habits and possibly the rest cure of Mitchell are indicated. It seems from personal experience that isolation often increases introspection. In anæmic cases iron, arsenic and strychnine should be given. Goddell's combination of the three valerianates is valuable:

℞ Ferri valeratis.
Quininæ valeratis.
Ammon. valeratis .....āā gr. xx.
M. et ft. in pil. xx.

S.—One pill after meals.

In cases of violent throbbing, a few doses of veratrum often quiet the general and cardiac nervousness. A belladonna plaster or a few drops of the fluidextract helps the patient greatly, probably affecting the peripheral fibers of the vagus. Sexual hygiene must be strictly observed.

℞ Fluidextr. veratri ..... gtt. x.
Fluidextr. belladonnæ ..... gtt. xv.
Aquæ .....q.s. ad ℥j.

M. et S.—One teaspoonful every half hour for two or three doses.

(*b*) When the cause is *reflex,* flatulency and constipation must be treated. Bromides are of value but must be given well diluted to avoid added gastro-intestinal disorder.

℞ Sodii bromidi ..... ℥j.
Spts. ammon. aromat. ..... ℥ss.
Syr. zingiberis ..... ℥iij.
Aquæ .....q.s. ad ℥ij.

M. et S.—One teaspoonful in hot water every half-hour for four doses.

A light evening meal of non-nitrogenous food is beneficial in children afflicted with night terrors or palpitation. (*c*) *The toxic type* is easily treated when the *noxa* is recognized. (*d*) *In valvular disease* (*q. v.*), the cardiants, morphia, belladonna and the ice-bag are indicated. Digitalis is of little use in the truly nervous type.

Tachycardia.—Definition.—Tachycardia is the usual term for rapid heart action, although polycardia and pyknocardia are better etymologically, for tachycardia implies quick (not rapid) heart action like the *pulsus celer.* Tachycardia, like palpitation, is only a symptom. Mackenzie regards it as a series of extra systoles. Some hearts normally beat 90 to 100 (120 in the case of Congreve).

Etiology.—Larcenia's division, somewhat modified, is as follows: (*a*) *Heart and vascular disease;* coronary or valvular affections and acute or chronic myocarditis. (*b*) *Nervous affections;* (i) brain and cord disease, as of the medulla (vagus centre), tumors, softening, hemorrhage, multiple sclerosis, bulbar paralysis, etc.; (ii) neuritis, compression of the trunk of the vagus by tumors, irritation of the accelerator nerve, *neuroses* as exophthalmic goitre, emotional tachycardia and reflex tachycardia from uterine or ovarian disease or the menopause. (*c*) *General diseases* as fevers, chronic troubles (tuberculosis, cancer); and *convalescence, exhaustion* or *excesses.* (*d*) *Toxic* causes; tea, coffee, alcohol, lead, digitalis, atropine, amyl nitrite, etc. Cases are recorded in which individuals by voluntarily moving the ear or skin muscles of the neck can raise the heart's rate, through the spinal accessory nerve and the vagus (Tarchanoff).

A special variety is the tachycardia paroxysmalis, described first by Pröbsting (1881) and later by Nothnagel, Bouveret and Martius. Nothnagel described the affection as a slumbering of the vagus centre. Paroxysms of rapid heart action occur abruptly, last from a few

seconds or minutes to days or even weeks, and usually end abruptly. This type is seen mostly in strong hard-working men, who indulge in excesses in alcohol, tobacco, etc. Broadbent saw a case at ten years of age.

The pulse becomes small, is usually weaker and soft, and its rate reaches 150, 250 (Bowles), or even 280 (Bristowe). It is at times strong (Brieger, Nothnagel) and is usually regular, though arrhythmia may develop at the beginning and end of the seizure. The sphygmogram reveals more beats than does palpation. The heart often dilates (Martius), for the tachycardia and dilatation are coordinate to the causal factor. The pause of the heart (the time between systoles) is lessened (François Frank), the systole is incomplete and the diastole is often shortened. The systolic tone is often clicking. The apex is apparently strong, but throbbing is an index of weakness and dilatation. Embryocardia is frequent (Stokes, Huchard), while heart murmurs are rare. The vessels, especially the carotids, pulsate violently and the patient is anxious, depressed and, late in the course, shows signs of venous stasis or cyanosis, although the face is at times flushed or pale. The urine is of low specific gravity. The lungs are usually normal but Riegel, Tuczek, Edinger and Kredel describe an acute distention of the lungs like that observed in valvular disease, which may be caused by vagus or phrenic irritation or tonic contraction of the diaphragm.

Diagnosis.—Permanent tachycardia suggests organic disease of the centres in the medulla or nerve trunks. The physiology of the vagus and accelerator nerves from the clinical standpoint is still obscure. It has been held that (*a*) increase in the pulse up to 120 indicates sympathetic irritation (accelerator stimulation), which is relieved by morphine; (*b*) increase from 120 to 180 indicates paralysis of the vagus, which is relieved by pressure on the vagus trunk; (*c*) increase over 180 is indicative of sympathetic irritation in addition to paralysis of the vagus. Sympathetic symptoms may prevail, as protrusion of the eyeball, wide pupils and increase in the interpalpebral fissure. Evidences of centric vagus involvement may exist, such as respiratory, laryngeal or gastric crises, polyuria, etc.; sudden onset and cessation suggest a bulbar origin. The *diagnosis* of *vagus neurosis* rests upon (*a*) the exclusion of organic heart and brain disease, (*b*) a high pulse rate, *e. g.,* 150; (*c*) occurrence in paroxysms with intervals of normal pulse rate; (*d*) inhibitive action of manual compression or of electrical stimulation of the vagus.

Prognosis.—The prognosis varies with the cause and type; the permanent variety is less favorable than the paroxysmal and temporary forms. The outlook is generally good. Bouveret holds that permanent cure is rare and has reported four cases of sudden death. The infrequent autopsies (only 16 in 135 cases collected by Hoffmann in 1900) show degeneration of the heart muscle. It is well to regard with doubt and caution all cases of tachycardia other than those resulting from emotional causes.

Treatment.—Stimulation of the vagus is effected by faradization of the neck, holding of the breath, by Valsalva's experiment, by firm bandaging of the abdomen, pressure on the right vagus, the use of the ice-bag, lying down, swallowing ice, or by induction of vomiting by apomorphine. Hoover describes two instances in which inverting the patient brought relief, by depleting the splanchnic circulation or stimulating the medulla by hyperæmia. Morphine, asafœtida, valerian and the bromides are frequently of value. The causal factors must receive consideration.

Bradycardia.—Definition.—Bradycardia, spanocardia or brachycardia is the usual term for *slow heart* but *oligocardia* is a better designation. A *pulsus tardus* is normal to some individuals and in certain families may be seen in successive generations. It is often an accommodative effort as in aortic stenosis. The apparent pulse rate must be verified by cardiac auscultation because the pulse record is often but half as frequent as the actual heart rate. Paroxysmal bradycardia may occur with weak heart, palpitation, difficulty in breathing or gastric distress.

Following Riegel's *classification,* two types are distinguished: (1) *Physiological* bradycardia, which is rather infrequent in occurrence; it is observed in starvation, during the puerperium, etc.; the rate is from 34 to 60. (2) *Pathological bradycardia* may show 6 to 9 beats a minute (Norfleet), and the pulse and breathing may run parallel.

Etiology.—The etiological factors of the pathological type are: (*a*) *Acute infections;* bradycardia is very common during or after typhoid, pneumonia, diphtheria and rheumatism; it is probably caused by acute myocarditis, which induces lessened conductivity or "heart-block," or by a vagus neuritis. (*b*) *Digestive disorders* caused the majority of Riegel's collection; jaundice, constipation, gastric cancer, ulcer, distention or vomiting (which causes bradycardia and arrhythmia in children), and gall stones are the most frequent causes. (*c*) *Respiratory disease* is a less frequent cause; emphysema occurs frequently with a slow strong pulse, which is sometimes an unfavorable symptom. (*d*) *Circulatory disease,* such as aortic stenosis, fatty heart, *coronary atheroma* or chronic myocarditis, is an etiological factor and lessens the conductivity and contractility of the heart muscle. Dehio uses atropine to distinguish between (i) disease of the heart muscle, upon which the drug has but little effect, and (ii) slow heart due to inhibition of the vagus (trunk or centre), in which it brings the heart to or above the normal rate. (*e*) *Toxic conditions* such as poisoning from lead, alcohol, tobacco (coronary spasm), muscarine, digitalis and other drugs may cause bradycardia; it also occurs in cholæmia and early in uræmia, in which cases it is unfavorable. (*f*) *General troubles* are also causative, such as diabetes, cachexia, anæmia or exhaustion, all of which lessen the irritability of the heart muscle. (*g*) *Nervous conditions,* apoplexy,

epilepsy, meningitis, brain tumor, disease or injury of the medulla or cervical cord, vagus stimulation by tumors, compression of the vagus trunk, are factors in its development. Jacob describes an angiospastic form (bradycardia with chills, wide pupils, cold skin, loud second aortic tone, etc.).

The *Adams-Stokes' syndrome* (described under fatty heart) is due to *disturbed conductivity* of the heart action through the *bundle of His*, who in 1903 found a band-like muscular bundle connecting the auricles and ventricles. It measures 18 millimeters in length, 2.5 millimeters in width and 1.5 millimeters in thickness, and courses posteriorly in the septum ventriculorum, whence it reaches the right and left ventricles and their valves. According to Erlanger (1906) slight compression of His' bundle increases the inter-ventricular pause; marked compression causes intermittency in the ventricular contractions; complete compression results in independent contraction of the auricles and ventricles, known as "heart-block." Its *causes* are various; mild, transient or incomplete heart block may occur after infections as typhoid, diphtheria, etc., from the prolonged use of digitalis or possibly from nervous causes. In the more severe, complete and permanent cases, organic changes are discovered in His' bundle. In the 16 published autopsies, gummata were frequent; in Stengel's case, an endocarditic patch affected the bundle; the writer sectioned one case in which marked myofibrosis obtained; acute post diphtheritic myocarditis (A. S. Dunn), acute necrosis or myomalacia cordis (Jellick, Cooper and Ophüls), tumors and changes in the medulla have been observed.

Symptoms.—The affection is recognized by the independent contractions of the auricles and ventricles. With the normal pulse of 70, one-fifth of a second intervenes between the auricular and ventricular systole; if this interval is much increased, there is disturbance of conduction and the transmission of the peristaltic contraction wave from the auricle to the ventricle is "blocked"; the ventricles contract two to four or more times less frequently than the auricles, whose movements may be seen in the pulsating cervical veins, the fluoroscope or in polygraph tracings. The heart beats may be reduced to 2 a minute. When the ventricles do not contract, a systolic tone may be heard due to closure of the auriculo-ventricular valves. The complete syndrome consists of (i) bradycardia; (ii) cerebral disturbance (syncope, apoplectiform or epileptiform seizures); and (iii) accessory symptoms (Cheyne-Stokes' respiration, angina pectoris, etc.).

In "heart-block" the vagus loses control of the ventricle but the accelerator does not. The epileptiform attacks of the syndrone are due to cerebral anæmia and the apoplectiform attacks result from cerebral venous congestion.

Prognosis.—The constant is less favorable than the paroxysmal type.

Treatment.—The causal therapy alone is of value. Morphine and atropine must be given with care where there is disease of the central nervous system. Strong cardiants, such as digitalis, are usually

contra-indicated and in the Adams-Stokes' syndrome are actually dangerous. Alcohol is injurious. In the Adams-Stokes' syndrome, nitroglycerine is indicated, while bromides are deleterious.

Arrhythmia.—There are five major varieties: (1) The respiratory or juvenile; (2) extra systole; (3) perpetual; (4) heart-block; and (5) the alternating pulse.

1. The *juvenile or respiratory* form is especially frequent between 8 and 10 years of age and is found in all children at some time. Normally inspiration increases and expiration decreases the pulse rate but it is pathological when induced by quiet breathing or persists in apnœa. It indicates nervous irritability. Peters maintains that most of the arrhythmias following scarlatina and diphtheria are respiratory in type. It also occurs in brain lesions and vagus disease (whence atropine often controls it).

2. *Extra systole* is the most frequent irregularity and the usual cause of intermittent pulse. Its general causation is an irritable myocardium, which answers to some abnormal stimulus and initiates a "premature," "abortive," "futile" contraction during the diastole. The specific causation; in one group of cases, there is no heart disease and the extra systole is physiological, as in young people or if the subject is nervous, as women at puberty or the menopause or the individual has overdone, mentally or physically; in a second group, the cause lies in poisoning (tobacco, alcohol, coffee, digitalis), in reflex alimentary origin or sometimes in infections where it is ominous; in the third group, there is organic heart disease, or increased arterial tension from atheroma or nephritis. If the extra systole occurs early in the diastole, no change in the pulse is noted, as the aortic valves are not opened and a muffled third sound is heard; if it falls later in the systole, the aortic valves are opened and the pulse shows the extra beat (pulsus bigeminus, trigeminus, polygeminus). The compensatory (diastolic) pause is usually lengthened. It is often recognized by the patient as a thump.

3. In *perpetual* arrhythmia, the pulse is permanently irregular for weeks or months, without tachy- or bradycardia, is unaffected by respiration, atropine or digitalis and is always associated with a ventricular (positive) venous pulse and with no auricular wave in the jugular tracing. There is no underlying rhythm (delirium cordis), and its cause is apparently always intracardial, from serious arteriosclerotic or myocarditic lesions.

4. Heart-block (see bradycardia).

5. The *pulsus alternans* shows in the sphygmogram as alternating high and small waves. It indicates lasting disturbed conductivity and is always serious, occurring in mitral lesions, angina, high blood pressure and cardiac fatigue. If the small wave is retarded, it is the genuine form.

Angina Pectoris.—Angina pectoris (stenocardia, breast-pang) was first described by Heberden and then by Hunter. Edward Jenner

first recognized the connection between angina pectoris and coronary disease. By far the best treatises are Osler's classic on *Angina Pectoris and Allied States,* and Neusser's Heft 2, *Ausgewählte Kapitel der klinischen Symptomatologie.*

Definition.—An arteriosclerotic cardiopathy, characterized (*a*) by paroxysms of excruciating pain over the heart, which radiates to the neck and arms, (*b*) by mental anguish, a sense of dying and (*c*) frequently by sudden death.

Etiology and Pathology.—The chief and practically constant pathological finding is arteriosclerosis involving the heart muscle directly by coronary atheroma, or indirectly by plaques in the aorta at the coronary orifices. The aorta is often atheromatous without coronary involvement and coronary sclerosis is frequent without much change in the aorta.

Though it is usually described under cardiac neuroses, its proper place is under arteriosclerosis. It is notable that atheroma is frequent and angina pectoris is rare. The etiology is that of arteriosclerosis (*q. v.*),—syphilis, gout, alcohol, contracted kidneys, sometimes infections such as rheumatism or influenza. A history of its occurrence through several generations of a family is sometimes elicited. It occurs chiefly in men (80 to 90 per cent. of cases or more), usually after the fortieth year (although cases in the second decennium have been reported) and in the well-to-do classes, professional men, hard workers and luxurious livers. Angina is far more common in private than in hospital practice.

Symptoms.—Mode of Onset.—(*a*) In some cases the *onset is gradual,* and is preceded by dyspnœa (especially when dressing or undressing, lacing the shoes, emptying the bowels, sitting up or beginning to exercise), by short deep respiration, precordial oppression, pulmonary œdema, bronchial catarrh, strong beating in the second or third left interspaces, and difficulty in moving the left arm. This cardiac weakness ("cardiac asthma"), is followed by genuine angina. Heart weakness and angina are coördinate phenomena, secondary to coronary disease. (*b*) In other cases *angina is the first important symptom* of cardiac disease. It is excited by muscular effort, as walking up hill; mental excitement, as anger or coitus; chilling or bathing; flatulent dyspepsia or overloading the stomach, which may induce the attack when the patient is quiet or just going to sleep, by way of the vagus. (*c*) *Death* may be the first symptom.

Paroxysm.—The attack itself consists of (1) *sudden agonizing pain,* which is lancinating and feels like an iron hand squeezing the heart. Its explanation is disputed and nearly one hundred theories have been elaborated. The pain emanates from the cardiac plexus lying behind the aorta, and in the nerve branches from it to the coronary arteries, whence its reflection to other parts, which will be mentioned later. The best explanation, pathologically, is that during the seizure the *heart muscle is ischæmic* from coronary obstruction or possibly from superinduced spasm of the vessel (Allan Burns,

Potain). In man and in the horse, atheroma or thrombosis of the abdominal aorta or its branches to the legs, shuts off the blood necessary during exertion; with moderate exercise the collateral circulation is sufficient, but prolonged exertion produces tingling, cramps and transient paraplegia ("intermittent claudication"). The heart-pang is caused by an analogous cardiac ischæma when unusual stress is thrown on the diseased coronaries. In the case of the author's father, two seizures of angina were followed by "claudication," albuminuric retinitis and the Adams-Stokes' syndrome; the abdominal aorta was clearly to be seen and felt as a hard, uneven rigid tube. The "unutterable" pain is usually *substernal* and *radiates* to the left shoulder, neck and arm. Irradiation may be observed to the chin, ear, jaw, occiput, larynx, and even to the gall-bladder and renal region, spine, stomach, left leg and testis (which may swell). In aortic disease, pain is propagated to the first four dorsal areas; in angina it also reaches the cervical and lower dorsal areas. Pain *along the ulnar nerve* to the fingers may precede the angina; it is usually associated with some motor weakness and, in Eichhorst's remarkable case, with ulnar atrophy. Numbness or hyperæsthesia of the arm, chest or mammary regions is frequent. The anginal pain produces vasomotor symptoms, such as great pallor, low temperature and profuse sweating. The patient may fall, perhaps in a fatal syncope, or more often may support himself for a few seconds or minutes until the paroxysm has passed.

(2) The *mental anguish,* the *angor animi,* which was first recognized by Latham, is the second component of the seizure and consists of a sense of dying, with great anxiety, complete consciousness, immobile attitude and perhaps inability to articulate.

(3) The *danger of sudden death* is the third element. Death may come without pain, in syncope (syncope anginosa) in the first seizure, after subsequent seizures years after the initial paroxysm, or after constantly and closely repeated attacks (the *état angineuse* of Houchard). Osler has called the anterior branch of the coronary artery the "artery of sudden death." Death in angina resembles death from experimental injury to Kronecker's point in the septum. It usually occurs in the second or third attack.

Other Signs and Symptoms.—The *heart is variable,* is sometimes normal in rate and strength, and again shows a tense or weak, slow or rapid or irregular pulse. The left radial may be smaller than the right. A slow pulse usually results from partial "heart-block." Dunin found the arterial pressure low in 45 per cent. and high in 20 per cent. of his cases. Delirium cordis, gallop-rhythm and shortening of the long pause are but signs of cardiac fatigue.

The second aortic tone is often loud and metallic; a rough or blowing systolic (and sometimes a diastolic) murmur is heard over the aorta, which is sometimes dilated; and the left ventricle may be dilated, thus sometimes relieving the attack by relative mitral leakage (Broadbent and Musser). These incidents are simply arteriosclerosis

and its sequences. *Constitutional disturbance* very frequently occurs, as disturbance in nutrition, loss in weight, color and physical endurance. *Dyspnœa* is not a part of angina pectoris, but is solely symptomatic of heart insufficiency. Involuntary evacuations or unsuccessful straining at stool are not frequent. The urine is often pale and abundant after the seizure. Dysphagia, singultus or gastric symptoms are due to sympathetic, phrenic and pneumogastric participation.

Diagnosis.—When the attack is typical and severe, there is little difficulty in diagnosis. Mild or early attacks may be confused with cardiac asthma, because angina may be preceded by cardiac distress, or associated with it. Seizures of *angina involving the abdomen* (angina abdominis) or legs (angina cruris) *are easily mistaken* unless the case is closely watched; these forms may appear first or may alternate with the more usual type—angina pectoris. The classical picture, together with hardness of the arteries and atheroma of the aorta or aortic valves, makes the diagnosis practically certain. The respiration is essentially normal during the attack. Atheroma with cardiac asthma is not angina pectoris.

Pseudo-angina (Latham and Walshe) is the most frequent cause of diagnostic error. Its varieties are (*a*) *nervous pseudo-angina,* which occurs in cases of neurasthenia and hysteria, in other neuroses and in the crises of tabes (which in Oppenheim's case was due to vagus neuritis). Huchard's widely quoted table is substantially as follows:

True Angina	*vs.*	Pseudo-angina.
Most common in men between forty and fifty.		Commonest in women, at any age.
Paroxysms induced by exertion, etc.; diurnal; few in number.		Spontaneous; often nocturnal and periodic; frequent.
Pain intense, of short duration, precordial, inarticulate; immobile attitude; sensation of cardiac compression.		Less severe, lasting hours; epigastric; agitation, activity; sensation of distention.
No nervous symptoms.		Neurasthenic or hysterical stigmata.
Vasomotor form rare.		Common.
Prognosis grave or fatal.		Never fatal.
Lesions; from coronary sclerosis.		From neuralgia.
Arterial medication.		Neuralgic medication successful.

(*b*) *Vasomotor angina* (Nothnagel) is attended by vasomotor spasm, coldness and numbness in the extremities, faintness and great pain in the heart. It may be reflex in origin.

(*c*) The *toxic pseudo-angina* (Beau, Graves and Stokes) is due to coffee, etc., but especially to tobacco. Vasomotor symptoms, amaurosis, dyspepsia, weak, rapid or irregular heart action, palpitation

and anginal pain are common. Functional angina from tobacco, which results, according to Huchard, from coronary spasm, heals when tobacco is withheld; but actual coronary disease, due to tobacco and other heart poisons, is not curable. (*d*) The crises of tabes and precordial pain due to brachial neuritis, vagus involvement or gout, etc., may be difficult to diagnosticate.

Prognosis.—The prognosis is self-evident. Death is frequent. In one instance the patient lived forty years. Few clear recoveries are reported.

Treatment.—The treatment concerns (*a*) *the etiology,* in which arteriosclerosis is the essential factor. Vierordt advises that the possibility of syphilis should always be considered. Gout and diabetes necessitate appropriate diet and therapy, as colchicum. Attention must be directed to the question of stimulants. Aside from syphilis, the iodides are valuable and should be given two-thirds of the time, for months and years. Small amounts are frequently tolerated less well than 10 or 20 grain doses. Care in their use is necessary in nephritis and even when they are well diluted, gastric disorder may result and possibly induce an attack of angina. Nitroglycerine or its derivatives are indicated in the intervals, in doses of gr. $\frac{1}{100}$ to $\frac{1}{50}$ four or more times daily, but it should be remembered that its effects last less than an hour. It may cause headache, flushing or (in larger doses) cyanosis or pallor, muscular relaxation, dyspnœa and irregular pulse. Erythrol tetranitrate in doses of gr. ss. t. i. d. has a more lasting effect than nitroglycerine. These vasodilators will be again considered under Arteriosclerosis. (*b*) *The causes precipitating paroxysms.* Prophylaxis should be maintained regarding exertion, excitement, worry, coitus, chilling, constipation and dyspepsia; late suppers and foods favoring flatulency should be avoided, and a light evening meal should be taken. The patient should be provided with a mixture of aromatics in order to dispel gastric flatulency; a single dose is contained in the following:

℞ Spiritus ammoniæ aromatici ..... ʒss.
Spiritus chloroformi ..... ʒss.
Spiritus glycerylis nitratis ..... gtt. i.
Spiritus ætheris compositi ..... ʒss.
M. et S.—To be taken in hot water.

(*c*) The *paroxysm. Amyl nitrite* was first advised by Brunton (1867); it is an analgesic and a vasodilator; the patient should carry pearls of the drug, which were invented by Solger and contain three minims of the remedy. They often operate powerfully and instantaneously, though sometimes they are useless. It may be better to have the patient carry the spts. glycerylis nitratis with him, for the physician but rarely sees the attack. Some member of the patient's family should be taught how to give a hypodermic injection of morphine and atropine. Morphine, and chloroform by inhalation have been held dangerous, especially when brain trouble exists, as is

sometimes the case in the Adams-Stokes' triad. Nevertheless they must be used in some cases. Yeo called attention to the large doses of morphia required and tolerated by subjects having angina. (*d*) *Cardiac weakness* is treated by diffusive stimulants as ammonia, camphor, alcohol and strychnine. When arterial tension is low, digitalis may be administered between paroxysms:

℞ Tr. digitalis ..... ʒiv.
Tr. strophanthi ..... ʒij.
Tr. valerianæ .....q.s. ad ʒiv.
M. et S.—One teaspoonful after meals.

Vierordt says that morphine should be carried in one hand and stimulants in the other. Coffee is said to be prophylactic against cardiac weakness and the paroxysms, and is said to dilate the coronaries. *Pseudo-angina* is treated according to the type. Electricity and arsenic are valuable.

DISEASES OF THE ARTERIES.

ARTERIOSCLEROSIS.

Synonyms.—Atheroma, arteriocapillary fibrosis (Gull and Sutton), endarteritis deformans (Virchow), angiosclerosis (Thoma).

Definition.—A regressive change, evidenced first by malnutrition in the walls of the large or small vessels and later by compensatory thickening, either localized or diffuse. Morgagni recognized atheroma.

Etiology.—1. Age.—Arteriosclerosis is the essential element in senile involution, although some individuals attain their hundredth year with no arterial change. Cazalis said, "A man is as old as his arteries." Though usually found after the fiftieth year, hereditary influences or certain infections, as syphilis, may develop hard arteries between the twentieth and fortieth year. In adolescence or even in infancy it is sometimes found as a result of the *acute infections;* Chiari found it in 27 per cent. of those under 25 years. In 1908 Fremont Smith collected 144 cases in children.

2. Sex.—Eighty per cent. of cases occur in men, from causes to be enumerated below. In women it usually occurs a decade later than in men. In factory women Jusserand found even a larger percentage than in men.

3. Arterial Poisons.—*Chronic alcoholism* causes 25 per cent. of all cases (Edgren), but it is not known how alcohol operates; it does not raise arterial pressure; lead, coffee, tea and tobacco (which is said to affect especially the coronary vessels) are causative factors. Angiosclerosis has been produced experimentally by adrenalin.

4. Overwork.—This is second only to alcohol in importance. Physical exertion brings one-third of the circulating blood to the muscles, thus lowering the tension in other vessels (*v. i.* Thoma's Theory). According to an older theory exertion raises arterial tension.

5. Poisons Produced Within the Body by Perverted Metabolism.—*Interstitial nephritis* bears a threefold relation to arteriosclerosis: (*a*) It results from diffuse arterial hardening; (*b*) it raises arterial pressure mechanically (Traube) by increased peripheral resistance in the arterioles, or by alteration in the quality of the blood (Bright); (*c*) both processes are coördinate to a common cause. *Gout, diabetes, high living, overeating, obesity* and overindulgence in meat come under this head.

6. Specific Infections.—Edgren and Camac consider syphilis of first etiological importance in 33 per cent. of cases. It operates by chronic toxæmia and endarteritis. Tuberculosis (in 16 per cent.) may produce local changes in the vessels. Typhoid, rheumatism, influenza, scarlatina and other infections are held by French writers to have a direct bearing on vascular sclerosis and endarteritis. Oppenheimer has recently described arteriosclerosis in children nine and ten years old, obviously due to acute infections (*v. s.*).

7. Other Causes.—Nervous factors (worry, high tension, neurasthenia) cause variations in blood pressure. Excessive brain effort tends toward cerebral localization, precisely as digestive excesses may develop changes in the mesenteric vessels, as working with the arms, as in butchers, may develop changes in the brachials, and as excessive walking may produce arteriosclerosis in the femorals. Extreme heat is thought to induce sclerosis in bakers and stokers.

Pathogenesis and Pathology.—(*A*) Thoma considers angiosclerosis a compensatory process which follows widening of the vessels and alteration of the blood. To restore the caliber of the widened vessels, the tunica media contracts, or when it cannot contract, the vessel increases in thickness by growth of the subendothelial connective tissue of the intima. Thus in the fœtus there is no connective tissue in the tunica intima, but it appears when the fœtal circulation changes at birth, new areas of circulation being opened up. All bloodvessels have a nervous supply ending in the Vater-Pacini bodies. They respond to persistent low pressure, or dilatation, by increased proliferation of the connective tissue. Thoma observed arteriosclerosis in experimentally produced aortic insufficiency. (*B*) Others regard arteriosclerosis as due to *inflammation in the nutrient vessels,* the vasa vasorum, leading to growth of connective tissue, *i. e.,* mesarteritis and periarteritis (Huchard, Köster, Hipp, Martin). Arteriosclerosis in vessels with no vasa vasorum must be simply degenerative.

Conceptions of its morbid anatomy differ widely, atheroma of the aorta and endarteritis or arteriocapillary fibrosis being sometimes described under separate headings, but the difference is rather anatomical than pathological. Large vessels, as the carotid, contain more

elastic than muscular tissue, but the small vessels are largely muscular. Two main forms are distinguished, (1) the *diffuse* and (2) the *nodular.* Without distinction as to form, Rokitansky found that arteriosclerosis occurred in the following order of frequency: ascending aorta, arch, thoracic, abdominal aorta, splenic, iliac, femoral, coronary (next after the aorta, according to Huchard and Sansom), cerebral, brachial and carotid arteries. It is rare in the gastric, mesenteric and hepatic vessels and least frequent in the pulmonary trunk. Bergmann found the two varieties as follows:

	Diffuse Form.	Nodular Variety.
	Per cent.	*Per cent.*
Radial	89	2
Ulnar	88	8
Tibial	87	8
Popliteal	64	25
Splenic	64	17
Femoral	58	14
Axillary	53	16
External iliac	49	15
Brachial	49	9
Subclavian	45	43
External carotid	44	22
Cerebral	40	43
Internal carotid	28	60
Ascending aorta	19	56
Common carotid	10	69
Abdominal aorta	0	75

(Both forms may co-exist in the same vessels.)

It will be noted that where the diffuse variety is most frequent the nodular form is least observed and that the converse is true. The *diffuse* form is most frequent in the extremities where arterial tension is most variable and more collateral circulation prevails; the *nodose form* is most common in the aorta and its primary branches, but is also seen in the circle of Willis, where arterial pressure varies greatly (Meynert).

To the *naked eye* the intima may be normal and smooth but most often is uneven and thickened. Small gelatinous or yellowish fatty areas are seen which though at first circumscribed, often fuse into large plaques; they occur where arterial pressure is greatest, as in certain places in the aortic arch (see Aneurysm), or where the vessel divides or gives off branches, as the subclavian or intercostal branches. These plaques of atheroma or nodular arteriosclerosis represent nature's effort to fill out areas of degeneration and dilatation and thus even the caliber of the vessel. *Microscopically* the intima may be increased three or four fold from swelling of its tissue, increase of connective tissue and round-cell infiltration. Granular detritus, fatty needles and cholesterin plates are seen in the *atheroma plaques,* a name indicating their pulpy, grumous appearance. Formerly they were wrongly called *atheromatous abscesses.* An atheromatous area rupturing into the lumen produces the *atheromatous ulcer,* which

may cause embolism, thrombosis or occlusion. Secondary deposit of carbonate or phosphate of lime in the atheromatous areas leads to *calcification* of the arteries or may produce rough areas in the aorta which break on manipulation or if handled carelessly may cut the finger. The lumen of the smaller vessels is often decreased or obliterated (endarteritis obliterans). The middle and outer coats of the smaller vessels are at times thickened from hyaline degeneration and fibrous tissue; the muscular coat may waste and all elastic tissue may disappear; the essential changes are in the elastic tunic. In the larger vessels dilatation or even aneurysm may develop where thickening of the intima fails to protect the vessel wall.

The general results of arteriosclerosis are (1) *vascular,* consisting of loss of arterial elasticity, deformity, tortuosity, obliteration, aneurysm and high arterial tension, and (2) *visceral* alterations in the heart, brain, kidney and other organs (*v. i.*).

Sclerosis in the *pulmonary artery* is most common in the stasis and increased pulmonary tension of mitral lesions and emphysema; the rare cases of Rhomberg, Klob and Neusser have shown that pulmonary atheroma may occur alone, sometimes with aneurysmatic dilatation of its branches and with pulmonary regurgitation.

The term angiosclerosis (Thoma) also includes *phlebosclerosis,* which frequently accompanies arteriosclerosis and sometimes occurs independently, in the lower extremities, lung and portal tributaries in cases of liver cirrhosis or pylephlebitis.

Symptoms and Diagnosis.—In diffuse forms there may be no symptoms, *i. e.,* the process is *latent.* When it is local, visceral symptoms may develop. The clinical picture is extremely variable.

1. The *peripheral arteries* may be involved alone or there may be changes in the aorta, heart, brain and kidneys. The small arteries are visible and appear *tortuous, pulsating and lengthened,* as in the temporals or brachials when the elbow is flexed and the arm is rotated outward; lifting the arm is said to decrease the pulsation. A distinction must be made between *hardness* of the vessel walls and *increased arterial tension,* although both conditions usually occur together. The normal systolic blood pressure ranges from 100 to 140 mm. of mercury. Dunin holds that the systolic pressure ranges between 130 and 150 mm. in arteriosclerosis, and that 21 per cent. have normal or decreased arterial tension. It must be remembered that normal blood pressure does not mean a normal movement of the blood, since accommodative vascular contraction may follow poor vascular filling. It is asserted that increased arterial tension precedes actual sclerosis of the vessels but in these early cases it is probable that some angiosclerosis has already developed. Pressing on the radial with one finger should obliterate the pulse beyond that point if the arteries are not sclerotic, unless there is an anastomotic or recurrent pulse from the ulnar to the radial vessel; this source of error is easily eliminated by simultaneous pressure on the ulnar vessel. The wall of the artery is firm, hard, often uneven or nodular,

and the serial atheromatous plaques sometimes feel like a rosary or the trachea of a goose. In extreme instances the vessel feels calcareous—the "pipe stem artery." The Roentgen rays clearly show the arterial change. The *pulse* is hard (*pulsus durus*), retarded (*pulsus tardus*) or slow (*pulsus rarus*); it rises slowly and is long main-

FIG. 28.

Pulse tracing in arteriosclerosis.

tained (*p. rotundus*), the vessel is firm between the pulse beats (Broadbent), the elastic wave is lessened or absent, the tracing has a rounded or flat top, and the pulse in the two radials may differ (*pulsus differens*). The pulsus tardus and bigeminus are most rare unless there are coronary changes. In vessel obliteration or absolutely rigid walls the pulse cannot be felt. Very slight pressure on the vessels often elicits a systolic murmur (Litten). The right

FIG. 29.

Pulse tracing in extreme arteriosclerosis (Sahli).

radial is normally larger and more often shows changes than the left. It must be remembered that changes may occur in internal vessels without change in the radials (and conversely).

2. *Heart changes.* (*a*) *Simple hypertrophy* of the left ventricle may occur, with heaving apex, loud second aortic tone, tense pulse and thickening of the arteries, which lead to a certain and immediate diagnosis. Some "consensual" right heart hypertrophy is common because of anastomosis between the ventricular muscular fibers or because of stasis in the lungs or emphysema. Those cases in which there is increased tension in the aorta without increased tension in the radials are more difficult of diagnosis. The heart is not hypertrophied in all cases nor, according to Rhomberg, in the majority of cases; hypertrophy, he maintains, does not necessarily follow arteriosclerosis in the extremities or brain, but most often occurs when the splanchnic vessels are involved; Hasenfeld also believes that cardiac hypertrophy only occurs when these vessels and the aorta are sclerosed. The apex may sink outward and to the left when the patient sits, from relaxation and lengthening of the aorta (Traube). (*b*) *Hypertrophy plus dilatation,* with heart insufficiency, dyspnœa, scanty urine and hydrothorax is very common. When the patient in this stage has a systolic murmur, a wrong diagnosis of mitral insufficiency may be made. Interstitial nephritis (*q. v.*) may be the cause, in part at least, of the hypertrophy and dilatation. There may be cardiac weakness even when the blood pressure registers 200 to 300 mm. In high arterial tension a double impulse may be felt and seen at the apex, which is

menacing as it precedes cardiac weakness. Œdema is not frequent. Gallop-rhythm is uncommon without coincident coronary or renal disease. The heart muscle may show various changes, such as atrophy, myocarditis, fatty heart, myofibrosis, myomalacia cordis, the *cor renale,* senile heart, heart aneurysm, angina pectoris, coronary embolism, cardiac asthma and arrhythmia, or sudden death may occur. Two forms of visceral sclerosis may be distinguished: (i) inflammatory, occurring about the vessels, and (ii) dystrophic (malnutritive) sclerosis, which is often somewhat distant from the vessel involved; the visceral sclerosis is best illustrated by the heart changes, such as myofibrosis or myomalacia cordis, induced by coronary atheroma. (*c*) *Valvular lesions* occur from what we may term cardiosclerosis.

3. In *the aorta* dilatation may occur, giving slight *dulness in the second (and third) right intercostal spaces*; this is confirmed by the fluoroscope; the aorta may be felt in the jugulum or abdomen. The normal aorta may be felt in these situations when the heart action is strong, but when there is atheroma, plaques or even a crackling may sometimes be felt. The normal aorta propels the blood onward during the diastole after the aortic valves close; if the aorta loses its tone the peripheral vessels receive inadequate blood, depending wholly on the ventricular systole; then the pulse may resemble the pulsus celer. The subclavian or innominate arteries may be felt even through their entire length when the aorta is dilated. *A short systolic murmur* over the aorta often results from atheromatous roughening or dilatation, and the *second aortic tone is then accentuated* and metallic. Its vibration may sometimes be felt; when much emphysema is present it is best heard in the jugulum. The accentuation may be caused by increased tension, widening or rigidity of the aorta. The murmur and sharp tone are often heard in the neck and back. A burning substernal pain may indicate involvement of the outer coat of the vessel. Atheroma of the arch may, by downward extension, cause aortic stenosis or more often aortic regurgitation.

4. *Renal findings* are next in frequency. The kidneys are often contracted (the senile arteriosclerotic and the genuine contracted kidney). The discussion, whether the renal findings are primary or secondary, is a separate literature in itself (see Interstitial Nephritis). The urine may be increased as a result of simple increase in the blood pressure.

5. *Brain symptoms* are next in importance. Syncope, vertigo, insomnia, headache, tinnitus, neurasthenic symptoms, transient aphasia or hemiplegia result from transient ischæmia. Brain hemorrhage and miliary aneurysms are frequent sequences. Softening of the brain (encephalomalacia) results from embolism or thrombosis of the cerebral arteries. Brain atrophy, dementia (cortical sclerosis), epileptic seizures and the Adams-Stokes' triad have been attributed to cerebral atheroma. Vascular brain lesions occur fairly often in cases which show arteriosclerosis in the extremities and temporal

vessels, though sclerosis of the aorta, coronary or splanchnic vessels occurs more often without peripheral angiosclerosis.

Charcot's *claudication intermittente* (Erb's dysbasia intermittens angiosclerotica, intermittent limping, angina cruris) occurs especially in nervous or neurasthenic subjects. It is chiefly due (*a*) to atheroma in the abdominal aorta and its branches in the limbs; it is seen in abdominal aneurysm in the horse. It appears also to result (*b*) from vessel spasm or (*c*) from atheroma in the spinal vessels, of which Déjèrine has seen but three cases. Erb, reviewing 127 cases in 1904, found that 94 per cent. were in men over 40 years of age; heredity is sometimes causal; 33 per cent. were Jews; syphilis (in 23 per cent.) was less frequently causal than tobacco; alcohol was not proven an etiological factor; most cases developed in the upper classes. Pain, paræsthesiæ and sometimes local cyanosis occur in the legs, which "give way" after moderate exertion. The legs are most comfortable when dependent. The dorsalis pedis (sometimes also the tibial and femoral vessels) usually cannot be felt. Gangrene may result (Boerhaave).

Some vasomotor neuroses—as erythromelalgia, Reynaud's disease, acroparæsthesia—and some neuritis and neuralgia are sometimes associated with angiosclerosis; sclerotic vessels are prone to spasm.

6. *The gastro-intestinal* symptoms probably rank next in frequency. Hasenfeld, Huchard, Allbutt, Litten, Edgren and others have described dyspepsia, round ulcer, arteriosclerotic gastritis, gastro-intestinal hemorrhages, intestinal perforation and ulceration. Some cases resemble gastric cancer. Severe abdominal pain (angina abdominis) has attracted some attention lately; it is due, as is angina, to local anæmia (Ortner's intermittent claudication of the abdominal vessels, Schützler's intermittent dysperistalsis); Pal thinks spasm occurs in the mesenteric and gastric vessels; it develops in two or three hours after eating, and is often accompanied by dyspnœa. Abdominal angina, in the writer's experience, is rather often associated with angina pectoris and angina cruris; in three cases these conditions appeared alternately.

7. The *retinal vessels* are not infrequently embolized, narrowed or aneurysmatic, which leads to softening, hemorrhage and exudation.

8. In the *respiratory tract,* emphysema is common and is sometimes regarded as a compensatory effort. The breathing is often somewhat irregular. Hæmoptysis is referable to stasis, infarction or possibly to atheroma of the pulmonary arterioles.

9. In the *peripheral vessels* the so-called spontaneous or senile gangrene results from obliterating endarteritis. *Thrombosis or embolism* may occur in the aorta, brain, or in the coronary, pulmonary, mesenteric, pancreaticoduodenal and other arteries.

10. The *general nutrition varies.* Some writers distinguish between (*a*) *a plethoric type,* which occurs in elderly individuals and the symptoms of which are good color, wide vessels, increased thirst, hemorrhages from the nose, increased urine, respiratory catarrh,

profuse sweats, muscular rheumatism, headache, enlargement of the liver, emphysema, tense pulse, thick bloodvessels and marked hypertrophy of the heart; and (*b*) *an anæmic type,* with chlorotic habitus, anorexia, dyspepsia, few external vascular findings, dilated heart and endarteritis. This type occurs in individuals between 30 and 50 years of age. French writers speak of a *cachexie arterielle,* in which the pallor is distinguished from the dusky anæmia of cardiac disease. Alfred Stengel has noted that the anæmia is more often apparent than real (pseudo-anæmia).

Early Diagnosis.—Early symptoms are referable to local disturbances in circulation and may only appear after exertion. The patient tires more easily and the arterial tension may rise; dyspnœa on moderate exertion, slight œdema of ankles or epistaxis are suggestive. Early *nervous symptoms* are headache, irritability, somnolence, difficulty in concentration or in undertaking new work, vertigo, neuralgias, insomnia, or neurasthenia appearing for the first time after 40 years of age. A slight albuminuria always should attract attention. Ocular and vasomotor disturbances may characterize the insidious onset. The above symptoms are important since successful therapy depends on the early recognition of what Huchard terms the "presclerotic" symptoms.

Prognosis.—The prognosis is relatively unfavorable, because the symptoms are frequently latent until the process is well established or far advanced. In the early stages arteriosclerosis may be arrested or may remain stationary for a long period, but there is constant danger of apoplexy, angina pectoris, myocardial paralysis, "heart block" or uræmia. It is a most common finding after death from acute infections, so that many deaths attributed to acute affections are more properly deaths from arteriosclerosis. Increased blood pressure may overcome moderate obstruction. The prognosis is most favorable in angiosclerosis of the peripheral vessels, and, as a rule, is rather unfavorable in atheroma of the aorta, because of the danger of coronary disease and aneurysm. Intestinal hemorrhage is ominous. A slow pulse is less favorable than arrhythmia and moderate increase in rate. The plethoric form is less dangerous than the anæmic.

Treatment.—*Prophylaxis* regarding the etiological factors and the patient's mode of life is most important. Alcohol and tobacco should be forbidden, iodides should be given in syphilis, the diet should be regulated in diabetes, colchicum should be given in gout, etc. In early cases the process may sometimes be arrested. Many cases seem to improve on a milk diet, although Rumpf claims that many of the salts ingested are deposited in the arteries. He found that milk sufficient to meet the needs of metabolism (70℥ or 2,175 c.c.) for 24 hours contained over one dram (5 grams) of calcium and magnesium. *Iodide of potash* has been advised, especially by Huchard, who gives 15 to 45 grains daily for twenty days; then after ten days intermission, *nitroglycerine* is used. (See Angina Pectoris and Treatment of Syphilis for details.) Iodides are also used in cases where

no suspicion of syphilis exists. Some physicians oppose its use and find on experimentation that it has no vasodilator effects. Rosenbach claims that it actually irritates the endothelial tissues. In some cases at least it has beneficial effects and in a non-syphilitic series of personally observed cases the walls of the nodular, hard vessels distinctly improved with administration of iodides. Rhomberg thinks that iodides operate less by vasodilatation than by decreasing the viscosity of the blood. It has been recently claimed that nitroglycerine dilates the peripheral vessels only, but on this assumption it is hard to explain the cerebral flushing, which follows its use; indiscriminate resort to nitroglycerine is to be condemned, as increased arterial tension, though pathological, is probably conservative. *Massage, a warm climate, passive exercise* and the *Schott method* of treatment may be employed as outlined on page 366. It is interesting to note that Skoda employed hot hand- and foot-baths to reduce peripheral resistance. Coffee may relieve anginal and abdominal pain.

Rest in bed, venesection, iron and digitalis are indicated when the heart muscle weakens. Huchard advises the following pill:

℞ Sodii iodidi ..... 3j.
Sparteinæ sulphatis ..... gr. xv.
Pulv. glycyrrhizæ ..... q.s.
M. et fac in pil. no. xl.
S.—One after meals.

Rest is more efficacious than are cardiants. Morphine is a valuable nervous sedative and tonic for severe cases of cardiac asthma, but must be used with great care.

ANEURYSM.

Definition.—A dilatation, usually localized and sac-like, of an artery. Aneurysm was described by Galen, Vesalius who made the first diagnosis of thoracic and abdominal aneurysm, Paré, Lancisi and Morgagni.

Etiology.—(*a*) *Arteriosclerosis* is the most important cause. The importance of *syphilis* is estimated at 36 per cent. (Fränkel), 80 per cent. (Malmsten) and 100 per cent. (Drummond). The author thinks the relationship between syphilis and aneurysm is as well established as that between syphilis and locomotor ataxia, and probably with the same percentage. Lancisi, in 1728, was the first to recognize the importance of lues and described "venereal aneurysm." (*b*) *Trauma* is an important factor but is probably overestimated. Sudden effort in strong men with weakened aortæ is a direct factor in partial rupture of the aorta and formation of aneurysm. (*c*) *Sex:* aneurysm is from two to eight times as frequent in males as in females. (*d*) *Age:* the old statement that aneurysms occur before the fiftieth year and arteriosclerosis after that age is still valid; they occur between 30 and 40 (Crisp), 30 to 50 (Orth), 30 to 60 (Roki-

tansky), 40 to 50 (v. Schrötter); later compilations, however, set the age higher. Eighty cases under 20 years of age were collected by Le Boutillier (1903). (*e*) *Embolic and mycotic aneurysms* are usually small and may be multiple in the small vessels. Arterial tuberculosis was found by Weinberger. (*f*) The *traction aneurysm* at the ductus arteriosus Botalli is rare (Thoma).

(*g*) The *arrosion aneurysm,* in which the inner coats bulge out when the adventitia is eroded, occurs largely in tuberculous cavities. The "aneurysmal diathesis" is spoken of by older writers; Pelletier found 63 in one case. Dieulafoy considers that multiple aneurysms of the arch are clearly syphilitic.

Pathology.—In relation to the lumen, aneurysms are *axial* or *peripheral.* According to Quain, the varieties are: (*a*) *true aneurysm,* in which the sac consists of one or more coats. Strictly speaking, a true aneurysm should contain all three arterial coats in its walls, but this is most uncommon. The sac is usually (i) fusiform and diffuse, or (ii) sacculated and circumscribed. (*b*) *False aneurysm,* in which all the coats rupture, or in which, according to some, one coat ruptures through the others, like a hernia. These are circumscribed or diffuse, especially in the transverse arch and in the abdominal aorta. (*c*) *Dissecting aneurysm,* wherein the intima of the vessel ruptures and the blood makes a channel down the vessel between the intima and media or between the layers of the latter. It is a partial rupture. The rupture back into the aorta is an attempt of nature toward recovery; the patient may live for years. (*d*) *Varicose* aneurysm, wherein the sac ruptures into a vein, especially the innominate vein or vena cava; this form is also called *arteriovenous* or aneurysmal varix, when the main change is in the vein.

Atheroma is held to be a common cause, though it occurs very frequently in comparison with aneurysm. It is thought that intimal changes induce aneurysm when the outer coats do not undergo vicarious thickening quickly enough to prevent dilatation. Köster held that *mesarteritis* was the principal change, but Eppinger found the greatest change to be loss of the elastic and muscle-tissue in the media. Syphilitic changes are discussed under Syphilis and are very important. In the sac, thrombi form from the roughened intima and slowed circulation and to a small extent guard against extension and rupture. Aneurysm occurs at those points in the aorta on which the blood stream impacts most strongly; 59 per cent. of thoracic aneurysms occur on the anterior surface of the ascending aorta, 29 per cent. on the convexity of the arch and 12 per cent. on the posterior surface of the descending segment. In 915 aneurysms the seat was in the pulmonary artery in but 0.4 per cent. (Crisp).

Symptoms.—Bramwell distinguishes three classes of cases: (*a*) Those which are entirely *latent,* and become manifest only by rupture or at the autopsy. (*b*) Those presenting *symptoms* of *intrathoracic pressure* but giving no positive physical signs. (*c*) Those *signalized by distinct physical signs, e. g.,* by pulsating expansile tumor.

The Purely Subjective Evidences of Aneurysm.—The clinical signs are most variable, and often change or disappear entirely when the aneurysm changes the direction of its growth; the latency of a large proportion of aneurysms gives a disproportionate value to symptoms *per se*.

1. *Pain.*—Pain is either intrinsic, resulting from subacute aortitis, relieved by reduction of the arterial tension, localized, and dull or aching in character; or it is extrinsic, resulting from pressure on nerve trunks especially in deep-seated tumors, or from the weight of the aneurysm, as illustrated by a case of Stokes in which pain was relieved by the use of crutches. Early pain is of the greatest diagnostic value. Sudden pain in the left chest, whether anginal or not, yielding to treatment, is suggestive of aneurysm. *Pain* in the majority of cases is the *first, the most notable,* and often *the most enduring symptom.* It is often alleviated by iodides; it is usually paroxysmal, sharp, lancinating or boring in character (when, for instance, the spine is eroded); it is augmented by movement, but may cease when the part compressed is entirely eroded. The pain is local or is reflected into the brachial plexus, the arm, neck or along the intercostal nerves and may cause numbness and paresis of the arm. Though it is more usual anteriorly, it is sometimes felt in the interscapular region. Anginal pain follows involvement of the cardiac plexus. Intercostal neuralgia is always suggestive of aneurysm.

2. *Dyspnœa.*—Dyspnœa is frequently *out of all proportion to the findings.* It is often a vagus neurosis, but is also due to narrowing of the chest space by compresison of the aneurysm upon the heart, phrenic nerve, trachea, bronchi, lungs or pulmonary artery; or to compression of the plexus cardiacus or pulmonicus, producing bronchial spasm, and laryngeal spasm or paralysis. Dyspnœa may occur with change of posture (Leube) and is paroxysmal; it is at times more or less urgent, or may sometimes subside entirely.

3. *Cough.*—Cough is frequent, but is one of the least distinctive features. It results from pressure on the air passages, vagus irritation, bronchitis or laryngeal involvement. It is often unproductive. Like all other eccentric pressure symptoms, it varies with the size and direction of the aneurysm; it is paroxysmal (Morgagni) and is prone to appear or disappear hand in hand with the pain and dyspnœa.

4. *Difficult Swallowing.*—Dysphagia results from direct compression, from pressure on the recurrent laryngeal nerve, inducing œsophageal spasm, or most often from adhesions between the aneurysm and the œsophagus. It occurs particularly in low aneurysms of the "recurrent type" (*v. i.* Larynx) and may produce distinct pulsations in a water tube introduced into the œsophagus. One of the earliest aneurysms reported by Stokes was the size of an egg, and produced pain, dysphagia and compression of the left bronchus, with rupture into the latter. Traube described a case of obstruction of the subclavian artery and compression of the left recurrent laryn-

geal nerve in which aneurysm was suspected; but the autopsy revealed carcinoma of the œsophagus, embracing in its regional invasion the left subclavian artery and the recurrent laryngeal nerve. Dysphagia may be intermittent or present only in one posture. Its infrequency is explained by the ready lateral luxation of the œsophagus. The danger of sounding the œsophagus is well known, for distressing lethal hemorrhages have occurred in the physician's consultation room.

5. *Compression of the Vagus.*—Vagus compression results in asthma, laryngeal spasm, vomiting, stenocardiac attacks, hiccough, dysphagia and pulmonary symptoms.

The Physical Signs of Aneurysm.—1. *Inspection.*—Stokes described *two areas of pulsation* in the chest, each distinct *"like two separate hearts."* This is most significant when the cardiac impulse is the weaker. Inspection is most advantageously made with the eye just below the level of the shoulder and near the chest. The typical *expansile pulsation* is best detected in superficial thin-walled aneurysmata in the ascending arch, or in those presenting in the jugulum. Pulsation is less a question of the size than of the location of an aneurysm, and visible pulsation may be absent even in the largest varieties; deeply situated aneurysms give diffuse or no pulsation, or the pulsation appears only during expiration. Throbbing over the aorta is least important when there is also marked arterial pulsation (aortic regurgitation). The *pulsation* must be *expansile* to render the diagnosis absolutely sure, though at autopsy the author saw one case which presented clinically an expanding throbbing aorta and dulness over the arch which was due to an enormous mass of callous connective tissue involving the pleura and pericardium. Another instance of a truly expansile pulsation low down in the left side of the chest, as well as near the spine, was observed in pernicious anæmia; the hands placed over it were lifted widely apart, and the pulsation was seen at a distance of forty feet; the autopsy revealed no aneurysm. Pulsation lent to a tumor, a collapsed lung, the liver or a pleural exudate can usually be distinguished without difficulty.

2. *Palpation.*—Pulsation is felt slightly later than the heart shock; this interval is more obvious the slower the heart rate becomes. The *expansile character of the pulsation* is sometimes more easily detected by the fingers than by the eye. Palpation may reveal remittent tension of the sac, *tenderness* on deeper pressure, resistance caused by thick lamellated thrombi and, in rare cases, elasticity or fluctuation of the tumor during the diastole. Pulsation in a deeply situated aneurysm is best elicited by placing one hand anteriorly and the other posteriorly on the chest. A *thrill* may be felt which is most commonly systolic. *A diastolic shock* sometimes occurs in the jugulum and over the aortic area (Lancisi), and this is not due to recoil from the aortic valves, but to retraction of the hypertrophic aorta lying between the heart and the aneurysm (Rosenbach). Rough palpation may induce embolism.

3. *Percussion.—Dulness* (Walshe) is an important finding only when it is present on either side of the sternum, but not when confined to the sternum. Dulness may be detected in the left interscapular region, though in this case percussion is less valuable, because of the thickness of the chest wall. Aneurysms are often obscured by an emphysema, and they may grow from a deep origin into the lung substance. The dulness may so merge with that of the spine, heart, liver or spleen that no certain statement can be made. Dulness when once detected *may cease or shift* to another place; this is an almost certain indication of aneurysm (Bramwell).

It is inexplicable that aneurysms often grow toward and press upon relatively unyielding structures, while the softer parts in their immediate vicinity, as the lungs, are less compressed (v. Schrötter).

4. *Auscultation.*—Auscultation is sometimes wholly negative, even in large sacs—explained by thrombi in the sac and weak heart action. *Two clear tones are most frequently heard;* the second tone, heard over a circumscribed area, is pathognomonic of aneurysm (v. Schrötter), and originates in the aneurysm itself; some writers attribute it to closure of the aortic cusps; it is most often heard in aneurysms near the ascending arch. Two tones in the abdominal aorta are scarcely ever heard normally. A *systolic murmur* is frequent, and is usually blowing, roaring or scraping; it is due to vibrations in the aortic wall, to entrance or exit of blood from the aorta into the sac, to blood waves initiated by angles in the direction of the current, by thrombi, or more rarely by pressure of the sac upon the aorta itself.

The less frequent *diastolic murmur* is most probably caused by (relative) aortic insufficiency, when the aneurysm lies in the first part of the aorta. It may be due to folds in the aorta, vibrations of clots, dilatation of the left ventricle, or to dissection of the aneurysm into the sinus Valsalvæ (Stokes, Marey). It is usually combined with a systolic murmur. A continuous murmur is rare. Of 132 cases (Sansom) in which the diagnosis was made, only 12 presented a systolic murmur, 25 gave systolic and diastolic murmurs, and in 6, diastolic murmurs alone were found. Douglas-Powell detected murmurs in about half of his cases of sacculated aneurysms.

The Heart.—Its position varies with the size and location of the aneurysm. (*a*) Large aneurysms of the ascending aorta not only depress it, but cause such axial rotation that the left lies lower than the right ventricle; aneurysm in this location may cause pressure atrophy of the right ventricle. (*b*) Aneurysm of the under surface of the arch may dislocate the heart to the median line. (*c*) Aneurysm of the descending segment may flatten the heart against the chest wall (Neusser), thus simulating mitral regurgitation. (*d*) Secondary aneurysmatic outgrowths from the chief aneurysm complicate the physical findings; these growths sometimes attain incredible proportions and produce confusing symptoms. (*e*) In uncomplicated cases myocardial alteration is entirely accidental (Stokes, Key and Skoda).

Failure of the heart to hypertrophy is explained by anæmia, arteriosclerosis, coronary disease or compression of the pulmonary artery, thereby decreasing the flow of blood to the left heart and coronary arteries. *Hypertrophy* of the left ventricle bears an intimate relation to aortic leakage; it may in rare cases result from aneurysm involving the entire periphery of the aorta. Stokes states that the most violent aneurysmal pulsation he ever witnessed occurred with marked myocardial atrophy. Compression of the pulmonary artery, early observed by Rindfleisch and Obermeier, may cause relative tricuspid regurgitation or pulmonary insufficiency (v. Schrötter).

The symptoms of aneurysm are given in their entirety in Stokes' early observations.

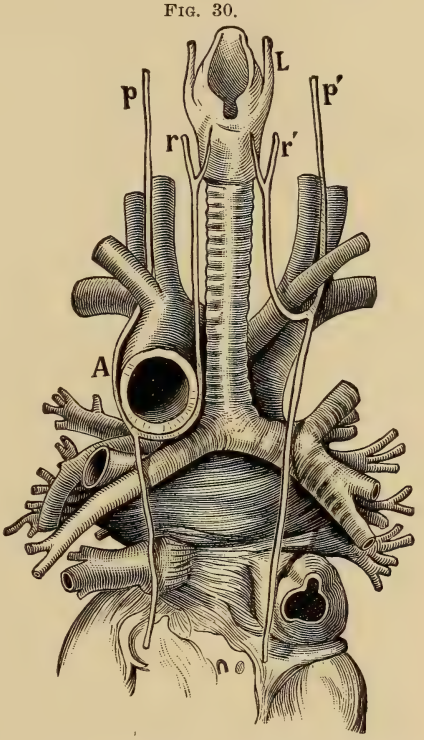
The Vessels.—The *pulsus differens* is frequently physiological, due to irregularities in the size or branches of the radial vessels. Stokes explicitly stated that the right radial is normally larger than the left, whence if the right radial pulse is smaller, it means more than if the left were slightly smaller. When one radial is small, its fellow ulnar is often vicariously larger. A pulse may grow smaller and later return to its normal size; this is due to change in the size and the direction of the aneurysm. When the radials differ essentially in volume it is advisable to compare the brachials. Asymmetrical sphygmographic and manometric findings may be due to other causes than aneurysm, as atheroma, coagula, embolism or reduction of the lumen of a vessel by the dragging of the aneurysm; v. Schrötter instances a case in which the aorta was so compressed by a sac lying between it and the spine that the abdominal and femoral pulsation was obliterated. Changes are observed most often in the carotid and subclavian vessels. The *pulsus paradoxus* and capillary pulsation may occur with large aneurysmatic sacs (Quincke). *Retardation* of the pulse occurs especially in fusiform aneurysms. The location of an aneurysm may sometimes be determined by regarding the vessels altered. If pressure is exerted upon the *superior cava,* dropsy of the upper portion of the body, of one or both arms, swelling of the face and of the tongue, sometimes exophthalmos and a "brawny" or "collar-like" distention of the neck occur. Obstruction above the azygos vein causes lividity and œdema which are confined to the head and arms; when it occurs below that vessel the chest is also congested. Obliteration of the innominate and azygos veins or the venæ cavæ may occur. *Rupture into the superior cava* is attended by cyanosis, and a continuous bruit like the *bruit de diable* is often present; in Ewart's case (not suspected during life) phlebotomy was performed, and bright-colored blood escaped in jets. The continuous murmur may occur in vascular sarcomata or in aneurysms rupturing into the pulmonary artery or right ventricle.

The Respiratory Tract.—*Tracheal tugging,* described by Oliver and Cardarelli simultaneously, is elicited by lifting the larynx with the fingers; a tugging sensation in the trachea is then felt with each systole; this symptom occurs in cases of cardiac overaction, left ven-

tricle hypertrophy, aneurysmata of the arch and of the innominate and carotid vessels, and in adhesions to the trachea.

Tracheal pulsation is present in many cases of aneurysm and may be demonstrated by the laryngoscope. The examination should be made by direct sunlight, and the observer should sit before the standing patient. Tracheal pulsation also occurs in tumors which compress the trachea; it may also be physiological. Drummond has described a systolic murmur over the trachea. A short murmur, synchronous with the systole, can sometimes be heard when the patient breathes quietly with the mouth open.

FIG. 30.



Dieulafoy's diagram of the region in which he describes his "recurrent type" of aneurysm; posterior view. A, aorta, in contact with the trachea and left bronchus; L, larynx (seen from behind); p, left and p', right vagus; r, left recurrent laryngeal nerve, passing under the aorta, and r', right recurrent, in relation to the subclavian artery.

Recurrent laryngeal paralysis was first described by Morgagni and Todd was the first to note laryngeal muscular atrophy on the affected side. Unilateral paralysis with the cadaveric position of the vocal cords, in the absence of obvious cause, is indicative of intrathoracic aneurysm, neoplasm or mitral stenosis. The cough is hoarse, clanging or brazen. It is a paralytic cough, *i. e.*, an imperfect expiration which lacks forcible closure of the glottis observed in ordinary coughing efforts; it was called the bovine cough by Wyllie from the fact that cattle have no false cords whose closure is an important mechanism in coughing. The right recurrent laryngeal nerve, curving around the subclavian artery, does not enter the thorax, and is therefore seldom involved; whereas the left recurrent nerve passes into the chest and curves between the aorta and left bronchus; Dieulafoy described a "recurrent type of aneurysm," which is usually small, syphilitic in origin, often multiple, involves the left recurrent nerve, and causes spasm of the larynx, due to recurrent laryngeal irritation contracting the posterior transverse arytenoid muscle and inducing dyspnœa and inspiratory stridor. Irritation of other vagus branches induces spasmodic dysphagia, anginal pain, etc.

LUNG SYMPTOMS.—Stenosis of the right bronchus is less frequent than that of the left, which lies nearer the aortic arch. The lung

moves less freely than normal; it suffers inspiratory retraction, and the breath sounds and fremitus are diminished. A short note elicited over the left upper lobe may arouse suspicion of an aneurysmal compression. It is common in the "recurrent type" of Dieulafoy.

Rokitansky's dictum that large aneurysms are almost never associated with *pulmonary tuberculosis,* was disproven by Stokes, Fuller and Jaccoud. Tuberculosis results in 13 per cent. of cases of aneurysm from trophic change, compression of the pulmonary artery or decreased lung excursion.

FIG. 31.

Dieulafoy—I–VII, Multiple syphilitic aneurysms.

Bronchiectasis, bronchorrhœa and suppuration of the lung is well called "aneurysmal phthisis" by Osler and Ross. *Pulmonary retraction* is observed from compression, adhesive pleurisy or interstitial pneumonia. *Pleurisy with effusion* may complicate and entirely obscure an aneurysm. *Pulmonary gangrene* occurs from compression of the nutrient artery of the lung or stagnation of mucus. Gangrene of the bronchial mucous membrane also occurs. Gangrene may produce subcutaneous emphysema. *Pulmonary hemorrhage* may occur from gross rupture into a large tube, from mere granulations in a compressed tube, or from an aneurysm slowly leaking into the parenchyma of the lung. The hemorrhage may be fatal, premonitory or continuous; it may be seen as flecks in the sputum, as rusty sputum, as prune-juice expectoration with alveolar epithelium and as discharges of pure blood or more frequently of imperfectly coagulated blood.

SYMPATHETIC SYMPTOMS.—The pupils are often involved; myosis results from sympathetic paralysis and high blood pressure, and mydriasis from sympathetic stimulation and low blood pressure. Irregular pupils may be syphilitic. Pallor, redness and sweating are infrequent.

Diagnosis.—The clinical signs and symptoms are most variable (Stokes). The presence of an aneurysm should be suspected when *marked pain* or *paroxysmal dyspnœa* occurs. *Variability* of physical signs is suggestive of aneurysm.

DIAGNOSIS OF LOCATION.—1. Aneurysm of the *ascending portion* is called by Broadbent the "aneurysm of physical signs." The usual signs are most often observed in the first interspace to the right of the sternum, which is often eroded. Luxation of the heart to the

left, slowing of both radials, right recurrent paralysis, compression of the superior vena cava and pulmonary artery with hypertrophy of the right ventricle, dyspnœa and phthisis occur. Anginal or other cardiac symptoms frequently occur, and the aneurysm, though small, is usually rapidly fatal from rupture into the pericardium, pleura, upper cava or heart-chambers. Aneurysms just above the valves tend to grow downward because of the reflux of blood from the aorta, whereas those located higher grow upward, because of impact of blood from the left ventricle. Sympathetic involvement is common.

2. *In the arch,* the "aneurysm of symptoms" is observed with left recurrent paralysis or spasm, stridor, pulsation in the jugulum, unusual prominence or luxation of the sternal ends of the clavicles, prominence of the subclavian arteries, dulness to the left of the sternum, myosis, slowing or lessening of the left radial pulse, compression of veins particularly the left innominate, of the trachea, thoracic duct, sympathetic ganglia, bronchi and left upper lobe, and disturbance in the brain circulation (hemiplegia) when the carotid artery is occluded. These aneurysms usually grow backward.

3. In the *descending segment,* the "aneurysm of latency" is most often encountered. If there are symptoms, pulsation in the left interscapular region, slowing of the femoral pulse, pressure on the azygos or hemiazygos, pressure on the spine with erosion and tenderness to touch, intercostal neuralgia (sometimes with recurrent attacks of herpes zoster), stenosis of the left bronchus or œsophagus, pressure upon the heart forcing it against the sternum, and increased heart-rate from plexus pressure, are present. The *x*-rays alone detect aneurysms lodged in the lung substance.

Differentiation.—Differentiation from *solid tumors* is determined by their propagated non-expansile pulsation, their more rapid development, leukocytosis and cachexia. In aneurysm the lymph glands are rarely enlarged from lymph stasis or inflammation; adenopathy is frequent in mediastinal tumor, and continued increase is directly indicative of neoplasm. Pain, recurrent paralysis and dysphagia are common to all tumors, both aneurysmatic and neoplastic. Venous ectasia is more common in tumor. A few rare instances of coincidence of tumor and aneurysm are observed. The pulsation in carcinoma is rarely associated with a diastolic "back stroke," which is frequent in aneurysm. An unequal pulse indicates aneurysm or atheroma. The bruit in tumor is usually systolic, but murmurs are frequently absent in aneurysm. Tugging is present in both but more common in the latter. Signs of aortic atheroma argue in favor of aneurysm. Aneurysm runs a much more protracted course than tumors. Exploratory puncture and diagnosis *ex juvantibus* with iodides are of differential importance.

Other causes for pulsation, such as a dynamic throbbing (aortic regurgitation), throbbing in acute aortitis, pulsation in an aorta dislocated from kyphosis, and pulsation in pneumonia (Graves), anæmias or in vascular tumor of the lung or pleura, are usually differen-

tiated when all symptoms and signs are carefully considered. In *pulsating empyema* the dulness is usually low in the chest and largely left-sided; puncture reveals pus and stops the pulsation.

Prognosis.—Recovery is rare, though spontaneous cure is possible by compensatory thickening of the intima, by organization and calcification of thrombi in the sac or by its inflammation. Sometimes one aneurysm obliterates another. The course is generally two years after the diagnosis is made. Patients have been known to live and work hard ten or twenty years. The author watched for twelve years a blacksmith who worked though he had an aneurysm of the ascending aorta, which was as large as his fist, and which protruded beyond the ribs. The size of the sac is of no prognostic value. Those of the small "recurrent type" are most dangerous, and large ones may be protected by connective tissue. Arteriosclerosis is prognostically unfavorable.

Mechanism of Death.—(*a*) *Rupture* occurs in 75 per cent. of cases: 20 per cent. into the left and 13 per cent. into the right pleura; 16 per cent. into the left bronchus; 12 per cent. into the pericardium; 9 per cent. into the left lung; 6 per cent. externally; 5 per cent. into the œsophagus; 4 per cent. into the trachea; (*b*) *suffocation;* (*c*) *marasmus,* especially in large aneurysms; (*d*) intercurrent disease; (*e*) embolism; and (*f*) brain lesions, are also immediate causes of death. Hemorrhage usually shortens the course, but in one case rupture into the bronchus occurred five years before death. Another patient suffered external rupture of the sac; he collected the blood in a basin and though he fainted from the hemorrhage he lived four months, only to die of typhus fever. Stokes described an external rupture in which the presence of mind of the nurse, in stuffing some cloth into the sac, kept the patient alive for a considerable time.

Treatment.—1. The *Tufnell treatment* of rest, low diet and limited fluid is the result of the methods used by Morgagni, Albertini, Valsalva and Bellingham, without the venesection recommended by Valsalva. Absolute mental and physical rest is required for several months, and food is reduced to the minimum. The heart action and blood pressure are greatly lessened. The treatment is of value only in cases of sacs with small necks. Active individuals will not tolerate this method. Relative quietude, and moderation in every phase of life must then be insisted upon. Ergotin, hypodermatically, has little effect. Tr. aconite, ♏i to iv, may relieve the throbbing.

2. The *iodides* have been given prominence by Balfour, though they were used first by Bouillaud and later by Graves, Nélaton and others. They are of equal value in syphilitic and non-syphilitic cases. It is not clear how they operate. They increase the secretions but do not inspissate the blood. It is questionable from the experimental standpoint whether arterial tension and dilatation of the sac are lessened; it is certain that pain is alleviated. Ten to twenty grains are given at a dose as in arteriosclerosis. The dose is lessened if the pulse rate increases. Aside from relief of pain, the iodides are

useless in large aneurysms with wide communications or in fusiform varieties.

3. *Cold* applications may relieve pain when the sac is near the surface. Supports may relieve the pain and protect against rupture. *Calcium chloride* in doses up to ten or fifteen grains has been used to promote coagulation, but apparently without effect.

4. *Gelatin injections* (1 or 2 per cent. solution) are recommended by Lancereaux to produce coagulation in the sac. It cannot be said that the results are convincing. Gelatin has been used for centuries in China and Japan to aid coagulation. There is danger of embolism and 23 deaths from tetanus were reported by Dieulafoy, 1903.

5. Electrolysis, *introduction of fine wire,* compression and surgical intervention have given discouraging results. A few cases of recovery are reported but one cannot help comparing their small number with the probably equal number of spontaneous recoveries (Börger, in 1906, compiled 19 cases of dissecting aneurysms of the aorta which recovered). Fatal thrombosis may follow what in a lesser degree of thrombosis might have been beneficial. Total embolism of the aorta may also occur.

6. *Dyspnœa* is treated as in cardiac disease. Venesection may afford temporary relief, and morphine is finally indicated. Tracheotomy may be necessary in laryngeal spasm (reflex recurrent irritation) but the actual compression by the sac is usually too low for operative interference. Digitalis is at all times contra-indicated.

Abdominal Aneurysm.—This type constitutes 5 per cent. of aneurysms of the aorta. Over two-thirds occur in the upper part of the abdomen and 40 per cent. either at or above the cœliac axis. The average size is that of an orange and the tumor develops from the anterior surface of the vessel, less frequently from its lateral or posterior wall, and grows downward. Most of them are false aneurysms.

Symptoms and Signs.—Four per cent. of abdominal aneurysms are *latent* clinically; a good sized sac may be hidden under the arch of the diaphragm. *Pain* is severe, constant and boring or paroxysmal. It may become less severe after hemorrhage from rupture. Relief is sometimes obtained by lying on the stomach. Indefinite *symptoms* are common in the *stomach* though they are less frequent than in the *duodenum,* where obstruction and hemorrhage may occur. *Inspection* shows pulsation in 65 per cent., *expansile pulsation* in 15 and *tumor* in 57 per cent. *Palpation* may elicit the expansile pulsation and in some cases a *thrill.* *Percussion* may reveal *dulness* but the close relations to the liver, spine, kidneys and heart are prone to give indefinite findings. The diaphragm bounds the aneurysm above; the kidney to the left; the liver, pancreas and kidney to the right; and the stomach, pancreas and colon in front. The tissues about the sac thicken and produce adhesions. On *auscultation,* a *systolic murmur* obtains in 50 per cent. or two tones are heard; a diastolic murmur is sometimes heard.

Rupture, diffusion by dissection, great thickening of the sac and thrombi formation may easily obscure all auscultatory signs.

Diagnosis.—In two-thirds of the cases an incorrect diagnosis is made.

1. Compression Symptoms.—Compression symptoms, gastric and duodenal, have been mentioned. Abdominal aneurysm may be mistaken for gastric ulcer or cancer. The spine is eroded in 10 per cent. of cases; *lumbar neuralgia, lumbago,* muscular rheumatism, myelitis, Pott's disease and spinal tumor may be simulated. Pulsation should be searched for along the spine. The affection may suggest renal colic, kidney tumor, perinephritis or psoas abscess (fluctuating blood from rupture). The ureter may be compressed. Pressure on the splenic vein may produce much splenic enlargement. The heart is rarely compressed and dyspnœa is uncommon. Retardation or absence of the femoral pulse may be of value in differentiation.

2. Abdominal Pulsation.—"Instead of being your first, it should be your last idea that abdominal pulsation is due to aneurysm" (Jenner). *Epigastric pulsation* may be confused with hypertrophy of the right heart or pulsation of the liver. The greatest danger of error lies in diagnosticating as aneurysm the pulsation which is so often seen in acute hemorrhage, or especially in neurasthenic subjects, who complain of pain and throbbing and in whom thrills, systolic and, according to Rosenbach, even diastolic murmurs may be felt and heard, especially when atheroma is developing. Rosenbach especially emphasizes the diagnostic importance of "intermittent dilatation of the aorta" which is frequently diagnosticated as aneurysm. The pulsation must be *distinctly expansile* (even then occasional errors are made) and a definite sac must be felt; otherwise constant errors will be made. The *dynamic* pulsation of aortic regurgitation concerns the same points.

3. Communicated Throbbing.—Communicated throbbing, lent to a tumor of the stomach lying on the aorta, may also be attended by a systolic murmur but the differentiation is usually made with ease, except in certain soft neoplasms. Tumors are often felt to better advantage when the subject is in the genupectoral attitude; they are more movable, more often produce dilated veins, ascites and peritoneal friction. Aneurysm of the abdominal aorta or of its branches may, in exceptional cases, be movable. Stokes held that tumors developed upward and aneurysms downward.

4. Aneurysm of the Branches of the Abdominal Aorta.—(*a*) Aneurysms of the *cœliac axis* are difficult or impossible to distinguish and may occur with aneurysm of the main trunk. More than 20 cases are recorded. They are often traumatic or embolic (mycotic). (*b*) *Superior* (20 cases, Baccelli, 1904) *or inferior* mesenteric aneurysms are very uncommon; they occur most frequently in children, from sepsis. (*c*) Over 20 cases of *splenic* aneurysm are recorded. The spleen is often enlarged. (*d*) In the *hepatic artery* (38 cases, Schupfer, 1906) it is induced by acute infections

(73 per cent.), trauma, embolism or gall-stones; 41 per cent. develop in the main trunk and 25 per cent. in its right branch; there is but one case in which it developed in the artery of the cystic duct. Pain (in 71 per cent.), hemorrhage (in 58 per cent.), icterus (in 63 per cent.), vomiting and large spleen have been noted; in 79 per cent. rupture occurred (in 10 cases into the peritoneum); they never pulsate, and in only one case was there a systolic murmur; Kehr operated on one case successfully. (*e*) Over 10 cases of aneurysm of the coronary artery of the *stomach* are recorded. (*f*) In the *renal* artery aneurysm is rare (26 cases, Skillern, 1906). Three cases were successfully operated on. Von Schrötter found only 1 case in nearly 20,000 autopsies.

Prognosis.—Death is usual and the clinical course averages one to three years. *Rupture* occurs in 66 to 75 per cent. of cases: 33 per cent. into the peritoneum; 16 per cent. into the pleura, though the crura of the diaphragm offer considerable resistance; 7 per cent. into the duodenum; and 7 per cent. into the retroperitoneal tissue. Death does not necessarily nor at once follow rupture even into the peritoneum.

Treatment.—Treatment is as in the thoracic type. From its accessibility, operative procedures are more common and somewhat more successful. Continued compression of the aorta has benefited cases of aneurysm located low in the abdomen.

ACUTE AORTITIS AND ARTERITIS.

These are rare lesions, occurring in the aorta as part of a sepsis, benignant or malignant endocarditis, or in the peripheral vessels after influenza or other infections. *Acute aortitis* was often confused with chronic atheroma, acute arteritis and with organizing thrombi in the arteries. Syphilis (*q. v.*) is a factor in aortitis, of which the symptoms are retrosternal burning, actual anginal pain, dyspnœa, anxiety and acute dilatation of the arch without violent throbbing of its branches. The diagnosis is uncertain and the treatment symptomatic.

Periarteritis nodosa was first described by Kussmaul and Maier (1866) and is called "arteritis proliferans nodosa" by Fletcher. This is a rare affection of which Versé in 1905 could find but 20 published cases; it consists of localized proliferation and round cell infiltration of the intima, with its rupture through the other coats, and leads to thrombosis or aneurysm. It is probably an infection. It occurs largely in men between twenty and thirty-five years of age; one case occurred in an infant three months old. The nodes are seen in the vessels of the muscles, heart, kidney, intestines and skin; they produce hydrops, muscular pains, nephritis, convulsions, paralysis, vomiting, diarrhœa, alimentary hemorrhage, epigastric pain, low temperature and a rapid pulse. The affection is fatal within six to twelve weeks, is usually mistaken for typhoid or meningitis and treatment is purely symptomatic. In one-quarter of the cases there were

syphilitic antecedents and Schmorl saw one cure with the use of mercury.

RUPTURE OF THE AORTA.

Rupture of the aorta may be traumatic or the result of severe strain, endarteritis or narrowing of the vessel. Tolot and Sarvonat (1904) collected but six cases of rupture of a sound aorta. All coats may be ruptured and this usually occurs in a transverse direction. If the rupture is partial, a dissecting aneurysm results which may burrow even to the popliteal arteries. It most often occurs in the first segment when hæmopericardium results. Rupture into the lung, mediastinum or retroperitoneal connective tissue may occur. The symptoms are sudden pain, a sense of "something ruptured" and the signs of hemorrhage; sudden death usually occurs, though prolongation of life for from one to eleven years has been recorded in cases of dissecting aneurysm.

Embolism of the aorta has a close relation to thrombosis, being due to impaction of atheromatous plaques, tumor tissue, echinococcus cysts, endocarditic plugs or ordinary thrombi. It is very uncommon. Sudden death results if the site is in the first part of the vessel. If it occurs near the division of the iliacs, collateral circulation is possible. There are pain and stiffness in the legs, sensory disturbances, paresis and contractures from anæmia of the spinal cord. The femoral pulsation is absent. The clot may back up toward the heart or it may resorb. The bladder and rectum are disturbed. Hemorrhage from the stomach, bowels and kidneys has been observed. The prognosis is bad and the treatment is symptomatic.

Thrombosis usually results from several coincident causes, as acute infections, weak heart, aneurysm, atheroma or compression. Most cases occur in the transverse arch between the left carotid and subclavian vessels or in the abdominal aorta at its point of division. The pain, paraplegia or anæsthesia produced is sometimes confused with spinal cord disease. It may be sudden; if gradual, collateral circulation may be established.

SECTION III.

DISEASES OF THE RESPIRATORY TRACT.

DISEASES OF THE NOSE.

ACUTE RHINITIS, CORYZA.

Definition.—An acute nasal infection.

Etiology.—(*a*) It is an *independent infection,* sometimes epidemic and contagious; it occurs oftenest in the spring and fall and is promoted by variations in temperature. Cold is only a predisposing factor; various bacteria are held causative, but Thompson and Hewlitt have shown that 84 per cent. of cultures are negative. (*b*) It is a *concomitant* of various *infections,* notably of influenza, measles, pertussis, glanders, syphilis, etc. (*c*) *Toxic factors,* as iodides, bromides, ipecac dust, dust, etc., may cause coryza. (*d*) *Local disease,* as chronic catarrh, polyps, ulceration and possibly gout, are predisposing factors.

Symptoms.—The initial symptoms are malaise, depression, anorexia, chilliness, fever of 101° to 102°, pains in the limbs and sometimes, in the young, delirium and convulsions. The mucosa is dry, reddened, burning, painful, swollen and obstructed. There is repeated sneezing and anosmia. Herpes is common. There follows a clear, watery, nasal discharge (*coryza*) for one or two days, which subsides at night and recurs in the morning; within two days it becomes thickened and purulent. Extension may occur to the eyes, frontal sinus, antrum of Highmore, ear by the Eustachian tube or to the pharynx or larynx. In infants nasal occlusion prevents nursing and may cause dyspnœa or suffocation, because the tongue lies high in the mouth near the palate. Acute rhinitis lasts one-half to one week and is seldom confused except with incipient measles.

Treatment.—At the very onset lavage with simple hot water is often sufficient; Dobell's solution (sod. borat. and sod. bicarb. āā ʒij, ac. carbolici ʒss to water ʒx), diluted freely to avoid irritation, is beneficial; adrenalin solution 1 to 1,000 is excellent, but excessive application may induce œdema of the throat and epiglottis. A cocaine solution (the plain alkaloid gr. j to albolene ʒj) relieves turgescence and frontal pain, but is a dangerous remedy to give into

the patient's hands. Dover's powder gr. x, a saline aperient and a hot bath at the onset, usually give relief, especially when followed by:

℞ Extr. belladonnæ ..... gr. iiss.
Fluidextr. aconiti ..... ♏x.
Acetphenetidini ..... gr. xv.
Camphoræ monobromatæ ..... gr. xv.
M. et ft. capsulæ x.
S.—One capsule every hour for five or six doses.

℞ Phenolis ..... gr. iii.
Mentholis ..... gr. iv.
Olei theobromatis ..... q.s.
M. et fac suppositoria no. x.
S.—One locally in nose every 2 hours.

Drugs are of little use unless the patient remains in bed to relieve internal congestion and to avoid extension or recurrence. Salicylates mitigate the frontal pain. Cool baths aid in averting recurrent attacks.

HAY FEVER.

Catarrhus æstivus or summer catarrh, was first described by Bostock (1819). Morill Wyman first described the "June" or "rose cold."

Etiology.—Its etiology is not known, but the disease has long been associated with the pollen of various plants and grains. Dunbar believes that pollen contains toxalbumins. It prevails especially in the United States and England, develops in persons between fifteen and thirty years of age, and twice as frequently in males as in females. It is more common in the upper educated classes, and perhaps in gouty individuals; it is most prevalent in the warm months and in cities. Three special factors are noted: (*a*) A *predisposition* in the form of nervous instability or other systemic tendency. (*b*) An *irritability* or other nasal abnormality, as undue nasal turgescence, or irritability to dust, odors, etc., hyperæsthesia of the inferior turbinates, polyps or septal irregularities; Austin Flint became asthmatic from sleeping on feather pillows. (*c*) The *exciting cause* or irritant, as pollen, dust, sudden change of temperature and apparently in some cases, mental excitement.

Symptoms.—The onset is that of the vulgar coryza, but the disease is attended by more constitutional reaction and greater *depression. Injection* of the *eyes* with lachrymation and photophobia usually accompanies or precedes the coryza. The throat, larynx and bronchi are dry and burning. Fever, 100° to 101°, is fairly common. There is pain over the frontal sinuses and sometimes over the temporal and occipital regions. The nasal vessels are congested and the *nose* itself becomes *swollen* and injected, resembling closely the alcoholic facies. In many cases *cough* develops; this is frequently followed by *asthmatic* seizures which simulate bronchial asthma (*q. v.*). Recurrences are usual and attacks of coryza may alternate with

asthmatic paroxysms. With the first frost the affection stops, as if by magic. The outlook is good as to life, for only isolated instances of death from asthma are recorded; as to recovery, the outlook is much less favorable.

Treatment.—(*a*) Prophylactic treatment; cauterization of irritable, congested or hypertrophic areas, and correction of polypoid or other abnormalities cure some cases and alleviate suffering in others. (*b*) *Nasal* treatment; much relief may be given, when curative measures fail, by nasal douches, by painting or irrigating the nose with 1 to 1,000 adrenalin or other astringent solutions; suprarenal extract in doses of gr. ij to v has been recently used internally, as has nearly every drug in the Pharmacopœia. (*c*) The *general physiological resistance* is increased by fresh air, cool rubs, tonics and nervines. (*d*) *Change of climate* is advisable before the date on which, with certain regularity, hay fever appears; a sojourn at the seaside, in northern Michigan, in Canada and especially in a high altitude, often greatly relieves or sometimes wholly checks the symptoms. (*e*) *Asthma* (*v.* Bronchial Asthma). (*f*) *Dunbar's serum,* patented as pollantin, is used in the nose (not hypodermically) every morning, as a powder. In 1,240 cases Dunbar claims that 56 per cent. were greatly helped, 31 per cent. benefited and 13 per cent. unaffected.

CHRONIC RHINITIS.

Chronic nasal catarrh properly comes under the care of the specialist, but minor cases are treated by the practitioner; some are due to scrofula, syphilis, anæmia or to other constitutional causes. Chronic catarrh may develop from repeated acute attacks, or may be chronic from the onset.

The Hypertrophic Form.—The nasal mucosa is red, swollen, spongy, and is covered with thick, gray-yellow secretion which may desiccate and sometimes form the basis of rhinoliths. Microscopically, the bloodvessels are wide and are surrounded by round cells, and the submucosa is thickened. The nasal obstruction necessitates oral breathing, partially nasal speech, obtunded taste and smell, and produces an elevation of the hard palate and a peculiar facial expression. The secretion is usually thick, yellow and purulent, rarely thin and abundant (rhinorrhœa). This type is often associated with adenoids and follicular hyperplasia in the pharynx. The lachrymal duct may be obstructed, and Eustachian occlusion may induce permanent deafness; laryngitis, bronchitis, bronchial asthma, polyps and sinus disease are not infrequent complications. Nasal disease may be the cause of blepharospasm or other facial spasms, limitation of the field of vision, vertigo and mental depression; and even psychoses are attributed to nasal reflexes. Fliess attributes dysmenorrhœa to chronic rhinitis. In its treatment possible constitutional factors, as anæmia, syphilis or scrofula, are to be considered. Douches of carbolic acid, insufflations of equal parts of alum and

calomel, and cauterization by the galvanic cautery or by chromic acid, are indicated. Douches should be given under low pressure, lest fluid reach the middle ear.

The Atrophic Form.—It is disputed whether this form begins as an atrophic process or results from hypertrophic disease. It is more frequent in females and usually begins early in life. The mucosa is pale and dry, its epithelium has suffered marked metaplasia, the erectile tissue wastes, the nasal passages are unduly roomy and the secretion dries in the nose and emits a most offensive odor (*ozæna*). Its actual cause is not determined. Various microörganisms have been found, as the Bacillus fœtidus, Bacillus mucosus and Bacillus fluorescens liquefaciens. Grosheintz and Meiser have noted its association with a widening of the face in 33 per cent. of cases. Its sequences are similar to those of the hypertrophic variety.

Treatment.—The results of treatment are not satisfactory. Douches of a 1 per cent. permanganate of potash solution, iodoform insufflations, massage with iodine one part, potas. iodide 10 and glycerine 100 parts, and packing with gauze soaked in balsam of Peru are usually employed. Paraffin injections beneath the mucous membrane are recommended to restore the normal lumen of the nasal passages.

RHINITIS FIBRINOSA.

Rhinitis fibrinosa has been considered under nasal diphtheria, to which it is most often due. It may result from streptococcus and staphylococcus infection; it occurs in measles, scarlatina, pneumonia, typhoid and also as an independent lesion. Irrigation with 1 per cent. carbolic acid or 1 to 1,000 sublimate solution is efficacious. Antitoxin is indicated in infection by the Klebs-Loeffler bacillus.

EPISTAXIS.

Epistaxis is a symptom only. Its causes may be grouped as follows: (*a*) *Local nasal* lesions, trauma, chronic and acute catarrh, picking of the nose, foreign bodies, ulceration (simple, tuberculous, syphilitic, carcinomatous) and angioma. Osler reports cases of epistaxis associated with telangiectasis of the skin and nasal mucosa. (*b*) *Hyperæmia,* either venous, as in cardiac, respiratory and other obstructive lesions, or arterial, as in hypertrophy of the left ventricle caused by aortic leakage, interstitial nephritis, atheroma, apoplectic habitus and gout. Some cases are seemingly vicarious to suppressed menstruation or hemorrhoidal bleeding. (*c*) *Acute infections,* principally typhoid, less frequently the others. (*d*) *Hemorrhagic diseases,* as pernicious anæmia, chlorosis, leukæmia, pseudoleukæmia, scurvy, hæmophilia, Werlhof's disease and cachexiæ. (*e*) Other conditions, as liver cirrhosis, pregnancy, icterus, diabetes and puberty; balloon ascensions or high altitudes are also causative.

The most frequent point of hemorrhage is the lower anterior part

of the cartilaginous septum. The symptoms are chiefly the local hemorrhage from the anterior nares, but occasionally bleeding may occur further back in the nose and, trickling into the pharynx, may simulate hemorrhage from the stomach when vomited, from the intestine when passed with the stool, or from the lung when coughed up.

Occasionally urgent acute anæmia or even death may occur, especially when there is "blood dissolution" as in blood diseases.

Treatment.—The patient should be kept quiet, in the semi-erect posture. After douching the nose with hot or ice-cold water to which adrenalin 1 to 1,000 or tr. ferri chloridi has been added, the nose should be tamponed by Bellocq's catheter, or in less serious cases the patient should be enjoined not to blow the nose, for this only dislodges the forming clot. Gelatin may be injected into the nose, besides being injected hypodermatically. Internal remedies, as ergotin, are very unsatisfactory. Erosions or ulcers may be cauterized later.

DISEASES OF THE LARYNX.

ACUTE CATARRHAL LARYNGITIS.

Etiology.—Though acute catarrhal laryngitis is observed at every age and in all climates, most cases occur in males between twenty and forty, and in cold, windy, changeable weather. (*a*) *Cold* is frequently an apparent factor, but probably operates only by lessening the resistance, and this occurs to a greater extent in delicate than in robust subjects; the bacteriology of these "rheumatic" cases is unknown but its occasionally epidemic prevalence is suggestive of an infection. (*b*) Acute laryngitis may complicate *acute* (or chronic) *affections* such as measles, scarlatina, pertussis, typhoid, hay fever or syphilis and tuberculosis. (*c*) *Extension* may be caused *per contiguitatem* from the nose, pharynx or bronchi; oral breathing is a somewhat predisposing factor. (*d*) *Chemical,* thermal or mechanical agents, as dust, foreign bodies, trauma, overuse or abuse of the voice, alcoholism, inhalation of tobacco smoke, chlorine or bromine, and internal use of iodides are etiological factors. (*e*) *Stasis* from thoracic lesions or enlarged thyroid, local ulcerations in the larynx, nephritis, liver disease and rhachitis are also causes.

Symptoms.—A sudden onset, with fever, chill or rapid pulse, is seldom observed. The local symptoms are a sense of *rawness* or tickling, dry, even spasmodic cough, and moderate pain on breathing, as the larynx is hypersensitive; sometimes external tenderness or some dysphagia may occur, usually from coincident pharyngitis. After a day a thin, transparent and slightly cellular sputum appears, which later becomes purulent, yellow or in very rare cases, hemor-

rhagic. Some cases void no sputum (laryngitis sicca). The voice becomes *hoarse* or is wholly lost. The laryngoscope reveals diffuse or circumscribed redness, viscid mucus, some swelling and occasionally epithelial erosions. The false cords are often swollen. The true cords lose their smooth, glistening appearance and become injected and uneven, so that their even coaptation is hindered. The swollen mucosa may get between the cords, and their infiltrated muscles are often paretic. The acute symptoms last for a day or two and the catarrh remains about a week longer.

Diagnosis.—Diagnosis is usually easy; nervous aphonia and cordal paralysis are distinguished by means of the mirror. (See Vagus Disease, under Nervous System.)

Treatment.—Prophylaxis covers the etiological factors; alcohol and tobacco should be forbidden. The general resistance is increased by cold rubbings, douches or baths; mufflers should be avoided and cold compresses should be applied over the larynx. The voice must be used carefully and the nose, uvula and pharynx should be examined for predisposing lesions.

The therapy *of the established lesion* is as follows: At the onset sweating should be induced by hot drinks and by a hot bath; Dover's powder and quinine, each gr. x, or aconite, are indicated as in coryza; rest in bed is necessary. The voice must not be used. If the symptoms persist, an ice-bag should be applied locally; codeine or morphine with bromides, and inhalations of moist air or steam should be given, as described under Acute Bronchitis.

℞ Heroin hydrochloridi ..... gr. j.
Terpini hydratis ..... gr. xl.
Ammonii chloridi ..... ʒss.
Extr. glycyrrhizæ ..... ʒj.
M. et ft. capsulæ xxx.
S.—Two after meals.

Painting the larynx with a 3 per cent. silver nitrate solution is condemned by some physicians as promoting submucous extension.

In children acute laryngitis is often attended by great swelling above and below the cords and by stenotic manifestations in the small, infantile larynx. The symptoms closely approach those of genuine croup (laryngeal diphtheria), from which it is distinguished as false croup, *faux croup* (Guérsant) or *laryngite striduleuse* (Bretonneau). After an attack of mild laryngitis, coryza or sore throat, the child becomes restless and anxious, usually during the night when the secretion accumulates. Inspiratory dyspnœa develops, there is inspiratory retraction of the interspaces and epigastrium, the accessory muscles of respiration come into play, cyanosis appears and asphyxia seems imminent. The paroxysm subsides, spontaneously or after an emetic has been given, to recur on the next two or three nights. The catarrhal symptoms may last from three days to a week. Trousseau remarked that there is rarely that complete aphonia observed in

laryngeal diphtheria. The *diagnosis* is made from the history of coryza; the absence of diphtheritic patches in the pharynx, for diphtheria is eminently a descending infection; the absence of diphtheria bacilli, found in diphtheria even when the throat seems normal; and the absence of severe constitutional reaction. *Laryngismus stridulus* (*v.* Vagus Nerve) is not attended by fever, laryngitis or aphonia. The laryngoscope may reveal the simple œdematous laryngitis, but examination is notoriously difficult in easily frightened children. Treatment is the same as in acute laryngitis of adults; hot drinks, inhalation of steaming water, and ipecac should be given to induce vomiting and evacuation of laryngeal secretion; von Niemeyer awakened the patient at intervals and gave a hot drink to evacuate the mucus. Widerhofer also noted excellent results from washing away any pharyngeal mucus, which he considered causative of the attacks. The attendant should be prepared, in doubtful cases, for intubation or tracheotomy.

Other Acute Forms.—Other acute forms are (*a*) *laryngitis acuta nodosa,* which is observed in acute rheumatism and yields to salicylates. (*b*) *Laryngitis fibrinosa,* which is usually diphtheritic, but may be caused by a streptococcus infection in measles, scarlatina or other infections, and sometimes results from inhalations of steam or chlorine. (*c*) *Laryngitis submucosa acuta* invades the submucous tissues and is seropurulent. This rare affection may develop as an independent lesion, may follow a similar process in the pharynx or may represent a septic localization. During invasion the symptoms are severe, with fever, rigor, delirium and other septic phenomena. The laryngoscope shows injection and yellow-white bulging areas of submucous suppuration which, without operation, almost invariably cause death by œdema of the larynx or by sepsis, for spontaneous rupture is exceptional. For treatment (*a*) an ice-bag should be placed over the larynx, morphine given for pain and whisky for the sepsis; (*b*) incision of the foci is indicated; and (*c*) if necessary, intubation or tracheotomy should be performed.

CHRONIC CATARRHAL LARYNGITIS.

Etiology.—The etiology is practically identical with that of the acute form, of which it frequently follows repeated attacks; it is often associated with or is dependent on pharyngitis or rhinitis.

Symptoms.—The symptoms are those of the acute form, though less intense. *Hoarseness* alternates with fairly normal phonation. By the mirror there may be seen *injection,* darker than in the acute form, *swelling,* secretion and sometimes small hemorrhages. There are forms with no secretion (*laryngitis sicca chronica*); the secretion is rarely like that of ozæna, with glandular atrophy. In some forms granules appear, less from glandular hyperplasia than from proliferation of connective tissue (*laryngitis granulosa*). Erosions and rhagades may develop on the edges of the cords or arytenoid cartilages. Ulcers are most often syphilitic or tuberculous.

Treatment.—Treatment of the cause and prophylactic measures are the same as in the acute form. Inhalations of 1 per cent. tannic acid or of 1 per cent. turpentine and sod. bicarbonate in solution; insufflations of tannic acid, lead acetate and alum āā gr. i; and painting the larynx every third day with a 2 to 10 per cent. solution of silver nitrate with the aid of the laryngoscope, are valuable. Terpine hydrate gr. iij t. i. d. is often helpful. Cold water applied over the larynx, faradization and sea air may be beneficial.

Other Chronic Forms.—*Chorditis tuberosa* occurs as small grayish nodes on the cordal edges at the junction of their anterior and middle thirds, especially in singers and in alcoholics (Störk); they are composed of fibrous and epithelial hypertrophy. Scarification and lactic acid are indicated (v. TUBERCULOSIS).

Pachydermia laryngis (Virchow) is an increase of the submucous tissue and transformed epithelium into which connective tissue grows; it is circumscribed or diffuse. *Warty excrescences* are common between the arytenoids, and the thick mucosa is sometimes umbilicated and may resemble cancer. Topical application of salicylic or lactic acid, iodine in glycerine, electrolysis and the cautery are indicated. *Polyps* may result from chronic laryngitis.

Laryngitis hypertrophica inferior is a hyperplasia of the mucous and submucous tissue below the cords, and is identified by some clinicians with rhinoscleroma, which is due to the extension of the rhinoscleroma bacillus from above downward. It may cause stenosis of the larynx, which the author saw in one of Kolisko's autopsies. It is treated by scarification, dilatation, prolonged intubation, iodides, or, when extreme, by tracheotomy.

ŒDEMA OF THE LARYNX.

Œdema laryngis is a more proper designation than œdema glottidis, as the vocal cords are very seldom involved. It is most marked where the submucous tissue abounds and is greatest therefore about the epiglottis and ary-epiglottidean folds; it may develop under the cords. It varies with its cause, and may be bilateral, unilateral, diffuse or circumscribed. According to Sestier, 70 per cent. of cases occur in males and 89 per cent. in those over fifteen years.

Etiology.—There are *three etiological forms* besides some obscure cases. (*a*) *Inflammatory œdema* results from various inflammatory diseases of the larynx, submucous suppuration or perichondritis; from typhoid, syphilitic, tuberculous or cancerous ulceration; from iodides, trauma, surgical measures, thermic, chemical and allied causes; extension from pharyngitis, parotitis or cervical cellulitis; and from infections, as typhoid or measles. (*b*) *Congestive and hydræmic forms* result from chronic heart and lung disease, from tumors and nephritis, in which it may antedate œdema elsewhere, or from cachectic conditions as carcinosis or amyloidosis. (*c*) *Angioneurotic œdema,* as in R. S. Morris' fatal case, is the last type.

Symptoms.—Symptoms appear with varying acuity, sometimes most suddenly, as in Bright's disease, at other times more gradually but progressively, and again quite slowly. *Hoarseness* or aphonia, and a *metallic cough* are common. The chief sign is *inspiratory dyspnœa,* which characterizes nearly all forms of laryngeal stenosis. It is usually stridulous or wheezing; and is attended by retraction of the interspaces, cyanosis, up-and-down excursion of the larynx and ultimate suffocation. The mirror shows a pallid swelling in the parts enumerated, incision of which allows the escape of a clear serum. Œdema of the epiglottis and ary-epiglottidean folds may be felt with the finger if the mouth is held open and the tongue drawn forward, or it may be seen even without the mirror if the larynx is pushed up and the tongue deeply depressed.

Treatment.—Treatment is urgent, irrespective of the cause, for the large mortality (75 per cent.) is due to delay until the patient's strength is exhausted. The placing of ice over the larynx, the swallowing of ice, drastic purgatives and painting the larynx with 10 per cent. silver solution are at best palliative. The œdematous tissue should be fully incised, and, in case this should fail, intubation or tracheotomy should be performed.

PERICHONDRITIS LARYNGIS.

Purulent inflammation beneath the perichondrium of the laryngeal cartilages is very seldom primary, but nearly always results *secondarily* to various inflammations or ulcerations. Pus accumulates between the perichondrium and cartilage, most frequently the arytenoid or cricoid, or less frequently the thyroid or epiglottic. The cartilage may become secondarily necrosed. Perichondritis laryngis most often develops in males between twenty and forty years of age.

Symptoms.—Its symptoms are commonly obscured by the causal disease. The rare primary cases are the most acute. Well localized pain and tenderness are peculiarly significant. *Dysphagia,* especially from arytenoid involvement, is frequent, and even aphagia may result. *Phonation is impaired* by the fundamental disease, by cord paresis and muscular infiltration. The cervical glands sometimes tumefy. *Stenosis* and *dyspnœa* are present. The *mirror* discloses focal accumulation of pus, swelling, redness, œdema and perhaps fistulæ which signify intralaryngeal rupture. The probe may elicit tenderness or may scrape on eroded cartilage. The *diagnosis* from laryngitis and œdema is often impossible, or at best but probable unless definite pus pockets are seen. The *outcome* may be suddenly fatal from rapid œdema; pus and cartilage fragments may rupture, thus giving relief or perhaps occluding the larynx; fistulæ, internal or external, may remain, and may burrow to the mediastinum, or sepsis may follow; aspiration pneumonia is not uncommon, and if immediate recovery ensues, a distorted or stenosed larynx may remain.

Treatment.—Treatment is similar to that of acute laryngitis; the

pain should be relieved by opiates, but early incision under cocaine is necessary, and the physician should be prepared to perform tracheotomy or intubation.

ULCERATIONS AND NEOPLASMS OF THE LARYNX.

The tuberculous and syphilitic ulcers have been considered. Superficial erosions may follow catarrh; deep ulcers result from submucous suppuration or from perichondritis, pressure ulcers from foreign bodies or laryngeal "bed sores" (*v.* TYPHOID) which are ulcerations analogous to intestinal ulceration. There are also the specific ulcerations of leprosy, glanders, etc.

Tumors include benign single or multiple papillomata located on the true cords (singer's nodes *v.s.*), fibromata, cysts, amyloid accretions, enchondromata, lipomata and the malignant tumors, of which carcinoma is far more frequent than sarcoma. Primary *cancer* develops slowly on the posterior third of the cords or in other locations as an excrescence which gradually assumes a cauliflower appearance; the basis of the later ulceration is nodular and there is no scar formation (*v.* SYPHILIS, LARYNX); hoarseness is an early symptom; all kinds of incipient tumors are frequently mistaken for laryngitis. Pain which may radiate to the ears or temples, cough and dysphagia are constant in the advanced stage. Early operation, either intralaryngeal or by splitting the thyroid, will alone afford relief; inoperable cases must necessarily be narcotized.

Nervous aphonia, the *paralyses* and *spasms* of the larynx are considered under affections of the vagus nerve.

DISEASES OF THE TRACHEA AND BRONCHI.

The *trachea* is seldom involved alone. Tracheitis is frequent with inflammation of the larynx and bronchi. Hemorrhage may result from diapedesis or from ruptured varices. Tuberculosis, syphilis and tumors are very infrequent.

ACUTE BRONCHITIS.

Acute bronchitis is a most frequent primary or secondary disease, in some localities constituting 10 to 15 per cent. of all diseases. It occurs most frequently in cold, dusty or variable weather; three-fourths of all cases occur between December and April. Its *forms* are (*a*) inflammation of the *larger air tubes*, bronchitis, tracheobronchitis or macrobronchitis, and (*b*) inflammation of the *smaller tubules*, known as bronchiolitis, capillary bronchitis or microbronchitis, which in

children is identical with bronchopneumonia (*q. v.*). Badham (1819) first used the term bronchitis but the condition was first described by Laennec, who remarked everything essential up to its bacteriology.

Etiology.—(*a*) It is a *symptom of various infections,* as *measles, pertussis, influenza* and *typhoid;* it also occurs frequently in scarlatina, pneumonia, diphtheria, syphilis, tuberculosis, sepsis, etc. In these maladies bronchitis is secondary and results from the germs or their toxins. (*b*) *Cold and bacteria* are also etiological factors. It is disputed whether cold alone is an adequate factor without the cooperation of bacteria, but it at least plays a potent predisposing part. Even in cases of apparently primary bronchitis the pneumococcus, pyogenic cocci, Friedländer's pneumo-bacillus and other bacteria are found (*v. s.*). Pfeiffer's *micrococcus catarrhalis* is found in 59 per cent. of cases (Ghon, Pfeiffer); it is larger than the staphylococcus and often occurs in pairs. (*c*) It may result from the *toxic effects* of potassium iodide or bromide, tobacco, less often carbolic acid, cantharides, mercury and alcohol which according to Hoffman, acts locally on the air passages. (*d*) *Mechanical irritants* are dust and other foreign particles, as particles of wool, stone, etc., which exist in many factories; overuse or abuse of the voice is also a mechanical irritant. (*e*) *Constitutional conditions,* as obesity, nephritis, rhachitis, gout, diabetes and cachexia of various kinds, such as cancer, syphilis or scurvy, are important etiological conditions. (*f*) *Venous stasis* in the lesser circuit favors bronchitis, by congesting the pulmonary veins, which empty chiefly into the vena cava superior and vena azygos; *mitral lesions* are the most important but other valvular disease, arteriosclerosis, scoliosis, abdominal tumors, ascites or pleurisy, all of which affect the diaphragmatic excursion, are possible factors. (*g*) *Respiratory diseases,* as pneumonia, tuberculosis, abscess or gangrene of the lung, asthma and bronchiectasis are usually associated with bronchitis. (*h*) *Age.* Acute bronchitis may develop at any age, but is peculiarly frequent in the *aged* and in *children* between six months and three years old.

Symptoms.—1. Acute bronchitis of the larger tubes (*macrobronchitis or tracheobronchitis*) begins (*a*) with some constitutional *depression,* sometimes with fever and chilliness, or in children even with marked toxæmic symptoms. It often follows acute coryza or pharyngitis. (*b*) The *local symptoms* are *substernal rawness* or tickling; actual pain or tenderness over the sternum is mostly tracheal or muscular, for the bronchi have apparently no pain nerves. On inspection of the trachea there is visible inflammation, redness, diffuse or circumscribed injection, submucous ecchymosis, swelling and laxness of the mucosa, and dryness, which is followed by the appearance of a viscid, transparent, glairy mucous secretion; the mucous glands are often distended by globules of mucin which slightly resemble young tubercles; most of these findings are also noted in the bronchi in the rare cases which come to autopsy, but injection and redness

largely disappear after death. Microscopically the cardinal signs of inflammation are found, such as paresis of the vessels, round cell emigration and desquamation of the ciliated epithelium. (*c*) The *cough* is at first dry and unproductive and may remain so. Later a viscid, transparent sputum is raised, consisting almost wholly of mucin and very few white cells (*sputum crudum*). A day or two later the coughing efforts become less severe as the sputum grows more abundant and mucopurulent (*sputum coctum*); it contains polymorphonuclear and sometimes abundant eosinophilic leukocytes, epithelial cells showing myeloid degeneration, and rarely ciliated epithelium. Localized inflammation at the tracheal bifurcation causes special irritation (Nothnagel). (*d*) The *symptoms resulting from cough* are disturbed sleep; hoarseness from forcible closure of the glottis in coughing efforts; pain in the side or at the diaphragmatic insertion, from unusual strain on the expiratory muscles during coughing; venous stasis which induces headache, vertigo, swollen cervical veins, cyanosis and epistaxis; mechanical sequences, as vomiting, involuntary urination, especially in weak and old subjects; and in women, even abortion, uterine prolapse or hernia may occur. (*e*) *Physical findings* are usually *bilateral;* unilateral signs, though possible, suggest other lesions, especially tuberculosis. Auscultation reveals *rhonchi,* which result from swelling of the bronchial mucosa, slight stenosis by bronchial secretion and air passing through or dislodging the secretion. The rhonchi at first are dry (*r. sicci*), and later moist (*r. humidi*). Over the large tubes these rales are sonorous, in the smaller tubules they are sibilant, and in both they vary acoustically. They may be heard simultaneously over the large and small passages, or over the large and then, by extension, over the small tubules. These rales, large and small, may be felt with the hand (mucus fremitus) or may be heard without the stethoscope. The breathing may be "cog-wheel," lengthened, slowed or sometimes increased by fever, but in quality it is only exaggerated or puerile, never bronchial. Thoracic breathing develops in men when the tubes in the lower lobes are invaded (Riegel). Percussion is wholly negative. Macrobronchitis lasts a few days, possibly longer than a week.

2. Microbronchitis or capillary bronchitis is essentially the same pathological process as macrobronchitis but will be considered under bronchopneumonia, because of its great, though not necessary, tendency toward pulmonary inflammation, especially in the aged and in children.

Treatment.—In the early stage a *full hot bath* may be given, followed by a *hot alcoholic drink.* A hot Turkish bath should never be taken, for exposure afterward is dangerous. *Hot fomentations* over the sternum are beneficial; a hot water bag or a large poultice may be applied over the sternum, but cold packs over the neck and chest are even more efficacious. Patients should always be kept in bed, for but few cases recover promptly without this treatment. A drink or two of well diluted whiskey is valuable. When the

initial symptoms appear, gr. x of *Dover's powder* should be given; it not only relieves pain, muscular soreness and coughing but also appears to modify the inflammation; in children it should be given with great caution because of the frequently unequal mixture of its constituents, and the peculiar susceptibility of the youthful nervous system to narcotics; in the aged it should likewise be administered with care, because the kidneys in old persons excrete slowly, and cerebral atheroma may induce psychical disturbance, Cheyne-Stokes' breathing and other basilar disturbances. The administration of opiates should be followed by some saline aperient. Other treatment is frequently superfluous, but if the symptoms and cough persist or if the initial symptoms are rather severe we administer:

℞ Vini antimonii ..... ℥iv.
Heroin hydrochloridi ..... gr. iss.
Vini ipecacuanhæ ..... ℥v.
Syr. tolutani .....q.s. ad ℥ij.
M. et S.—One teaspoonful every three or four hours. The physician should watch lest gastric symptoms appear.

Brown's mixture may be given (see CHRONIC BRONCHITIS).

℞ Potassii citratis ..... ℥iiss. ℥j.
Codeinæ sulphatis ..... gr. iv. gr. j.
Syrupi ipecacuanhæ ..... ℥iv. ℥iss.
Succi limonis ..... ℥j. ℥ss.
Aquæ .....q.s. ad ℥ij. ℥ij.
M. et S.—One teaspoonful every two hours. The first column is the dosage for adults, the second for children.

The fever rarely requires treatment; if high, aconite and belladonna may be given, as in coryza, but tepid sponging is more beneficial. A kettle of water (with co. tr. benzoin ℥i–Oj) should be kept boiling in the room to moisten the atmosphere, or a steam tent may be advisable as in croup (see DIPHTHERIA). If the bronchitis lingers longer, the formulæ given under chronic bronchitis will be useful.

CHRONIC BRONCHITIS.

Etiology.—The etiology is essentially that of the acute type. Chronic bronchitis is more common in advanced life, and may develop slowly as a chronic process, or, more often, follows repeated acute attacks. It most often develops on change of weather or in the cold season, as the "winter cough."

Pathology.—The larger and medium sized bronchi are most involved. Their color is a gray red or brown; the bloodvessels are thickened, and the bronchi become thicker from extravasation of leukocytes and proliferation of the peribronchial connective tissue; the mucous membrane is sometimes hypertrophied, and even protrudes in polypoid and papillary excrescences, or is atrophied, and appears thin and pale like a serous membrane. Erosions are not common

but are sometimes seen at the tracheal bifurcation, especially in the aged. Stagnation of secretion is common. The inner surface of the bronchi is at times reticular, due to the prominence and hyperplasia of some elastic fibers and the wasting and sinking of other elastic or muscular fibers. Emphysema and some interstitial fibrous overgrowth are frequently found at autopsy.

Symptoms.—Fever and pain are absent, except in the frequent acute exacerbations. The usual intrinsic physical findings in *chronic macrobronchitis* are *large sonorous rales,* and sibilant rales when acute exacerbations cause invasion of the finer tubules. The cough varies with the intensity of the inflammation, with the amount and character of the secretion, or with changes in the weather. The sputum varies greatly, and upon its character is based the division into the following forms: (*a*) *Dry bronchitis,* the *bronchitis sicca* of Laennec, which is seen in the aged and is almost always associated with emphysema; in this type the sputum is wholly absent or very scant and viscid. (*b*) *Bronchorrhœa,* in which the excessive bronchial secretion may be mucous, serous or purulent; in the serous form the secretion is thin and transparent, as in Laennec's case, in which two quarts were raised daily for twelve years; Strümpell observed a serous bronchorrhœa resulting from enlarged peribronchial glands; the purulent form may be confused, on account of its abundant sputum, with bronchiectasis, lung abscess or rupture of an empyema into the bronchi. (*c*) *Putrid bronchitis,* which has been described by Laennec, Andral and Traube. It may complicate chronic bronchitis but occurs more often with bronchiectasis, foreign intrabronchial bodies and tuberculosis; in this form the sputum stinks and is acidulous, particularly when first voided. It consists of three layers; an upper foamy, a middle serous and a lower one of granular gray-green or bluish sediment, in which are the *mucotic plugs* of Dittrich (1850); these plugs contain various bacteria, leptothrix, strepto- and staphylococci, etc. "Acid-fast" bacilli are found in putrid bronchitis and in gangrene of the lung; they closely resemble tubercle bacilli, though they are narrower, more pointed at their ends and do not give inoculation results. Fat and myelin droplets, fatty needles, leucin and tyrosin are also present. Fever is common, "drum stick" fingers may develop, and complications may follow, as lung induration, diphtheroid ulceration, gangrene, pleurisy, hæmoptysis, cerebrospinal abscess, meningitis or pseudorheumatism. (*d*) *Blood-stained sputum* occurs particularly in the brown induration of the lungs, due to stasis (see page 408).

Chronic emphysema (*v. i.*) is an almost regular complication and is marked by a tympanitic percussion note, voluminous lungs, prolonged and, on exertion, difficult expiration, weakened vesicular murmur, hypertrophy of the cervical muscles, right heart dilatation, and eventually tricuspid insufficiency, liver congestion and œdema.

Diagnosis.—The diagnosis is generally easy. Determination of its relation to tuberculosis and other causal factors is most important. In every case chronic nephritis should be considered.

Prognosis.—The prognosis depends on the etiological factors. Absolute recovery is most unlikely.

Treatment.—1. Prophylaxis. Causal affections require treatment. Woolen underwear should be worn to protect against sudden changes of temperature.

2. Climate.—When feasible, a dry, warm, even climate is beneficial, as that of Florida, southern California, New Mexico, the Carolinas, Egypt and the Riviera.

3. Cough.—Codein, morphine, creosote, cod-liver oil and other remedies are employed as in tuberculosis (*q. v.*). Ammon. carb. (*v.* Pneumonia) gr. iij can be given with syr. senegæ ʒj; syr. scillæ ʒj is incompatible with ammon. carb. but may be combined with ammon. chlor. gr. v to x.

℞ Ammonii chloridi ..... ʒiss.
Misturæ glycyrrhizæ compositæ ..... ʒiv.
M. et S.—One teaspoonful after meals.

The mistura glycyrrhizæ composita (Brown's mixture) contains twelve parts of paregoric, six parts of antimonial wine and three parts of sweet spirits of nitre, which enhance its sedative action; it may be given in doses of from one to four drams. If the heart is weak, it is contraindicated because of the antimony it contains. From all cough preparations, the stomach may become deranged by the ammonium, syrups, etc. Chloroform is particularly valuable in paroxysmal unproductive coughs, which opiates may not alleviate because they dry up the secretion.

℞ Ammonii carbonatis ..... ʒj.
Spiritus chloroformi ..... ʒiss.
Syrupi senegæ .....q.s. ad ʒiij.
M. et S.—One teaspoonful in hot water as indicated.

℞ Apomorphinæ hydrochloridi ..... gr. ss.
Heroin hydrochloridi ..... gr. j.
Spiritus chloroformi ..... ʒj.
Aquæ .....q.s. ad ʒij.
M. et S.—One teaspoonful two or three times a day.

Syr. scillæ compositus (Hive Syrup) also contains senega and tartar emetic and is given in doses of gtt. xx to xl. The antimony it contains may depress the heart. Terpini hydras is excellent, gr. iii t. i. d., given in capsules with equal amounts of benzoic acid. The *balsams* are even more efficacious, as *emulsum ol. terebinthinæ* ʒj in milk; ol. santali ♏x to xx in emulsion is less likely to disturb the kidneys than is turpentine; *balsamum Peruvianum* ʒss ranks with the best. The balsams and creosote are directly indicated in fœtid bronchitis. Retention of secretion which does not yield to the action of drugs, may be treated by rhythmical compression of the chest during expiration. Potas. iodide gr. v to x t. i. d. is excellent.

4. Cardiac Weakness.—Cardiac weakness is relieved most efficaciously by strychnine, which is more valuable than digitalis,

5. Coincident Emphysema.—Coincident emphysema and asthma (*q. v.*) necessitate the prolonged use of iodides and belladonna.

FIBRINOUS BRONCHITIS.

Etiology.—There are three types of fibrinous or croupous bronchitis. (*a*) The *primary* form is an infrequent disease, of which McPhedran collected 204 cases (1907); it occurs in males (66 per cent. of cases) principally between ten and thirty years of age, and in weakly and anæmic subjects; it results from the Bacillus diphtheriæ, pneumococcus, strepto- and staphylococcus, aspergillus fungus and protozoa, or from their toxins. (*b*) The *secondary* type develops by extension from laryngeal and pulmonary disease, or in infections, as diphtheria, pneumonia, typhoid, measles and scarlatina; tuberculosis causes 50 per cent. of this group; some cases occur in skin disease. (*c*) *Mechanical, thermal* and chemical agents are causative factors in a few cases, and some have occurred in connection with valvular heart disease and exophthalmic goitre. Fibrinous bronchitis was first clearly described by Clark, in 1697, though Hippocrates and Galen mentioned the condition.

Symptoms.—The early symptoms are those of bronchitis, sometimes with fever, chills and hæmoptysis (in 33 per cent.), but they are ambiguous until bronchial casts of fibrin or mucin are expectorated, after a severe coughing paroxysm. The casts may measure even eight inches long, and may be as thick as a lead pencil or a finger; their upper and larger parts are circular or somewhat flattened, solid or hollow, but the lower small portions are branched, solid, spiral and often beaded with air and clubbed at their tips. Though usually termed fibrinous or croupous, many casts do not take Weigert's fibrin stain; these are bronchitis mucinosa or pseudomembranacea. Liebermeister holds that fibrin is more frequent than mucus formation. The microscopic appearance of the casts is hyaline, their texture is fibrillar, and their surface is often covered with blood disks, hæmatoidin or Charcot-Leyden crystals. Smaller casts may not be seen until the sputum is agitated in water, which washes off the adherent blood, pus or mucus. Pending their evacuation, there are *signs of unilateral bronchial stenosis, e. g.,* dyspnœa, cyanosis and inspiratory retraction, and absence of breath sounds in the corresponding lung area. Slipping or moving of the casts in the bronchus may cause peculiar flapping rales. After their evacuation new casts form in one or more days. Niemeyer observed the daily shedding of a complete cast of a bronchus and its divisions.

Diagnosis.—In some cases casts are not voided during life, so that the autopsy alone determines the diagnosis. Asthma (*q. v.*) and bronchitis fibrinosa have certain common symptoms, as paroxysmal occurrence, eosinophilia, crystals and spirals, but are usually differentiated with ease.

Course and Prognosis.—In *the acute form,* which lasts two weeks or

less, there is great risk of sudden asphyxia during the evacuation of the casts; there is a death rate of 75 per cent. in the young and 50 per cent. in adults. In *chronic forms* the process lasts for months or even years (cases of twenty-three and twenty-five years duration have been observed). The prognosis is uncertain. Complications include splenic tumor, pneumonia, pleurisy, emphysema and albuminuria.

Treatment.—Treatment is unsatisfactory if not wholly futile. Inhalation of steam and alkaline vapors, and pilocarpin should be given in order to loosen the casts. Emetics may facilitate their evacuation and iodides and mercurial inunctions may inhibit their reformation. The iodides are the most effective.

BRONCHIAL DILATATION, BRONCHIECTASIS.

Etiology and Pathology.—Laennec first described bronchiectasis, in 1819. It is found in 2 per cent. of autopsies and is *always secondary* (*a*) to *bronchial disease, stenosis* preëminently, and then influenza, pertussis, inhalation of dust, foreign bodies or pressure from aortic aneurysm; (*b*) *to lung disease,* tuberculosis, interstitial or bronchopneumonia, hypostasis or atelectasis; in these conditions coughing increases the expiratory pressure, even forty fold; (*c*) to *pleural disease,* such as adhesions pulling on the bronchi. Nearly all cases are *acquired* and occur mostly in middle aged males (77 per cent.). Rare congenital cases are recorded. These may be divided into (i) the unilateral, universal or diffuse form (Grawitz), (ii) those due to congenital atelectasis (Heller) or atrophy and (iii) those resulting from prenatal bronchial (syphilitic) stenosis.

There are *two forms* of bronchiectasis: (*a*) The cylindrical or fusiform, multiple or *pater noster* form, which develops from the atrophy of the muscular and elastic fibers induced by protracted severe coughing and perhaps by stagnation of secretion; it may follow measles, catarrhal pneumonia, whooping cough or emphysema. (*b*) The rarer *sacculated form,* in which the bronchus may reach a diameter of even 3 inches and which might be called a bronchial aneurysm. The connective tissue usually disappears, the ciliated epithelia are replaced by flat cells and the mucosa is thin, red, swollen and sometimes villous or varicose. The sacs close at their necks, particularly when caused by aneurysm, syphilis or tuberculosis; this converts them into sacs filled with gelatinous, cheesy or calcareous contents. Lung stones may develop, of calcium carbonate and phosphate with traces of magnesia; they have an organic nucleus and sometimes cause "bronchial colic," or symptoms like phthisis.

Bronchiectasis is seldom single; in multiple bronchiectasis the lung sometimes resembles a sponge or porous cheese. It occurs principally in the lower lobes and in bronchi of the third and fourth order. It is bilateral in 60 per cent. but the congenital forms are always unilateral. Adhesions are very common when the cavity reaches the pleura.

Symptoms.—Most cases are not discovered clinically, and Weil therefore concluded that "most cavities escape detection and most diagnoses of cavities are incorrect." Sacculated forms produce more symptoms and signs than do the cylindrical forms, which are almost always impossible to recognize.

1. Sputum and Cough.—The sputum and cough are paroxysmal. When secretion fills the sac and flows over onto the normal sensitive mucous membrane beyond it, cough develops and quantities of mucopus are voided in the "*mouth full" fashion* described by Wintrich. Change of posture produces similar effects. The largest amount is raised in the morning and is frequently followed by vomiting. The daily quantity may amount to a quart. Its odor is acid, sweat-like and offensive, but it does not attain the fœtor of putrid bronchitis or the mature foulness of gangrene. When collected it forms in three layers; the upper one is foamy, the middle serous and the lower purulent. A nummular or globular sputum may be noted, as in tuberculosis, but if put in water it is more flocculent, more granular and airless; and microscopically it contains altered leukocytes and fatty needles, occasionally red cells, hæmatoidin crystals and very rarely, when the bronchial wall is ulcerated, elastic fibers.

2. Physical Examination.—(*a*) *Inspection* may reveal diminished respiratory movement, inspiratory retraction over the cavity or flattening of the chest wall. The patient generally leans toward the diseased side, to lessen the trickling out of the secretion. In the rare cases of bronchiectasis in an upper lobe the constant escape of the secretion results in exhausting inauspicious coughing (Skoda). The *x*-rays may show the location of the cavity. In rare cases the cavity may bulge through the interspaces. (*b*) *Palpation* and (*c*) *percussion* give variable results depending on whether the cavity is full or empty. Dulness prevails when it is full; when it is empty a tympanitic note results. A higher note on opening the mouth, on inspiration or on change of posture has the same value as in tuberculous cavity (*q. v.*). (*d*) On *auscultation, bronchial* or *metallic breathing* may be heard when the cavities are superficial but it is absent in dilatations which are small, deep or full of secretion. *Metamorphosing breathing* is a certain sign of a cavity. Skoda's superficial, "veiled-puff" sound is frequent. *Large* and *small rales,* if heard persistently in the same locality, are suggestive of a deep bronchiectasis. The sound tissue over the cavity imparts a metallic consonance.

3. Complications.—The general nutrition sometimes remains normal; slight cyanosis together with anæmia is frequent. Hectic fever is often absent, but death may result, with symptoms resembling those of phthisis. Putrid bronchitis is frequent. Hæmoptysis from ulceration of varices occurs in 40 per cent. of cases; pneumothorax, pleurisy, empyema, lung induration or gangrene, emphysema, lobular pneumonia, amyloidosis and hypertrophy and dilatation of the right ventricle are fairly common. Marie's *ostéo-arthropathie hypertro-*

phiante pneumonique, rheumatoid affections (Senator), suppurative meningitis or abscesses in the brain are occasional.

Diagnosis.—Diagnosis depends upon the character of the sputum, its paroxysmal voidance and the signs of cavity formation. *Differentiation* is required (*a*) from *tuberculous cavities;* this is made by means of the habitus, presence of elastic fibers and tubercle bacilli in the sputum, which are definitely determined by inoculation; tuberculous cavities are most often apical, but frequently bilateral; they void less sputum and suffer less rapid variation in their physical signs. Bronchiectatic cavities occur mostly in the lower lobes, void more sputum, less often induce hemorrhage and more frequently cause right heart changes. (*b*) From *perforating empyema,* in which cholesterin and hæmatoidin crystals are far more frequent than in bronchiectasis; (*c*) from *abscess;* (*d*) from *gangrene* of the lung (*v. i.*); (*e*) from *putrid bronchitis,* in which there is no sudden volume of evacuated sputum or signs of cavity; (*f*) from *encapsulated pyopneumothorax,* which may be very difficult of differentiation; and (*g*) aneurysm or tumor causing bronchiectasis.

Prognosis.—The immediate prognosis is rather favorable, but ultimate recovery is exceptional, except in acute cases, *e. g.,* postinfluenzal; patients have lived half a century. The complications require consideration (*v. s.*).

Treatment.—The therapeutic indications are (*a*) *evacuation of the secretion,* for which expectorants and expiratory compression of the chest, as in bronchitis, are somewhat useful; sedatives should never be given, lest gangrene develop; (*b*) modification of *putrescence* by balsamic remedies, as in fœtid bronchitis; inhalations of creosote may be given, though they are most irritating at first (Chaplin, 1895); a dram of creosote is slowly vaporized over a flame, and the patient, in a closed room, inhales the fumes with the eyes closed and the anterior nares plugged; the procedure may be gradually lengthened from a few minutes to half an hour; the irritation evacuates the residual secretion; (*c*) *surgical drainage.* An accessible cavity may be incised and drained, though accurate diagnosis of its location is difficult, the mortality high and the affection multiple. In Garré's collection of 57 cases (1902), 63 per cent. recovered and 37 per cent. died; (*d*) *change of climate* (*v.* Tuberculosis).

TRACHEAL AND BRONCHIAL STENOSIS.

I. Tracheal Stenosis.—Etiology.—The cause is less often within than without the trachea. *Tracheal causes* are rhinoscleroma (Störk, 1874), syphilis which in 40 per cent. of cases also implicates the bronchi, tumors (cancer, polyp, fibroma, enchondroma, sarcoma or aberrant thyroid), foreign bodies, tracheotomy granulations, and in rare cases perichondritis. Causes *outside the trachea* are more frequent, as thyroid enlargement, particularly the retrosternal, cystic and circular forms, or struma in which hemorrhage or inflammation

has occurred; in some cases sudden death results, notably when the cartilages have been absorbed. Aneurysm, adenopathies, mediastinal tumors, vertebral tumor or caries and thymus hyperplasia are other factors. Thiesen (1906) collated 135 cases of tracheal tumor, of which 89 were benignant and 46 malignant; 24 were fibromata and 10, intratracheal struma (penetrating the rings of the trachea, Paltauf).

Symptoms.—The symptoms may be ranged in *three series or stages;* (*a*) when there are no symptoms or when they occur only on exertion; (*b*) when there is constant dyspnœa, and (*c*) when suffocative attacks terminate in final asphyxia. The *dyspnœa* is of a mixed type, *i. e.,* inspiratory and expiratory; the number of respirations is lessened, there is inspiratory stridor and intercostal retraction, all the accessory muscles come into play, the head is held well forward and there is little or no laryngeal excursion. The pulse is more tense, varies in rate and is sometimes of the paradoxical type. Tracheoscopy may reveal the location of the lesion, most advantageously in the strong sunlight; the patient should stand with his back to the light and with the head bent forward, and the observer should be seated on a low seat before him. Fränkel affirms that in 25 per cent. of tracheal stenoses due to tumor or aneurysm, there is coincident paralysis of the vocal cords.

II. Bronchial Stenosis.—**Etiology.**—*Intrabronchial* causes are most common and consist of pus, mucus, blood, fibrin and foreign bodies; obstruction due to the latter is laryngeal in 33 per cent., tracheal in 26 per cent. and bronchial in 41 per cent., of the cases. *Extrabronchial* factors are next in frequency; aneurysm, mediastinal lymphosarcoma and perilymphadenitis leading to fibrosis occur most often; less common are pericarditis with effusion, dilatation of the left auricle, cancer of the œsophagus, tumor of the lung and thyroid or thymic strumas. Twenty cases have been reported in which lymph glands ruptured into the trachea or bronchi, causing suffocation; only three were saved by tracheotomy. *Interbronchial causes* (*i. e.,* in the bronchial wall) are the least common, as syphilis, glanders, rhinoscleroma and tumors. Thirty-six cases of primary cancer are recorded. The right is stenosed nearly twice as often as the left bronchus.

Symptoms.—Occlusion of a bronchus lessens the amount of air entering the lung which it supplies, whence the *vocal fremitus* and *breath sounds* are immediately *weakened* or suspended and the respiratory excursion and Litten's diaphragmatic sign are decreased. There is inspiratory retraction of the intercostal spaces on the affected side. *Inspiratory dyspnœa* occurs, particularly in acute cases. The head is thrown forward and the larynx moves up and down but little. The percussion note is not essentially altered; and this excludes many causes of dyspnœa, as pneumonia, etc.; after a time a somewhat tympanitic note may result from relaxation of the lung or acute emphysema. A local stenotic murmur over the bronchus is present in a few cases. Paroxysmal dyspnœa may result from stagnating secre-

tion. Coughing is usual and may resemble that of pertussis. The pulse is more tense and often slower. The *x*-rays may locate the obstruction. Bronchoscopy is sometimes helpful, but it is difficult to perform even for the experienced specialist. Death results from (*a*) the original disease, as aneurysm or tumor; (*b*) failure of the right heart, (*c*) asphyxia, with delirium, cyanosis, convulsions or Cheyne-Stokes' breathing, or (*d*) pulmonary œdema, abscess, gangrene, tuberculosis, lobular pneumonia or hemorrhage.

Diagnosis.—(*a*) *Of the location* of stenosis: In laryngeal obstruction there are local findings and free up-and-down laryngeal excursion; the subject holds the head well back, and the ingress of air to both lungs is equally impaired; tracheoscopy, bilateral lung involvement, the absence of laryngeal excursion and the holding of the head forward are sufficient proof of tracheal obstruction; in bronchial stenosis but one lung suffers for air, the larynx is nearly immobile and the head is thrown forward. (*b*) *Of the nature* of stenosis, exact diagnosis is more difficult, and is impossible without distinct physical signs, as those of aneurysm, or without history of the presence of foreign bodies.

The prognosis depends on the cause.

Treatment.—Treatment is also largely that of the cause, as syphilis, tuberculosis, etc. Thyroid extract and iodine are efficacious in over 50 per cent. of cases of thyroid swellings. When the obstruction is aneurysmatic, rest, mercury, iodides, venesection and gelatin injections often afford considerable relief. When there are foreign bodies, the patient should be inverted, sharply struck on the back and shaken; the physician should have long forceps and tracheotomy instruments in readiness. The mortality in foreign bodies of the lower air passages is 52 per cent. with expectant treatment, and 23 per cent. with operation.

Bronchial and lung stones (*v. s.*) result from tuberculosis of the bronchial glands, from bronchiectasis, petrification of the bronchi, calcification and ossification of the lungs, gout and foreign bodies. Boerhaave described the case of the botanist, Vaillant, who expectorated 400 calculi. Symptoms are frequently absent or are those of the primary disease; purulent bronchitis, asthma ("bronchial colic"), hectic fever and hæmoptysis mark some cases.

Stenosis of the bronchioles may result from *bronchiolitis fibrinosa obliterans* (Lange and Fränkel).

BRONCHIAL ASTHMA.

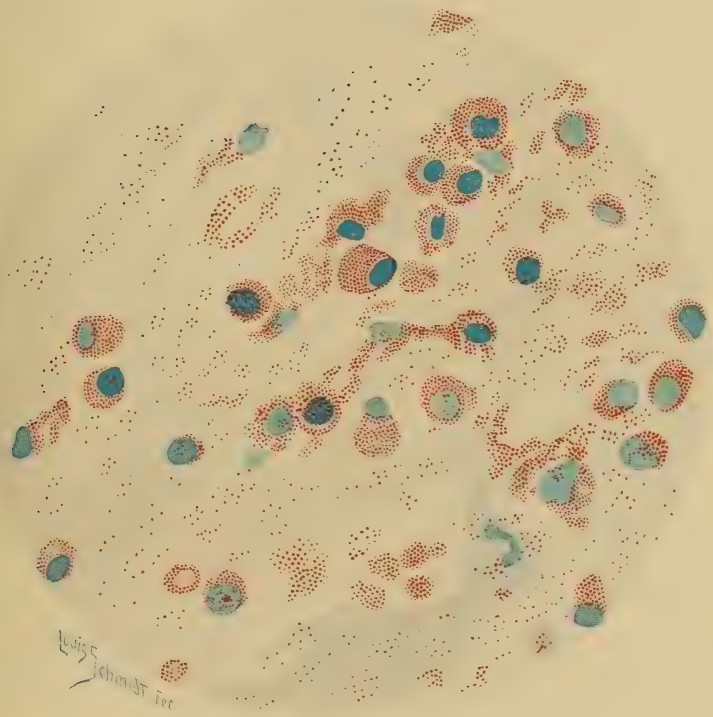
Definition.—A paroxysmal dyspnœa, due to stenosis or spasm of the smaller bronchioles, which is nervous or reflex in origin and is characterized by expiratory dyspnœa, slowing of respiration, congestion of the bronchioles, a peculiar mucous exudation and acute emphysema. It is also known as asthma spasmodicum or nervosum, to distinguish it from other so-called asthmas, as the cardiac or renal types.

Etiology.—When due to no obvious cause, asthma is termed essential or *primary,* and when due to a clear cause it is called *symptomatic* or reflex asthma (which occurs through the vagus). (*a*) *Nasal* and nasopharyngeal disease is the source of the reflex asthma in numbers of cases, as proven by Voltolini, Hack, Hänisch, Porter, Daly, Roe and others; it may be caused by rhinitis, tonsillar hypertrophy, adenoids or pharyngitis granulosa; the nose may be so hypersensitive that the odor of flowers, dust, hot air or smoke precipitates an attack. (*b*) Asthma may be initiated by *pressure on the vagus* trunk, by thyroid tumors or enlargement of the bronchial glands, following tuberculosis, measles, pertussis or rickets. (*c*) *Abdominal diseases* are possible though not frequent causes; the asthma dyspepticum of Henoch, Meyer and Pribrâm has been especially emphasized of late by Hayem who describes asthma resulting from gastric dilatation. Ovarian and uterine disease and pregnancy have been considered causal factors. (*d*) *Asthma toxicum* has been reported in lead and mercurial poisoning; *uræmic* and gouty asthma are due to toxins. (*e*) It occurs especially in weakly, anæmic, nervous, scrofulous and rhachitic subjects, in males (66 per cent.) between the ages of twenty and forty (though it has recently been claimed that 33 per cent. of the asthma cases develop under ten years), in the upper classes and in professional men.

There are various theories as to the *mechanism* of the asthmatic attack: (*a*) It is a *spasm* of the *bronchial muscles* (Trousseau, Rhomberg and Biermer). Trousseau spoke of asthma as "an epilepsy of the lungs"; (*b*) it is a very acute catarrh (Louis and Traube); Störk found the larynx and trachea deeply injected; (*c*) it is a *vasomotor* disturbance (Weber); or (*d*) an acute exudative bronchitis, causing bronchial spasm (Curschmann, Lebert). Its pathology is uncertain, as but half a dozen autopsies are recorded; the ciliated epithelium is desquamated, the bronchioles are congested and there is eosinophilic exudation.

Symptoms.—The attack *begins very suddenly,* often at night and with or without such causes as fright, cold, heat, change of residence, etc. Sometimes there are prodromes, as conjunctivitis, coryza or flatulence and sometimes the attack coincides with the menses. (*a*) *Dyspnœa* is the first and the essential symptom; though at first slight, it soon becomes urgent; the patient opens the windows, sits upright, grasps the arms of the chair to brace the accessory respiratory muscles, and exhibits extreme distress, anxiousness and pallor, which is followed by cyanosis; the dyspnœa is expiratory and the abdominal muscles are board-like during expiration; inspiration is somewhat lengthened and *expiration is greatly prolonged,* wheezing *and whistling;* respiration is generally slow. (*b*) There is marked *cyanosis* in the face and extremities, the cervical veins bulge out like cords, the skin is clammy, and the pulse indicates poor oxygenation by its tenseness, smallness and frequency; carbon dioxide intoxication causes the occasional headache, delirium, tinnitus, twitchings, convulsions

PLATE XII



Sputum from a Case of Bronchial Asthma, showing large numbers of Eosinophilic Leukocytes and Free Granules. (Simon.)

It will be noted that the leukocytes are all mononuclear.
(Eye-piece 1, objective 1-8, Bausch & Lomb.)

and coma. (*c*) The *cough is dry* and unproductive. (*d*) The voice is faint and hoarse. (*e*) *Other physical signs* are as follows: On *palpation,* the vocal fremitus is found decreased by the bronchial spasm or secretion; rales are often felt. The normal *percussion* note is replaced by a *tympanitic* note, due to acute pulmonary emphysema; the lungs are voluminous, and distend the thorax, cover the heart and push down the diaphragm, whose excursion is thus reduced to a minimum. On *auscultation,* the vesicular murmur is usually faint or absent (Laennec) because of secretion in the bronchioles, and loud whistling, piping or *sibilant rales* are heard often without the stethoscope and largely during expiration. They are due to bronchial stenosis. (*f*) *Sputum* is voided for the first time toward the end of the attack; this occurs as a species of crisis to the threatening, but very seldom fatal, asphyxia. A few drams of tenacious mucus, resembling egg albumin, are voided. Minute gray balls are seen; these are the "perles" of Laennec; when unrolled the "pearls" are seen to contain the *spirals* of Leyden, Ungar and Curschmann; they are best detected with the naked eye, and on a black background; they measure 2 or 3 to 10 x $\frac{1}{2}$ to 1 mm.; they are *mucin casts of the bronchioles,* to which may cling leukocytes, epithelia, fat or myelin droplets; some show a central band of mucin; it is thought that they occur from the passage of mucin through the contracted bronchioles; they disappear

FIG. 32.

Spirals and crystals in bronchial asthma. *B*, Charcot-Leyden crystals; *A*, spirals magnified, and *C*, their natural size.

within a day or two; spirals are also present in catarrhal or fibrinous bronchitis and in pneumonia. The *Charcot-Leyden crystals* may be seen in yellow dots in the sputum; they are pointed and octahedral, consist of an organic basis with phosphoric acid, and originate from the eosinophile leukocytes which are increased in the sputum and blood, constituting a quarter to more than half of the white cells. The crystals, spirals and the occasional fibrinous casts are a result rather than a cause of the asthma. (Plate XII.)

The attacks last one or two hours and may be *repeated* at long or short intervals. In the hebdomadal form, attacks recur every Sunday or Monday; they are probably due to change in routine or to digestive excesses. The urine after the attack contains increased uric acid; constipation and flatulence may result from fatigue of the diaphragm and expiratory muscles.

Diagnosis.—The diagnosis is made by (*a*) the paroxysmal occurrence of asthma, (*b*) the expiratory dyspnœa and (*c*) the acute transient emphysema.

Differentiation.—(*a*) All diseases causing *inspiratory dyspnœa* can be at once excluded, such as œdema or spasm of the glottis, paralysis of the post. crico-arytenoid muscles and tracheal and bronchial stenosis. In *spasm of the glottis,* the spasm is inspiratory and of short duration, the larynx moves up and down, and the epigastrium retracts during inspiration; there is no emphysema and no spirals are found. Spasm of the *diaphragm* is very rare (*v.* Phrenic Nerve); it lasts a much shorter time, the epigastrium bulges during inspiration, there is spasm of all the inspiratory muscles, hysterical stigmata are often observed and the lungs are normal. (*b*) *Expiratory dyspnœa* may also occur in (i) *chronic bronchitis with emphysema.* In these cases the emphysema is constant, the lungs show constant rales and few eosinophiles are present in the sputum. (ii) It also occurs in *immovable tumors* below the vocal cords which may turn upward during expiration (this is shown by the laryngoscope). (*c*) *Asthma cardiale* is of a mixed type, *i. e.,* inspiratory and expiratory, and is therefore readily differentiated. The "lungenstarr" or lung distention (*v.* Signs of Valvular Heart Disease), due to its engorged vessels, may simulate the emphysema of bronchial asthma. Cardiac asthma may occur either with high or low arterial tension, whence the condition of the pulse is no certain criterion. The eosinophiles are not increased to the same degree as in bronchial asthma.

Prognosis.—The prognosis is uncertain and depends on the etiological accessibility of the asthma. It is more favorable in young individuals than in those of advanced years. Cases which recurred for 64 years are recorded. Chronic bronchitis and emphysema are likely to develop. Death in an attack is very rare.

Treatment.—1. Of the Attack.—A hypodermic injection of *morphine,* gr. ¼, with *atropine* gr. 1/80 and spt. glycerylis nitratis ♏ij gives the most prompt and reliable results. Other measures are purely accessory. Chloral is almost useless, for the condition usually requires single doses of gr. xxx to xl, which are too large for safety. Adrenalin solution, 1–1000 ♏v–x hypodermatically, usually gives instant relief; it is not without danger in arterio-sclerotics. Fresh air, strong coffee, whiskey, a cigar or cigarette, the application of cocaine to the nose, fluidextract of grindelia ♏x to xx, especially in bronchitic types, nitrite of amyl pearls, or a few whiffs of chloroform, sometimes give relief. The vexing question arises as to leaving chloroform or morphine in the hands of the patient whose attacks often

come and go without medical aid; as a rule they should never be left with the patient. Inhalation powders, which are burned like incense, are very generally used; they all contain some such palliative as lobelia, stramonium, belladonna, hyoscine, etc. (Lobelia, powdered black tea and powdered stramonium leaves, each one ounce; soak well in two ounces of a saturated solution of potassium nitrate; dry.) Excellent cigarettes are made containing arsenic, potas. nitrate, belladonna and stramonium; their antispasmodic effects on deep inhalation of the smoke is sometimes remarkable. According to Burney Yeo, the celebrated *Cigarettes d'Espic* are said to be made of the following ingredients:

Belladonna leaves	5½ parts.
Hyoscyamus leaves	2¾ parts.
Stramonium leaves	2¾ parts.
Phellandrium aquaticum	1 part.
Extract of opium	⅛ part.
Cherry-laurel water	a sufficiency.

Oxygen is occasionally efficacious and strong coffee may sometimes abort the attack.

2. Of the Tendency.—(*v.* Etiology.)—The nose requires especial attention; cauterization of sensitive areas, even when there are no gross lesions, is imperative. Strümpell has effected 11 cures with sweating by strong lamps. The diet should be regulated; the evening meal should be light, and carbohydrates and tea should be restricted; an emetic may abort an attack of the gastric type. Nervines (valerian, arsenic, bromides) may be used when necessary. The best remedy between attacks is potassium iodide; this treatment should be continued for months; potassium iodide is combined to advantage with Fowler's solution and belladonna, not only to correct the action of the iodides on the skin and secretory glands, but also for their direct nervous and antispasmodic effects:

℞	Liq. potassii arsenitis	ʒj.
	Tr. belladonnæ	ʒj.
	Potassii iodidi	ʒij.
	Fluidextr. grindeliæ (U. S. P.)	ʒvj.
	Aquæ	q.s. ad ʒiv.

M. et S.—One teaspoonful after meals, in water.

Alkalies may be vaporized and inhaled, as sodium bicarbonate or sodium chloride. Climatic treatment is as beneficial as in bronchitis; a low level near the sea and in a semitropical climate is an ideal location. General hygiene and the hardening of the patient by fresh air and cold rubs are important.

DISEASES OF THE LUNG.

EMPHYSEMA.

Definition.—A permanent disease of the lungs, characterized pathologically by atrophy of the alveolar walls and great distention of the alveoli, and clinically by a large chest of the inspiratory type, distended lungs with poor excursion, and hypertrophy and dilatation of the right ventricle. Emphysema means a "blowing up" or distention of the lungs, of which the various types will be considered under DIFFERENTIATION. It was first described by Laennec (1826). It constitutes $\frac{7}{10}$ per cent. of all autopsies (Virchow).

Etiology.—Substantive or alveolar emphysema occurs mostly in individuals over 30 or 40 years of age, in men more than in women, and in cold climates. The immediate cause is chronic bronchitis and bronchiolitis (Laennec), *catarrhe sec;* it may follow pertussis, bronchial asthma, etc.; its comparative infrequency is not generally understood, whence many writers assume a congenital weakness or hypoplasia of the elastic tissue of the lung. Coughing, blowing, as in the use of wind instruments, straining during parturition or physical toil, artificial respiration in the new-born, and alcoholism are also considered causative.

Pathology.—On opening the chest at autopsy, the lungs are found distended (*volumen pulmonum auctum,* Traube); they overlap the heart, depress the diaphragm and narrow the mediastinum. Their surface is pale rose-colored, is somewhat anæmic, and presents numerous small vesicles, $\frac{1}{2}$ to 4 mm. in diameter, representing the fusion of several alveoli. Some large bullous areas are found under the pleura. The lungs crepitate slightly to the fingers, under which they feel, as Laennec put it, "like a pillow of down." The lungs collapse but little on section. In some places the pigment deposit is less than usual,—called *albinism* by Virchow. Emphysema is most marked on the surface of the lung, particularly over the upper lobes, on their convex anterior aspect, and behind near the spine. The normally thin edges of the lung are greatly rounded. *Histologically,* two findings are characteristic: (*a*) There is *atrophy* of the *interalveolar elastic tissue,* so that the *alveoli fuse,* corresponding to the vesicles seen on the surface; slight intercommunication of the alveoli is normal, but the coalescence of several alveoli is extreme in emphysema; these changes account for nearly all the clinical findings in the lung. (*b*) The *capillaries* are *obliterated* by *thrombosis,* which explains the clinical hypertrophy, dilatation and ultimate failure of the right heart. The bronchi are frequently inflamed, thickened, shortened and sometimes dilated.

Pathogenesis.—It is not wholly clear whether the elastic tissue wasting or the vascular occlusion is the primary change; or, if the elastic atrophy is primary, whether it is acquired or congenital. The changes may be brought about (*a*) *mechanically* by forced inspiration

(Laennec, Rokitansky, Gairdner) or by forced expiration (Jenner, Mendelsohn). This theory is most favored by clinicians, though pathologists think that emphysema is due to (*b*) *nutritive alterations,* as vascular changes or atrophy of elastic or muscular tissue; calcification of the costal cartilages is probably more sequential than causal.

Symptoms.—Symptoms may be considered under two general headings: (1) Those of expiratory insufficiency of the lungs, due to their distention which results in turn from the loss of elastic tissue; and (2) those of cardiac insufficiency, which follow the above and result from obliteration of the vessels in the lesser circuit.

1. Expiratory Pulmonary Insufficiency.—Normally expiration is wholly passive, resulting from the elasticity of the lungs and collapse of the chest. Expiration in emphysema is incomplete, as shown by Waldenburg's pneumotometry and Riegel's stethography; in the latter the curve resembles one resulting from division of the vagus. The *cyrtometer* shows that the chest is more circular; the *spirometer* shows a decrease of 20 to 60 per cent. in the normal vital capacity (2–4000 c.c.); and *mensuration* shows a decided reduction in the normal expansion of 2 to 3 inches. Upon *inspection,* dyspnœa is the most conspicuous finding; it is expiratory and is due to the loss of lung elasticity and consequent impairment of lung circulation and oxygenation; it is enhanced by exertion, by bronchial catarrh, by accessions of true asthma and by cardiac insufficiency. If emphysema develops gradually it may cause no dyspnœa (Traube). The frequency of respiration is increased to twenty-five or thirty and the type is costal. The *facies* and *habitus* are most characteristic; the eyes are somewhat prominent, the nose is bluish and slightly thickened, the head is thrown back and the chest forward to give play to the accessory muscles of respiration, and the skin is cyanotic even during rest and when dyspnœa is not urgent. A network of dilated veins is often seen over the lower chest, resulting merely from venous obstruction. The jugular veins fill enormously on expiration and often pulsate during the diastole. The neck is short, and the hypertrophied sternomastoids, scaleni and trapezii protrude rigidly. The back and abdominal muscles are rigid, and thus supplement the inadequate action of the diaphragm. The shoulders are elevated, though stooped; the chest is of the permanent inspiratory type and is usually *barrel-shaped* (from loss of the elastic tissue), rarely of the paralytic type; the anteroposterior, oblique and sometimes the transverse dimensions of the chest are increased. The raised clavicles accentuate the supraclavicular notches, in which the apices sometimes protrude, on coughing, as hernial tumors. The angle of Louis is prominent and the interspaces are wider in the upper and narrower in the lower parts of the chest, and retract with each inspiration. The lower thorax shows a groove indicating the attachment of the expiratory muscles. The play of the diaphragm is limited, as shown by the *x*-rays and by the absence of Litten's sign. *Palpation* detects the rigidity and dilatation of the

thorax, its poor excursion, the hardness of the muscles, the thick, often calcified costal cartilages and decreased vocal fremitus. The edge of the liver may be found depressed, though the spleen is seldom palpable. *Percussion,* absolutely essential to a diagnosis, gives a note which is loud and deep—hyperresonant—or on the sides and back is actually "*box-like*" or tympanitic; it is also described as muffled or wooden and in extreme cases impresses one as dull (Skoda). The voluminous lungs narrow or wholly cover the cardiac dulness, depress the upper level of the splenic and hepatic dulness, invade Traube's space, and show either no respiratory excursion or movement not exceeding a finger's breadth. At the back the lungs may reach the twelfth dorsal or even the second lumbar vertebra. Upon *auscultation,* expiration is found greatly lengthened so that it is to inspiration as 2 or 4 to 1, the converse of the normal. Vesicular breathing is absent, or the breathing is indeterminate; even when the stethoscope rises and falls there may be no audible sound. A rubbing sensation may result from muscular contraction or possibly from the emphysema vesicles. Rales result from bronchitis or stasis; a pearly-colored mucin with few cells may be evacuated after much coughing.

2. Cardiac Insufficiency.—The work of the right ventricle is increased because the pulmonary arterioles are in part occluded, and because the imperfect expiration allows neither free aspiration of venous blood into the auricles nor adequate filling of the aorta. The right ventricle, therefore, hypertrophies, and this causes the accentuated second pulmonic tone; hypertrophy occurs mostly about the pulmonary conus arteriosus. The diaphragm is low, and therefore the hypertrophied and more horizontal heart beats in the epigastrium. Dilatation soon follows and is accompanied, in advanced cases, by tricuspid leakage, nutmeg liver, congested kidneys, ascites, œdema and other signs of cardiac insufficiency. The alteration in the heart, so marked at autopsy, is often obscured during life, for the lungs cover the heart and muffle its tones. Functional heart murmurs are mostly systolic, very rarely diastolic, and result chiefly from degeneration or relaxation of the heart muscle.

The *clinical course* is long and often covers decades. Patients may acquire emphysema in childhood and live to be 60 or 70 years old.

Diagnosis.—Developed types are easily recognized by the barrel-chest, its hyperresonance, the low borders of the lung, their lack of excursion, the obscuration of cardiac dulness, the expiratory dyspnœa and cardiac stasis.

Differentiation.—(*a*) *Acute distention of the lung* or acute emphysema, as after asthma or pertussis, cannot be distinguished by one examination. It is seen repeatedly in bronchial stenosis due to aortic aneurysm. It regresses rapidly even after months, when its cause is removed. (*b*) *Vicarious or compensatory emphysema* often develops in one lung when contralateral pleurisy or cirrhosis of the

lung exists; it may occur on the same side with the lesion; marked apical emphysema almost certainly indicates some deeper induration; it may develop in the death agony; it may eventually cause wasting of the lung. (*c*) *Emphysema senile,* or Jenner's "small-lunged" emphysema, is merely atrophy; the connective tissue wastes and the alveoli fuse, but the lungs are not enlarged; they are indeed smaller, the diaphragm stands higher, the cardiac dulness is increased, the chest flattened, the ribs oblique, the respiratory muscles wasted, the right heart is not hypertrophied and respiratory excursion is retained. (*d*) *From interalveolar interstitial emphysema* the first differential point is the etiology; (i) it may be caused by trauma, external or internal, which occurs in 57 per cent. of tracheotomies (Money), and is promoted by raising the deep fascia from the trachea during the operation (Champneys); it is especially frequent with pneumothorax. (ii) It may result from foreign bodies. (iii) Ulceration of the air passages, gangrene, abscess or tuberculosis and less often gastric ulcer or cancer may be its cause. Other etiological factors are (iv) forced entrance of air, as in resuscitation of the new-born; (v) severe coughing efforts, as in whooping cough; (vi) severe pressure efforts, as in parturition; and (vii) spasm of the glottis. The second point is a series of symptoms which are not, however, always present, as creaking in the mediastinum, sometimes with each systole; emphysema and crepitation of the cervical cellular tissue, bulging of the interspaces, creaking and tympany at the edge of the ribs when the emphysema is under the costal pleura; absence of the vesicular murmur; sometimes pneumothorax; and obliteration of the heart dulness, dyspnœa and air embolism. It is usually fatal. (*e*) In *congenital hypertrophy* of the lung (*pulmo excessivus*) the lungs are very voluminous, but their excursion, auscultation and percussion note are normal. To the author it appears especially common among Poles. (*f*) *Pneumothorax* (*q. v.*) is unilateral, is associated with fluid in the pleura, has a more tympanitic note and is associated with succussion, tinkling rales and other pathognomonic signs.

Prognosis.—Recovery is unusual in the genuine substantive form, which is permanent. Transient forms resulting from pertussis, foreign bodies or asthma, often or usually regress completely. Life, even relative comfort, may be sustained for years or decades. Much depends on associated conditions, as frequent severe bronchitis, chronic nephritis, arteriosclerosis or myocarditis. Extreme forms impede the flow of chyle which, with dyspnœa and fatigue, impairs nutrition. Severe complications are failure of the right heart upon which compensation devolves, cyanosis, gastric disturbance, diarrhœa, hemorrhage from the lungs or bowels (in arteriosclerosis or infarction), scanty urine and anæmia (in the aged).

Death usually results from cardiac failure or from œdema pulmonum. It may occur from apoplexy, pneumothorax, hæmoptysis, uræmia and very rarely from pulmonary inflammation, to which subjects of emphysema show no predisposition. The teaching that

tuberculosis (Rokitansky) or valvular disease (Bouillaud) excludes emphysema, is incorrect.

Treatment.—(*a*) *Prophylaxis* concerns chiefly the concomitant or causal bronchitis. Severe exercise is to be avoided. (*b*) *Coincident gastric, renal* or gouty disease must be treated. (*c*) *Climate* is as important a factor as it is in chronic bronchitis, an almost inseparable associate of emphysema; high altitudes are to be avoided as they directly promote emphysema. (*d*) *Pneumotherapy* is sometimes beneficial. It includes inhalation of thinned air, or, where there is marked catarrh, of compressed air. Two or three treatments of ten minutes should be given daily, and the time should soon be extended to half an hour. (*e*) *Chronic bronchitis* (*q. v.*), and (*f*) *asthma* (*q. v.*) are important considerations in treatment. (*g*) *Rhythmic compression* of the chest, three or four times daily, should be practiced during 20 to 30 expirations; it increases the circulation in the lung but not respiration. (*h*) *Cardiac insufficiency* is treated as in valvular or myocardial disease (*q. v.*) and strychnine, venesection and other measures should be used. The topic is discussed briefly, as no remedy or measure can restore lost elastic tissue or return resilience to atrophied contractile fibers.

BRONCHOPNEUMONIA.

This affection, partially outlined by Sydenham and Gebhart, was first well described in 1840, by Rilliet and Barthez, who separated it from lobar pneumonia and recognized it as a secondary disease. It might be well classed among the infections, such as fibrinous pneumonia, except that unlike the latter it is not due to a single specific microörganism.

Definition.—Bronchopneumonia is defined with difficulty; (*a*) *etiologically* it is due to various microbes, is generally secondary to the bronchiolitis of some specific infection, as measles or pertussis and occurs chiefly in children; (*b*) *pathologically* it develops about the bronchioles (bronchopneumonia) and in a few lobules (p. lobularis, p. insularis or nodosa) rather than throughout a lobe; its exudate is rather catarrhal (p. catarrhalis) than fibrinous, and its distribution is usually multiple and often disseminated (p. disseminata); (*c*) *clinically* it is identical with capillary bronchitis, which, if it occurs in the first years of life, almost never spares the lung tissue; it is marked by fever, cough, dyspnœa and cyanosis, which may often co-exist without obvious signs of hepatization.

Etiology.—(*a*) Some cases, especially in children under two years are *primary;* these are estimated by Holt and Connor at 35 per cent., by others at a much lower figure; (*b*) most cases are *secondary* to infections affecting the upper air passages. In 279 cases of infantile bronchopneumonia reported by Samuel West, 32 per cent. followed measles, 24 per cent. pertussis, 16 per cent. diphtheria, 15 per cent. macrobronchitis, 7 per cent. ileocolitis, 3 per cent. scarlatina, 2 per

cent. influenza, ½ per cent. each varicella and erysipelas. (*c*) *Predisposing causes* in the young are poor sanitation, malnutrition, syphilis, tuberculosis and rickets. Bronchopneumonia causes 9 per cent. of deaths in foundling asylums (Müller). (*d*) *Age.* Most cases are observed in children two or three years old. Seventy-five per cent. of cases of pneumonia in children under five years of age are lobular (Holt). It is less frequent in adults; in weak, cachectic, nephritic and aged persons it may cause death. (*e*) In some cases microbes may act directly on the lungs, as in the primary cases in children, in the influenza of adults, and in aspiration or inhalation pneumonia. "Schluck pneumonie" is prone to develop in diseases in which the laryngeal sensibility is decreased, as in diphtheritic pareses or bulbar palsies; in ether anæsthesia in which the germ-laden saliva is drawn into the lungs; in comatose states, as apoplexy or uræmia; in mental diseases; in the new-born; in cases of partial drowning; in operations on the throat; or when there is persistent vomiting. Smoke, vapors and dust are exceptional causes. (*f*) It is probable that *infection* may sometimes occur by the *lymph or blood streams,* especially in septic affections.

Bacteriology.—Bronchopneumonia is no specific bacteriological disease. According to Netter, *in adults* about one-third of the cases show more than one organism, among which the pneumococcus ranks first and the streptococcus second; and about two-thirds show one germ, of which the pneumococcus constitutes 39 per cent., the streptococcus 31 per cent., Friedländer's bacillus 23 per cent., and the staphylococcus 7 per cent.; *in children* mixed infection is present in half the cases, the streptococcus predominating, while in the other half there is but one organism, the pneumococcus being the most common. In Wollstein's cases, 67 per cent. were pneumococcic. Other germs found are the influenza, typhoid, tubercle and colon bacilli, Pfeiffer's Micrococcus catarrhalis, Micrococcus tetragenus and meningococcus. The tubercle bacillus is often associated with the pneumococcus.

Pathology.—The foci of consolidation are usually diffused in the lower and sometimes in the upper lobes, posteriorly and usually bilaterally. They vary in size from a pin head to a walnut or larger; many or most foci lie near the pleura, through which they may often be seen and felt; the pleura is often granular or slightly fibrinous, sometimes ecchymotic. On section the consolidated nodes appear reddish-brown and later yellowish from fatty degeneration of the cells; they are firm, airless, whence they sink in water, and on section are generally smooth and glistening, though some pneumococcus infections show the granulation and viscid secretion of genuine fibrinous pneumonia. The bronchioles supplying the consolidated nodes are plugged with muco-pus, so that air cannot be forced through them; the bronchioles are reddened, swollen, sometimes ecchymotic or dilated. Many areas are collapsed, which some consider is preliminary to consolidation, but though atelectasis is frequent its importance has been overestimated. In fortunately made sections a den-

dritic racemose or grape-like arrangement may be seen in which the bronchioles correspond to the stems and the alveoli to the leaves or grapes. *Microscopically* the bronchioles show dilated vessels and leukocyte emigration, the alveoli show epithelial desquamation and proliferation, serous exudation, leukocyte outpouring and a scattering of red cells; the alveolar walls show much interstitial inflammation. The usual statement that no fibrin is found is a mistake; though influenza pneumonia is strictly cellular (catarrhal), aspiration, pneumococcic and other forms not infrequently show a fibrin network, though decidedly less than in lobar types. Near the foci and in the upper lobes compensatory emphysema often develops. The bronchial lymph glands are hyperæmic, swollen and succulent. *Extension* occurs directly from alveolus to alveolus or along the peribronchial connective tissue. In some forms numerous lobular foci increase in size and perhaps fuse so that the greater part of a lobe may be consolidated; this pseudolobar form is often due to the pneumococcus, is seen in measles and diphtheria and may clinically present a close resemblance to lobar pneumonia, though pathologically areas of collapsed, dark, vascular or sound tissue lie between the bronchopneumonic patches.

Symptoms.—The clinical picture varies considerably, accordingly as the bronchopneumonia is primary or secondary. *In the primary form,* most often pneumococcic, the onset is brusque, with chill, convulsions, vomiting or pain in the side. The fever rises suddenly, remains high and often falls by crisis. Capillary bronchitis is absent and the consolidation is often marked. The course is short and the death rate is low. In adults this form is frequently unilateral. The *more common secondary form* begins insidiously, especially if it follows a microbronchitis; it is often obscured by the disease which it complicates. The fever gradually rises to 103° or 104°, if fever were already present, and is generally irregular or remittent, rising with the formation of each new focus; it may be low or absent in marantic subjects. There may be no physical findings, or if present they are frequently indeterminate. If recovery ensues, lysis is the usual solution. Von Ziemssen held that fever in bronchitis seldom exceeds 102° and that higher registrations indicate pneumonia.

Subjective and General Manifestations.—These are often *more obvious* than the objective or pulmonary symptoms. The pulse rises to 120 or even 180 and in convalescence is more tardy in reaching normal than is the fever. The respiration is often 60 or 80, the expiration is frequently catchy or grunting, and the respiration-pulse ratio, normally 1 to 4, becomes 1 to 3 or 2; dyspnœa develops early and with it restlessness and irritability. Cyanosis develops in the face and then in other parts from impaired flow of the blood to the heart which is due to continued coughing and stagnant secretion. The cervical veins are prominent. Pain is not as common as in lobar pneumonia. The cough becomes drier and more distressing; the sputum is usually swallowed in patients under seven years of age, but

if obtained shows nothing characteristic, though von Niemeyer found that it sank in water or floated under the surface. Meteorism is not uncommon in severe or moribund cases.

Lung Findings.—On *inspection,* irregular excursion is sometimes seen. The accessory muscles of the nose, neck, chest and abdomen are in full play. The interspaces, ribs, sternum and epigastrium in young plastic chests, retract greatly with each inspiration; this must not be confused with the slight physiological retraction observed in the beginning of inspiration. *Palpation* may show increased fremitus in a focus near the surface and measuring 2 x 5 cm. Stagnant secretion in the air tubes may temporarily suspend fremitus. *Percussion* is often negative for a few days or even throughout the course, as it is obvious that deep, isolated foci cannot produce dulness. In the thin, infantile thorax, percussion must be made lightly and special importance should be attached to palpatory percussion. Dulness obtains over a focus having an area of 2 x 5 cm. It is most often found along the spine over the lower lobes, but sometimes also over the scapular spines. Dulness is most marked in the pseudolobar (confluent) forms. Atelectasis may cause dulness but not bronchial breathing. A hyperresonant note in front indicates emphysema; behind it shows relaxation. *Auscultation* reveals subcrepitant or crepitant rales; they are often heard over an area not exceeding the bell of the stethoscope. Bronchial breathing and bronchophony are fairly frequent, and occur especially when the child cries. In other cases the breathing may be rude or puerile.

Special Types.—Special types have been considered under measles, pertussis, etc. Abt (following Holt, Carmichael, Ashby and Wright) offers the following pathological classification, which "undoubtedly gives the clearest basis for an understanding of the physical findings: (*a*) *In capillary bronchitis* there is no sign of consolidation in any part of the lung. Sibilant rales, coarse and fine crepitant rales are distributed over one or both lungs. The sibilant rales are due to congestion and swelling of the mucous membrane of the larger and smaller tubes. A feeble respiratory murmur indicates areas of congestion. (*b*) In *disseminated bronchopneumonia,* which is characterized by small areas of consolidation, percussion usually gives negative results, though sometimes there is very slight dulness. The vocal fremitus is usually not altered. As a rule the consolidated areas are situated deeply in the lung, or are too small to cause any appreciable difference in the percussion note. Auscultation reveals crepitant rales and bronchovesicular breathing over the consolidated areas. Vocal and crying resonance is exaggerated. (*c*) In *acute generalized bronchopneumonia,* the percussion note is dull over a large area, even an entire lobe. Palpation shows an increase in vocal fremitus. Auscultation reveals bronchial breathing and fine moist rales over the dull areas, while the rest of the lung shows evidence of bronchitis."

Issues.—*Death* may occur from carbonic acid narcosis, irritability

passing into apathy, the harassing cough becoming less marked and causing stagnation of bronchial secretion in the large tubes ("suffocative catarrh"); delirium develops, the pupils narrow and Cheyne-Stokes' breathing and heart failure result. *Complete resolution* is the rule in cases which recover. *Delayed resolution* with remissions and exacerbations may occur. *Complications* may develop; (*a*) pleurisy is the most frequent; (*b*) gangrene; (*c*) abscess, especially in aspiration and influenzal forms; (*d*) pulmonary induration; (*e*) bronchiectasis; (*f*) tuberculosis, which is less often a sequel than a previous condition awakened by the new infection; (*g*) pericarditis, endocarditis, otitis, emphysema and fibrinous bronchitis are other possible complications.

Diagnosis.—Reliance on physical signs alone leads to many errors and a diagnosis is often determined by the development of sudden high fever or mild fever suddenly rising during bronchitis, by dyspnœa, irregular rapid respiration, cyanosis and pain in the chest. In very young or marantic children and in the aged the onset and symptoms may be very atypical. *Lobar pneumonia* (*q. v.* variations in children) occurs mostly in sound children over three years of age, runs a shorter cyclical typical course, ends by crisis, is unilateral and involves the greater part of one or more lobes; while bronchopneumonia occurs more often in younger weakly children, is often a sequel of bronchiolitis, runs a longer, more atypical, unfavorable and relapsing course, resolves by lysis, is bilateral and is less marked by definite pulmonary findings. *Tuberculosis* is often distinguished only by the course; it invades the upper lobes more than does bronchopneumonia, and bacilli-laden sputum may be recovered by washing the stomach or by swabbing the throat when the child raises mucus after coughing. *Pleurisy* (*q. v.*) is diagnosticated by means of the aspirating needle.

Course and Prognosis.—The *course* may be (*a*) *acute,* lasting about a week, of which the measles-pneumonia is the prototype; (*b*) *subacute,* lasting from two to eight weeks, exemplified by the pertussis-pneumonia, or (*c*) *chronic,* covering months, as in tuberculous bronchopneumonia.

The *prognosis* depends on the patient's social situation, previous history, age, the disease of which the pneumonia is a complication and the extent of the pneumonia. The outlook in general is serious, especially in secondary forms, among which measles and pertussis represent the more serious types; in the very young it is almost as fatal as infantile diarrhœa; aspiration forms are very serious, as are those in marantic and aged subjects. In private practice the death-rate ranges from 10 to 33 per cent., in hospitals 33 to 50 per cent. and in asylums even 50 to 100 per cent. Early cyanosis, dyspnœa and rapid pulse are unfavorable signs.

Treatment.—1. Prophylaxis—(*a*) In infections which produce nasal or pharyngeal inflammation the nose and throat should be frequently cleaned with mild alkaline antiseptics. (*b*) When there is

laryngeal or bronchial inflammation, congestion and atelectasis should be avoided by frequent and regular change of posture. (*c*) Deep breathing should be encouraged in older subjects, or enforced in the young by affusions of cold water on the neck and upper thorax. (*d*) Exposure should be avoided, especially at night, by pinning the bedclothes or firmly fastening the nightgown to the foot of the bed.

2. THERAPY.—Actual treatment is symptomatic, as the varied etiology precludes specific therapy. (*a*) The patient should have plenty of *fresh air.* The temperature of the room should be maintained at 67° to 70° with constant moderated ventilation. (*b*) The diet should be *fluid,* diluted or predigested; egg albumin, milk and plenty of water are indicated. At the onset or during the course calomel gr. $\frac{1}{10}$ every hour for five or six doses should be given, followed by castor oil, particularly if there is indigestion or tympany. (*c*) *Expectorants,* as ammonium carbonate, tartar emetic or ipecac, should not be given generally, as they derange the digestion, and good digestion is of great importance to strong as well as to weak subjects. Aromatic spirits of ammonia, ♏v to xx, every three or four hours, is least deranging and is a diffusive stimulant. (*d*) *Pain* rarely necessitates the use of opiates, which induce stagnation of secretion; dyspnœa, pain, restlessness, cough, cyanosis and rapid pulse may be relieved by paregoric, which is preferable to Dover's powder or morphine; but great care is imperative in the use of any opiate in young, old or weak subjects. *Fresh air is better than drugs.* (*e*) *Local applications,* as poultices or pneumonia jackets and blisters, are useless. (*f*) In the treatment of *fever,* antipyretics, aconite, antimony and other cardiac depressants should be wholly avoided; cool applications are equally efficacious; they also lessen tympany and carbon dioxide narcosis, stimulate the heart and brain centres and deepen the respiration. A cold pack should be applied over the neck, chest and abdomen and should be changed frequently. Cold must be used with care in very young and in marantic subjects; in such cases a warm sponge or bath may be given, during which cold water may be poured on the neck to stimulate deep breathing. (*g*) For *cardiac stimulants,* whisky and brandy may be given in daily doses of ʒss to ʒiij, very well diluted in water or in peptonized milk. Aromatic spirits of ammonia, digitalis, saline infusions or enemata, camphor or strychnine may be used. (See page 91 for dosage in childhood.) Hypodermics of ether may cause sloughing. (*h*) Respiratory stimulants include tr. belladonna, oxygen, thoracic faradization and mechanical compression of the chest. (*i*) *Emetics* are unreliable; ipecac and similar remedies have no effect when there is pronounced carbon dioxide intoxication, whence it has been recommended to combine them with coffee, camphor or cognac. For carbon-dioxide intoxication phlebotomy should seldom be practised. (*f*) *Inhalations of steam* (*v.* DIPHTHERIA and CROUP) may be beneficial; compound tincture of benzoin and sodium bicarbonate are often added to the vaporizing water. (*k*) *During con-*

valescence fresh air or a sojourn in the country or in a warm climate is advisable.

INDURATIVE PNEUMONIA, LUNG CIRRHOSIS, FIBROID PHTHISIS.

Definition.—An almost invariably secondary overgrowth of the pulmonary connective tissue.

Etiology.—1. Diseases of the Lung.—These may induce *local*, lobular or lobar, unilateral or bilateral fibrous induration. Local induration frequently occurs about tumors, parasites, tubercles, gummata, abscess, gangrene, trauma or pneumokoniosis, and is of no essential clinical importance, except as it demarks dangerous foci of disease. *Diffuse* induration is important. (*a*) *Fibrinous pneumonia* sometimes results in lobar induration; Fränkel observed it seven times in 1,000 cases; it occurs notably in marantic or aged subjects from delayed resolution and lack of regeneration of the alveolar epithelium; the lung becomes airless and dense from development of fibrous tissue within the alveoli whence it involves contiguous structures; on section its surface is smooth, and the connective tissue is strikingly translucent. Subacute induration may reach considerable development in one or two months. (*b*) *Bronchopneumonia* in the course of measles, pertussis or influenza, sometimes leads to subacute or chronic induration, beginning from an alveolitis or peribronchitis. (*c*) *Foreign bodies* and *aspiration pneumonia;* (*d*) *chronic tuberculosis;* and (*e*) syphilis, gangrene, etc., are etiological factors.

2. Bronchial Affections.—(*a*) *Bronchiectasis,* which was noted by Corrigan, who first used the term lung cirrhosis; (*b*) *bronchial stenosis,* as by aneurysm or gumma; and (*c*) *pneumokoniosis* and putrid and chronic bronchitis, may cause indurative pneumonia.

3. Pleural Affections.—It is still disputed whether pleurisy is primary to lung induration or secondary; primary pleurisy may sometimes invade and cirrhose the lungs—the "pleurogenous" interstitial pneumonia of Charcot; strands of connective tissue run into the lung and approach and fuse toward the hilum.

Other causes are uncertain, *e. g.,* diabetes, alcoholism and malaria.

Pathology.—The pathology of an affection with such numerous causes can scarcely be described. The early induration is reddish, and the older connective tissue is darker, slate colored or gray; to them Charcot gave the names of *induration rouge* and *induration grise,* respectively. The new connective tissue may be formed in the bronchi, septi, alveoli, bloodvessels or pleuræ. The alveoli nearly always show a hyperplasia of connective tissue. The lung is airless and dense; is cut with difficulty and on section presents either a granulated or a smooth surface, the latter in cases following lobar pneumonia and sometimes in those consecutive to gangrene or aspiration pneumonia. The volume of the lung is decreased. Its shrinking produces changes in the mediastinum, heart, diaphragm and sound lung, which will be considered under Symptoms.

Symptoms.—Incipient or localized lesions are usually undetected unless they are observed in the course of pneumonia or tuberculosis. Symptoms may be *inseparable from the causal affection* (tuberculosis, bronchiectasis) or may follow closely upon it (pneumonia, pleurisy). There may be a low fever, cough, dyspnœa on exertion, pain in the side, rapid pulse, cyanosis, anæmia, malnutrition and night sweats. In cases where the original malady has ended, there may be only slight cough or some shortness of breath on exertion.

Physical findings of the typical chronic case are as follows: (*a*) On *inspection* the interspaces are narrowed on the affected side, the nipple and scapula are nearer to the median line, the shoulder is lower, the spine is convex toward the sound side and the measurement is unilaterally decreased. In left-sided induration the lung retracts from the heart, leaving its pulsation more widely exposed and disclosing the pulmonary artery, over which may be seen and felt its systolic filling and the strong diastolic closure of the pulmonary valves; in right-sided induration the heart beat may appear to the right of the sternum or may be covered up by the compensatory emphysema of the left lung. (*b*) *Palpation* corroborates these findings, and, as a rule, shows increased vocal fremitus. The second pulmonic tone is much accentuated, corresponding to the hypertrophy of the right ventricle and the occasional atheroma of the pulmonary artery. (*c*) *Percussion* elicits dulness below and sometimes a slightly hyperresonant note above the indurated area; the apex of the upper lobe is on a lower level and the diaphragm stands higher (the liver rising or Traube's space increasing); there is no respiratory excursion. (*d*) *Auscultation* brings out the absence of breath sounds below, and bronchial breathing and subcrepitant, crepitant and bubbling rales above. Additional signs result from the right heart hypertrophy and dilatation and from coincident bronchiectasis or cavity formation on the same side, and the well developed compensatory emphysema on the opposite and otherwise healthy side. There is nearly always some sputum.

Diagnosis.—Diagnosis is more easily made in the very chronic than in the subacute forms; subacute post-influenzal or post-pneumonic induration may easily suggest tuberculosis or interlobular suppuration. Slow-growing tumors of the lung may cause confusion.

Prognosis.—The prognosis varies with the etiology. Ulcers may form from arterial occlusion or from infected bronchial secretion, whence the frequency of lung hemorrhage (in 50 per cent.) and its occasional role as a cause of death. Amyloidosis sometimes results, but death is usually caused by right heart failure, which may occur during some acute intercurrent disease.

Treatment.—Therapy is unavailing, though life may be prolonged for decades under proper hygiene. Exercises with the arms may check the early shrinking, but nothing can remove an established induration. Warm climates at the sea level or at slight elevation are beneficial. Some writers hold that turpentine is of benefit in checking induration.

PNEUMOKONIOSIS.

Zenker employed the term to include various indurative diseases of the lungs due to "dust" inhalation.

Etiology and Pathology.—1. *Anthracosis* is due to inhalation of carbon, coal dust, soot, charcoal or graphite, and is known as "coal miner's disease" or phthisis melanotica. (*a*) Moderate amounts of carbon inhaled into the trachea and bronchi are absorbed by the leukocytes, worked upward by the ciliated epithelial cells and expectorated. (*b*) Larger amounts penetrate the bronchial mucosa and lodge in the connective tissue, or enter the lymph stream whence they reach the small lymph nodes around the bloodvessels, bronchi, pleuræ or mediastinal lymph glands. A moderate lung pigmentation occurs in all city inhabitants, while the lungs of countrymen are pink and devoid of soot deposit. It is possible that the lymph nodes may later throw out some of the pigment into the bronchial lumen. (*c*) Very large amounts may reach the alveoli. In these cases the lungs are ink-black. When they are, as it were, saturated, *connective tissue proliferation* develops in *insular foci* (endoperialveolitis nodosa and endoperilymphangitis fibrosa); this is sometimes called fibrous bronchopneumonic induration because of its localization; it occasionally measures 5 x 15 cm.; on section the foci are hard and exude an inky fluid. *Diffuse induration* is common. The *mediastinal lymph glands* are *indurated,* and the process often extends outside the glands (perilymphadenitis); by this latter process the glands may become adherent to and rupture into the pulmonary veins, whence pigment-metastases into the liver, spleen, kidneys and mesenteric glands are not infrequent. Bronchial perilymphadenitis may also lead to mediastinopericarditis, stenosis of the trachea, bronchi, œsophagus, cava, vena azygos and pulmonary artery, to œsophageal diverticulum, to fatal hemorrhage into the pericardium or into the bronchi when the aorta is also eroded, to aspiration pneumonia by intrabronchial rupture or to recurrent laryngeal paralysis by pressure.

2. *Siderosis pulmonum* (Zenker) is a very similar change, due to inhalation of metallic dusts, as iron, brass or bronze; there are the same interstitial deposit, lung induration, bronchial gland cirrhosis and metastases. The irritation is greater, nodes are more frequent and induration is more intense than in anthracosis. The iron deposits are yellow or red from the oxide, or black from the phosphate. The lungs contain 1½ per cent. of iron.

3. *Chalicosis pulmonum* (Peacock) is due to inhalation of alumina, quartz or sandstone, known as "stone-cutter's or millstone-maker's phthisis," "grinder's rot" or "potter's asthma." Calcium is less irritating than silica or quartz. Of all forms, the most nodules and induration occur in chalicosis.

4. Similar changes are observed from inhalation of wool, flax, cotton, grain, tobacco, glass, porcelain, pepper, cinnamon, bone and horn. Claisse and Jossué place special stress on bacterial coöperation.

Symptoms.—These may appear only after many years, when the symptoms of *chronic bronchitis, asthma, emphysema* and finally of *lung cirrhosis* appear. The *sputum* contains coal dust, soot, charcoal particles, as in Traube's celebrated case, and yellow oxide or dark phosphate of iron, quartz and silica. *Cavities* may form from bronchiectasis, softening of the nodose indurations or tuberculous complications; in some cases lung stones, set free by ulceration, may be raised. The mediastinum may be exposed by retraction of the lungs. Tuberculosis is common, occurring, according to Philip, in 13 per cent. of anthracosis and in 44 per cent. of chalicosis. Scissor-grinders rarely live beyond forty years of age and blasters in mines usually succumb in four years.

Treatment.—Treatment is largely prophylactic and that of the concomitant bronchitis and emphysema.

ATELECTASIS.

Atelectasis is a congenital or acquired "absence of air." It is also called apneumatosis.

I. Congenital Atelectasis.—The congenital atelectasis, first described by Jörg (1834), who differentiated it from pneumonia, is the persistence of the normal fœtal condition of the lungs; it develops in various forms of dystocia, and in weakly or premature infants.

Etiology and Pathology.—This variety is due to deficient inspiratory efforts from weak muscles, insufficient stimulation of the medulla centres and aspiration of mucus or meconium. The base, lower anterior margin and lingual lobe are most involved and the distribution is lobular. The color of the lung is steel-blue, and if the subject lives, the atelectatic portions do not become pigmented, for no air enters them. The airless areas are superficial, sunken, lax, do not crepitate and sink when put in water; in the early stage the part can be distended by inflation of the bronchus supplying it and on section exudes a serous or slightly blood-stained fluid. Cylindrical and cystic bronchiectasis may result; the ductus Botalli and foramen ovale are usually patent, the right heart is commonly dilated and thrombosis is frequent in the brain sinuses, kidney and right heart.

Symptoms.—The new-born with atelectasis breathe superficially and cry but little. Inspiration is marked by retraction of the epigastrium, subclavicular and intercostal spaces, for the lungs cannot follow the inspiratory distention of the thorax. If the undistended area measures 2 by 5 cm. it may be dull and increased fremitus with bronchial breathing may be detected, but more often the breathing is weak or entirely absent. Carbon dioxide narcosis develops, the pulse becomes small, the sensorium becomes dulled and, without active intervention, asphyxia causes death. If the patient lives, the chest is peculiarly deformed, its lower parts being sunken, so as to show the ribs. Köstlin describes a total collapse of one lung in a man aged twenty-two.

Treatment.—Removal of meconium and mucus by swabbing or

aspiration through a catheter, Sylvester's artificial respiration, Schultz's swinging, faradization and warm baths with cold affusions to the neck and upper chest are indicated.

II. Acquired Atelectasis.—Acquired atelectasis in children was first described by Legendre and Bailly (1844), and that in adults by Traube.

Etiology.—It may be due to marasmus, obstruction or compression; the lungs, already distended and ventilated, collapse and the air contained is absorbed. (*a*) The marantic form occurs in individuals reduced by typhoid, infantile diarrhœa or cachectic conditions, and by lying for a long while in the dorsal decubitus. (*b*) Obstructive collapse develops most frequently in bronchopneumonia, as the bronchioles in the young are disproportionately narrow; it may develop in any laryngeal, tracheal or bronchial stenosis. These forms affect the posterior parts particularly. (*c*) Compressive apneumatosis follows compression by aneurysm, dilated heart and exudative pericarditis, by pulmonary tumors, pleural exudates or pneumothorax, by mediastinal tumors or adenopathies and by abdominal tumors, ascites or meteorism. The part of the lung involved depends on the direction of the compression. In kyphoscoliosis permanent compression of the lung frequently leads to atelectasis, and if it occurs early in life, to hypoplasia.

Pathology.—Pathologically the same general findings obtain as in the congenital type. The lung is darker, red-brown or bluish. In the congenital and acquired types, hyperæmia may cause a flesh-like appearance (carnification), or serous transudation may cause a spleen-like aspect (splenization). There may be coincident inflammation or, in the healthy parts, vicarious emphysema. Long-standing atelectasis results in bronchiectasis, fatty degeneration of the alveolar epithelium and hyperplasia of the interalveolar and interinfundibular connective tissue, known as collapse induration.

Symptoms.—Symptoms are easily obscured by pleural effusion and by the causal bronchopneumonia, with whose foci it may be confused. Inspiratory retraction, lessened vesicular breathing, a slightly tympanitic note from relaxation of the collapsed lung and crepitant rales are common. The vocal fremitus is seldom increased and bronchial breathing is uncommon, unless the foci are both large and superficial. Dilatation of the right heart is common. In recent cases the *physical signs disappear after a few hours of change of posture, or after deep breathing* either voluntary or induced by cold affusions; this, with absence of fever, pain and cough, effectually differentiates from infarct, pneumonia or effusive pleurisy. After sleep for some hours on the back, a few crepitant rales may be heard physiologically over the lower posterior parts for three or four inspirations.

Prognosis and Treatment.—The prognosis and treatment vary according to the cause. Sudden death is not uncommon in persons having deformed spines. In all fevers, as typhoid, and in all respiratory inflammations, the posture must be changed frequently; orders to this

effect should be written, and the change should be recorded on the history sheet. Cold affusions to the neck, Brand's hydrotherapy and cardiac stimulants are indicated.

ABSCESS OF THE LUNG.

Suppuration is not a common lesion, but may occur in a sound or diseased lung.

Etiology.—(*a*) *Lobar pneumonia* causes 62 per cent. of the cases (Tuffier); localization in an upper lobe, alcoholism, marasmus, pneumonia complicating emphysema and lung induration, and hemorrhagic types are the predisposing factors. (*b*) *Lobular pneumonia* may suppurate, especially in the influenzal type. (*c*) *Aspiration pneumonia* is causal in 18 per cent. (Tuffier), particularly in insane or delirious persons; foreign bodies, food, saliva or pus from lesions in the upper respiratory tract, severe vomiting, œsophageal cancer or diverticulum rupturing into a bronchus, are its usual causes. (*d*) *Embolic suppuration* produces 17 per cent. of the cases. (*e*) *Trauma* is an etiological factor. (*f*) Abscess may complicate other pulmonary or bronchial affections, as tuberculosis, bronchiectasis, echinococcus, etc. (*g*) *Rupture into the lung* of contiguous foci of suppuration, such as empyema, suppurating peribronchial glands, subphrenic abscess, spinal caries and gastric ulcer, is the cause of 3 per cent. of lung abscesses. Most of the cases occur in middle-aged males.

Bacteriology.—The staphylococcus is most frequently the causative microörganism; the streptococcus, pneumococcus, pneumobacillus, influenza bacillus, colon bacillus and leptothrix are also found.

Pathology.—The cavity varies from the size of a hazelnut to an orange; in rare instances it may occupy an entire lobe or lung. The abscess is usually single, except in the embolic form. Its shape is irregular and it is sometimes multilocular. Its inner surface is irregular with floccular mural shreds, and is walled with pus-secreting granulations, which in some cases fill and obliterate the cavity. Fibrous tissue develops about the cavity, and in chronic abscess becomes dense and scar-like. The cavity is usually near the lung's surface, whence the frequency of complicating pleurisy; 80 per cent. of lung abscesses are in a lower lobe (Tuffier).

Symptoms.—The development of symptoms varies according to the causal factor; in lobar pneumonia the crisis or lysis is delayed and resolution is incomplete; in influenza the respiratory symptoms and fever persist; in sepsis, multiple lung involvement perpetuates the septic process. (*a*) *The sputum* is purulent, creamy, is often offensive or rancid without putridity, is alkaline and may measure a pint to a quart daily. It separates, on standing, into two layers, a granular sediment and a serous upper layer, sometimes with frothy surface if the coughing is intense. In metapneumonic cases the sputum is sometimes grass-green. The sediment almost invariably reveals *shreds of lung tissue* (Traube and Leyden) and yellowish-

green or gray flocculi; these are absolutely essential to diagnosis and consist of elastic fibres to which alveolar epithelium adheres. *Hæmatoidin* crystals or platelets, short sheaths of *fatty crystals, bacteria* (v. s.) and in chronic cases *cholesterin* and scar-like connective tissue are also found. The sputum is voided periodically, as from cavities of other causation; "mouthful expectoration" is the rule. (*b*) The *decubitus* varies with the location of the cavity; for instance, the patient assumes the erect posture if the cavity is in a lower lobe, to prevent leaking of the secretion into the contiguous air passages. (*c*) *Signs of cavity* are found if the exulceration is large and superficial; 80 per cent. of them occur in the lower lobes. They are essentially those of tuberculous or bronchiectatic cavities (*q. v.*) and vary with the stagnation or evacuation of the pus; thus a tympanitic note, bronchial or amphoric breathing and increased vocal fremitus are found when the *vomica* is empty, and dulness, weakened breath sounds and decreased fremitus are present when it is full. The *x*-rays also serve to localize, but when their findings and the physical signs differ the latter are more trustworthy. (*d*) *Hectic fever,* sweats, rigors and leukocytosis occur from resorption.

Diagnosis.—The etiology and cavity formation are often, and the sputum is always, characteristic. (*a*) *Abscess breaking into the lung* from the spine or from the subphrenic or pleural spaces, is sudden, sometimes suffocative in onset, and the hæmatoidin crystals and shreds of pulmonary tissue are absent. (*b*) *Gangrene* (*v. i.*). (*c*) *Tuberculosis* usually occurs in an upper lobe; abscess in a lower one; elastic fibres and hæmatoidin crystals are more abundant in abscess, and tubercle bacilli are found in tuberculosis. (*d*) In *bronchiectasis* elastic fibres are seldom voided.

Prognosis.—Spontaneous recovery may follow granulation and cicatrization, especially in lobar pneumonia, where recovery is the rule. In influenzal or aspiration pneumonia or in sepsis the outlook is far less favorable; marasmus, hectic fever, amyloidosis, lung induration, empyema, pneumothorax and pericarditis are frequent issues.

Treatment.—(*a*) The *general strength* should be maintained by a full diet, tonics and alcohol. (*b*) *Decomposition,* if present, should be modified as in bronchiectasis or gangrene, by administration of creosote or turpentine internally. (*c*) *Surgical intervention* is indicated, according to Fränkel, (i) when the abscess is large, shows no tendency toward spontaneous cure and the contents decompose; (ii) when rupture into the pleura occurs; and (iii) when the abscess results from the breaking into the lung of a contiguous focus of suppuration. Of Körte's 78 operative cases, 80 per cent. recovered; in Garré's 96 cases, 80 per cent.; in Tuffier's series, 77 per cent.; in Freyhan's acute cases, 90, and in his chronic cases 14 per cent.

GANGRENE OF THE LUNG.

Definition.—A necrosis and mortification of lung tissue. Gangrene, though more common than abscess, is an infrequent affection; Eich-

horst found gangrene in 0.1 per cent. of his clinical cases and Hensel in 1.6 per cent. of his autopsies.

Etiology.—Reduced physiological resistance, *e. g.,* from diabetes, is apparently a necessary etiological factor. *Lobar pneumonia* may terminate in gangrene when, as in abscess, the patient is weakly, diabetic, addicted to alcohol or suffering from putrid bronchitis; its frequency is but 0.4 per cent. (Fränkel) or in less than 1 case in 1,500 pneumonias (Aufrecht); pneumonia causes 23 per cent. of lung gangrene (Coupland and Hensel). *Lobular pneumonia* is less frequently causal, though grippal pneumonias may terminate in gangrene (7 per cent. in Fränkel's series). In Coupland and Hensel's combined cases, 14 per cent. were due to *pulmonary tuberculosis* and 10 per cent. to *tumor of the lung.* Gangrene may complicate lung abscess or echinococcus. *Aspiration pneumonia* resulting in gangrene occurs particularly in insane, delirious, paralyzed or emaciated subjects; foreign bodies, necrotic diseases of the throat, bronchi or larynx, bronchiectasis, putrid bronchitis and bronchial stenosis promote gangrene. *Embolic gangrene,* a cause of 19 per cent. of lung gangrene, may result from suppurative gangrenous lesions in peripheral parts, as in the appendix, uterus or extremities. *Rupture into the lung,* as of œsophageal cancer or diverticulum, putrid pleurisy, subphrenic abscess or gastric ulcer may cause gangrene. Gangrene usually occurs within the *second to fourth decades* of life and *in males.*

Bacteriology.—Some maintain that certain germs produce the necrosis and other germs the gangrene, but probably one microörganism may produce both changes. The staphylococcus is found, also the streptococcus, colon bacillus and pseudotubercle bacillus which retains its stain when treated with acids like the bacilli found in putrid bronchitis and bronchiectasis; the Bacillus pyocyaneus and the Micrococcus tetragenus are also found; Hirschler and Terray have cultivated a micrococcus which liquefies gelatin and produces skatol and indol, and therefore causes a putrid odor in the cultures. Babes described bacilli resembling those of malignant œdema.

Pathology.—Genuine gangrene must not be confused with softening of the lung, which is due to regurgitation of the gastric juice into the lung during the death agony or after death. In rare cases there is non-putrid pneumomalacia, analogous to myomalacia cordis. Lung gangrene, according to Hensel, occurs in the right lung in 45 per cent., in the left in 37 per cent. and in both lungs in 18 per cent. of the cases. Laennec, who first described pulmonary gangrene, distinguished *two types,* the circumscribed and the diffuse. (*a*) *Circumscribed* gangrene occurs chiefly in the lower lobes near the surface, and oftener on the right than on the left side. The focus may attain the size of an orange. Laennec distinguished three stages: (i) The formation of a yellow, green or brown slough, which may be expectorated entire; (ii) liquefaction, or moist gangrene; and (iii) formation of a cavity which is irregular in shape, has ragged walls and contains a stinking liquid resembling the sputum of gangrene

(*v. i.*). The cavity is limited by granulations and later by connective tissue, or gradually extends, corroding more and more lung tissue. Extension may also result from aspiration of the septic matter from the bronchi into other lung areas. Recovery may follow localization, organization of granulations and contraction of connective tissue. Microscopically there is (i) an inner zone of offensive fluid or necrotic tissue, bacteria and emigrated leukocytes; (ii) outside of this is a zone of granulations, beyond which is (iii) congested or œdematous lung. (*b*) The *rarer diffuse gangrene* has no demarkation, and a large area, an entire lobe, or even the whole lung may be macerated, stinking and discolored. The diffuse form occurs more often in the upper lobes, and in the right more frequently than in the left side. In both forms the sputum irritates and injects the bronchial mucosa, the bronchial glands are tumefied and degeneration or metastasis may occur in various viscera.

Symptoms.—1. Sputum.—The sputum is characteristic and almost absolutely essential to a diagnosis, and it is absent only when the putrid focus has no bronchial connection. Its odor is extremely offensive, fæcal, stinking, garlicky or sometimes sweetish. This odor is less marked in diabetics; it is most offensive at the moment of evacuation, and on standing may gradually lessen. The sputum, which is *voided periodically* and sometimes in mouthfuls, is usually abundant (a pint to a quart), is alkaline, and on standing separates into three layers, an upper layer of foam, a middle of green or gray serum, and a lower one of green or brownish granular sediment. In the sediment various important structures are found; there are *flocculi* of lung tissue, which are seen best against a dark background, and which vary in size from small particles to pieces as large as the thumb and show alveolar arrangement; elastic fibers are not nearly as common as they are in abscess of the lung, unless the process is very acute, for a trypsin-like ferment rapidly digests the fibers; *Dittrich's plugs* are usually found, consisting of detritus and bacteria, and emitting a most fœtid odor (described under Putrid Bronchitis and Bronchiectasis); *long fatty needles* (Virchow), lung pigment, pus cells, red corpuscles, hæmatoidin crystals, bacteria, infusoria, triple phosphates, leucin, tyrosin, formic and other allied acids, phenol, indol, skatol, ammonia and sulphuretted hydrogen are also found.

2. Chest Findings.—Besides the findings of *bronchitis* caused by the offensive sputum, signs of infiltration or cavity formation are obtained if the focus is superficial and measures two and a half inches. In the *common circumscribed form* the signs of cavity are identical with those in tuberculosis, bronchiectasis or abscess, *i. e.*, a tympanitic note, increased vocal fremitus, bronchial or amphoric breathing and metallic rales are obtained when the cavity is empty, or dulness, decreased fremitus and weak breathing when it is full. In the *rare diffuse form* there are signs of infiltration as in pneumonia, which signs are also found in the initial stage of the circumscribed gangrene. The *x*-rays are valuable in localization of the gangrene. The

patient lies so that the cavity is dependent, to avoid constant trickling of the secretion into the bronchi, *i. e.*, on his back when it is posterior or on his face when it is anterior or in an upper lobe.

3. General Symptoms.—General symptoms result from the causal disease or from sepsis. Remittent fever, chills, sweats, prostration, emaciation, pallor, rapid bounding pulse, vomiting, diarrhœa, leukocytosis, albuminuria and in severe cases a profound typhoid state mark the degree of intoxication.

4. Complications.—Complications may dominate the clinical picture. (*a*) Pleurisy (in 12 per cent.), usually purulent or putrid, may be one of the chief symptoms, and operation may evacuate most offensive fluid with large fragments of necrotic lung. (*b*) Hæmoptysis is seldom fatal though frequently profuse, and the blood raised may be dark brown. (*c*) Pyopneumothorax, or (*d*) rupture into the thoracic wall, œsophagus, aorta, mediastinum, pericardium or peritoneum may occur. (*e*) There may be metastatic deposits in the brain, liver, joints or muscles. (*f*) Clubbed fingers occasionally result from septic absorption. (*g*) Amyloid degeneration sometimes results from chronic gangrene.

Course and Prognosis.—The course may be acute, subacute or chronic; in general it is more rapid and usually fatal within a week in diffuse and more chronic in circumscribed forms. The prognosis depends on the etiology, physical strength of the patient, degree of absorption, localization and surgical accessibility (*v.* Therapy).

Diagnosis.—Exploratory puncture is dangerous, for it favors diffusion. (*a*) In *fœtor ex ore* the breath is fœtid but the sputum, if any, shows no characteristic findings of gangrene. (*b*) In *putrid bronchitis* the sputum shows Dittrich's plugs and fœtor, but no fatty crystals, elastic fibers or pulmonary tissue. (*c*) In *abscess* elastic fibers and pus are more abundant, the fatty crystals are oftener in sheaths than in long needles and hæmatoidin crystals are more common; bronchial plugs and fœtor are absent. (*d*) In *bronchiectasis* elastic fibers are very rare and lung tissue has never been found. (*e*) In *tuberculosis* coincidence of the two affections and the pseudotubercle bacillus may alone cause confusion (*v.* Tuberculosis)

Treatment.—1. Surgical.—Expectant treatment results in a mortality of 53 per cent. (Eichhorst) to 80 per cent. (Lenhartz). In 15 personal cases, 66 per cent. recovered under medical treatment. With operative treatment Tuffier gives the death-rate as 40 per cent., and Freyhans 30 per cent.; Garré in 122 cases records 34 per cent. of fatalities. The surgical indications are (*a*) the vital indication when acute gangrene assumes the *foudroyant* type, or (*b*) when, in chronic forms, there is no tendency toward spontaneous healing. Bull reports recovery even in multiple gangrene. Accurate localization by physical findings or by the *x*-rays was possible in 65 per cent. of Tuffier's 74 cases.

2. Expectant Treatment.—This is indicated in deep-seated gangrene, in hopeless metastases as in the brain or in apparent demarka-

tion. The patient should be kept on his back to avoid leakage into sound bronchi. The diet should support the strength and include full alcoholic stimulation. Carbolic acid, balsam of Peru or creosote may be given internally. Turpentine is most efficacious when there is a complicating hemorrhage (Skoda).

TUMORS OF THE LUNG.

I. Carcinoma.—Cancer of the lung may be primary or more often secondary to that of the mamma or the digestive or genital organs. It occurs in 0.17 per cent. of autopsies or in 1.8 per cent. of cancer autopsies (Pässler). Most primary cases develop between forty and sixty years of age. In primary forms Pässler found 73 per cent. in males; the secondary forms are more frequent in women.

Etiology.—Trauma is an uncertain factor. Cancer usually develops in the bronchi, in which previous ulceration, syphilis or adherent rupturing lymph glands are predisposing factors. Carcinoma probably may develop from the bronchial mucous glands or even from the alveolar epithelium. Histologically the cylindrical type is by far the most frequent, though medullary, scirrhous and other forms may be encountered. Cancer usually begins at the hilum, and may extend toward the pleura, either by contiguity along the bronchi or by the lymph spaces. The upper lobes are more often involved than the lower and the right twice as frequently as the left. The disease is generally unilateral. In 85 per cent. of cases metastases occur in the liver, bones, brain, mediastinum, testes and kidney. Secondary cancer is generally bilateral; the lungs are strewn with miliary nodules resembling miliary tubercles, or present many larger nodules. Calcification and ossification may occur in tumors of the lung.

Symptoms.—With a primary accessible tumor in another part of the body, cyanosis, dyspnœa, blood-stained sputum and signs of lung infiltration or of cavity, facilitate a diagnosis of lung metastasis. When there is a hidden primary focus or a primary lung tumor, diagnosis is for a long time difficult or impossible.

1. Early General Symptoms.—These are usually ambiguous; they are suggestive of tuberculosis, pleurisy or bronchitis, all of which may indeed complicate neoplasm. Thoracic oppression, asthmatic attacks, stridor and *dyspnœa* result from pressure on the trachea and vagus or wide substitution of tumor for lung tissue. Cyanosis, dyspnœa, rapid breathing, emaciation and sweats may suggest miliary tuberculosis; this resemblance may also be noted at autopsy in miliary carcinosis; fever develops in over half the cases. Cough is common.

2. Sputum.—The sputum in over half the cases is rather characteristic. Altered blood gives it a reddish-brown color or it resembles raspberry jelly in hue and in consistency; it is sometimes grass-green or prune-juice in color. Occasionally macro- and microscopic bits of cancer tissue, in characteristic nest-like arrangement, are seen.

3. Physical Signs.—(*a*) *Inspection:* The patient usually lies on the affected side to give freer play to the sound lung. Marked *ectasia* may result; sometimes the diseased side is shrunken from complicating lung cirrhosis, atelectasis or bronchial stenosis. (*b*) *Palpation* elicits *increased fremitus* if the tumor grows into the bronchial lumen or compresses it from without. (*c*) *Percussion* gives an *irregular dulness,* increasing to absolute flatness, in outlining which the finger perceives greatly *increased resistance;* it most often occurs over an upper lobe or under the sternum. (*d*) *Auscultation* varies with the patency of the bronchus, giving *bronchial breathing* when it is open and weak or *absent breathing* when occluded.

4. Compression Symptoms.—Besides the early dyspnœa and cough, (*a*) the heart is often luxated or its yielding auricles are crowded. (*b*) The venous trunks suffer compression with frequent cyanosis and œdema of the face, neck and arms and with dilatation of other veins of the thorax, due to formation of a collateral circulation. (*c*) There is compression of the vagus, with rapid or slow heart, and recurrent laryngeal compression with phonic changes. (*d*) Dysphagia, (*e*) intercostal neuralgia, brachial neuralgia or paralysis, and (*f*) bronchial stenosis are other symptoms of compression.

5. Metastasis.—This occurs in 85 per cent. of cases; there may also be extension, by contiguity, to the axillary or cervical glands.

6. Complications.—These include hæmoptysis from necrosis and cavity formation; gangrene and bronchiectasis; and pleurisy, which is usually serous; hemorrhagic effusions occurred in 12 per cent. of Moutard-Martin's 200 cases; the fluid may resemble venous blood; adipose or chyliform effusions are rare; hydropic, fatty and granular cells may be obtained by puncture, and Hellendall advises aspiration for flocculi of tumor tissue, with the syringe and needle partly filled with salt solution, before aspiration. Carcinomatous nodes may develop at the site of puncture, though hard nodules at the site of tapping not infrequently occur after puncture of simple pleuritis.

II. Sarcoma and Lymphosarcoma.—Sarcoma and lymphosarcoma may be primary or secondary, and are more frequent than cancer. They probably begin chiefly in the lymph nodes of the bronchi, and oftener on the left than on the right side. Extension in all directions occurs along the peribronchial tissues from the hilum. A primary very malignant lymphosarcoma develops in the lungs of miners in the cobalt mines of Saxony; it causes 75 per cent. of the deaths among them; secondary sarcomata in the liver are common. Ulceration is less common than in cancer, but the symptoms are, in the main, identical. Sarcomatous tissue is occasionally voided in the sputum or aspirated by the exploring needle. The diagnosis of malignant lung tumors must be made chiefly from aneurysm (*q. v.*) and pleurisy (*q. v.*). The *x*-rays may be of some value. There are three main clinical groups, (*a*) in which a neoplasm, perhaps its exact type, can be diagnosticated; (*b*) in which some growth is present but differentiation from aneurysm is uncertain; and (*c*) in which the disease entirely escapes detection or is only suspected.

Prognosis.—Death generally occurs in less than a year with cachexia or suffocation, but it may result in a few weeks after the first symptoms appear.

III. Other Tumors of the Lung.—Fibroma, lipoma, osteoma, hypernephroma and enchondroma are of solely anatomical interest.

Treatment.—The treatment of tumors of the lung is only palliative. Morphine must be used when the dyspnœa and cough are extreme. The *x*-rays offer little encouragement. Arsenic seems to help when given in full doses.

CIRCULATORY AFFECTIONS OF THE LUNG.

I. Acute Congestion.—As a secondary lesion acute hyperæmia occurs commonly with acute tuberculosis, pneumonia, influenza, effusive pleurisy, with inhalation of hot vapors or with cardiac strain; when extensive lesions occur in the lungs the obstructed blood may seek other parts of the lungs and possibly produce *collateral congestion,* though this is justly disputed. French writers describe an *acute primary congestion* to which they give the name of Woillez (*Maladie de Woillez*), though previously described by Dechambre, Legendre, Bailly and Fournet. Its status as an independent lesion is doubtful; some cases are apparently larval or abortive pneumonia which occurs in any large epidemic. According to Carriére it is due to attenuated pneumococci, which he has demonstrated by puncture of the lung. Cold, exposure, alcoholism and violent exertion are alleged causes. It develops like pneumonia, with chill, fever of 101° to 103°, pain in the side, dyspnœa, cough and a sputum which is at first scanty and later is more abundant and viscid but not blood-stained. Physical examination is said to elicit somewhat impaired resonance, weak or occasionally bronchial breathing, rales which are coarser than in pneumonia and at times a pleural rub. All these symptoms and signs strongly suggest atypical fibrinous pneumonia. Venesection and vasodilator remedies, as aconite or veratrum, are said to be of benefit.

II. Passive Congestion; Hypostasis.—In broken compensation, particularly in mitral stenosis, the lungs are distended, heavy, indurated, pigmented and on section are brownish-red; this is brown induration (*induratio pulmonum fusca*). Histologically the capillaries are turgid, the connective tissue is increased, the lungs are tinged with altered blood pigment and the sputum contains the so-called heart disease cells (see SYMPTOMS OF VALVULAR DISEASE). The breathing is sometimes puerile and the lungs voluminous.

Hypostasis is a species of passive congestion which results from weak heart, in typhoid and other fevers, in old age, cachexiæ, marasmus, in opium and other narcoses or in apoplectic and other comas. Even under these conditions it rarely occurs if the patient's position is changed every few hours. The lung is dark red, engorged with blood and serum, and may also show aspiration pneumonia and atelectasis; the latter conditions are often incorrectly called hyposta-

tic pneumonia. On physical examination, there is moderate dulness over the lower lobes posteriorly; in apoplexy this may occur only on the paralyzed side; the breathing is more often weak than bronchial, and moist or sometimes crepitant rales are heard. Fever is no part of hypostasis.

Treatment.—Treatment of passive congestion is that of the fundamental lesion; phlebotomy, cardiac stimulation and in hypostasis, frequent change of posture and cardiants are indicated.

III. Infarct; Embolism.—Etiology.—Embolism was first observed by Laennec; Virchow, Cohnheim and Litten established its pathology. The most frequent source of lung embolism is (*a*) fibrinous coagula from the right heart, the frequency and mechanism of which have been discussed under Acute and Chronic Endocarditis and Septic Infections. Clots may also reach the lung from peripheral thromboses, and are either simple (*i. e.*, non-suppurative, marantic) or inflammatory and septic. Rarer causes are (*b*) tumor or hepatic tissue, (*c*) fat embolism after fractures and liver injury, (*d*) air embolism or (*e*) echinococcus cysts.

Pathology.—The embolus may obstruct a main branch of the pulmonary artery, straddle one of its secondary bifurcations or lodge in a smaller vessel. Not every embolism produces an infarct, for death may be instantaneous, the occlusion incomplete or collateral circulation possible. Occlusion of an end-vessel results in the hemorrhagic infarct, which is often wedge-shaped with its base toward the pleura, is dark at first and later lighter, is airless, on section projects above the cut surface, often measures 2 to 6 cm., and is attended by extravasation of blood into the lung cells and sustentacular tissue, though the lung is not torn. This is known as a fibrinous or *simple infarct.* If the embolus contains pus or putrefactive microörganisms, it develops *abscess* or *gangrene.* Infarcts occur in the right lower lobe in 46 per cent., right upper in 21, left lower in 15, left upper in 12 and right middle lobe in 6 per cent. (Tiedermann). They are often multiple. Small infarcts may be wholly absorbed, but the larger ones leave pigmented cicatrices.

Symptoms.—(*a*) Infarction of the main pulmonary trunk may result in immediate death, as sudden death from sitting up after childbirth (two-thirds of the puerperal cases die). In other cases death occurs in a short time from cerebral anæmia attended by convulsions, dyspnœa and cardiac failure. The infarct may cover the greater part of the lobe involved; if the patient lives for some time, a sudden systolic murmur may develop over the pulmonary artery (Litten). (*b*) Infarction of a smaller vessel is often ushered in with chill, vomiting, dyspnœa, cyanosis and fever, even with bland embolism. The sputum contains dark blood and there is a "stitch in the side" from pleuritic participation. Physical examination frequently elicits a pleural rub and later, in some cases, slight dulness, especially behind, between the spine and scapular line; crepitant rales and bronchial breathing are frequently present. Pyogenic or putrid clots cause abscess, gangrene, empyema or pneumothorax.

Diagnosis.—Diagnosis is often at best merely a probability. Sudden cardiac failure is very difficult to differentiate from an embolic occlusion of a main pulmonary trunk, especially when death occurs without hæmoptysis. The etiology, the sudden onset and circumscribed chest signs are of diagnostic importance. Treatment lies solely in cardiac stimulation and prophylaxis, in rest during endocarditis, cardiac insufficiency or phlebitis.

IV. Œdema of the Lungs.—Etiology.—Pulmonary œdema was described by Laennec (1819). In the etiology of this secondary, common, frequently terminal or agonal affection, *three important causes* stand in the foreground, *viz., heart, kidney* and *lung disease.* Œdema consists of the pouring out into the air cells and interstitial tissue of a serous transudate. A precise classification of the mechanism of pulmonary œdema is difficult:—(*a*) *Stasis* due to weakened heart is probably its most frequent mechanism. Cohnheim and Welch demonstrated experimentally that it may result from failure of the left ventricle, whereas there is continued activity of the right ventricle; this may be common in the death agony, but the explanation is not entirely satisfactory, as cases of pulmonary œdema occur in which the pulse (and therefore the action of the left ventricle) is strong; however, general cardiac anasarca may occur when the radial pulse is apparently sufficiently strong, though it is known that the heart must be weak. Grossmann advances the theory that there is a spasm of the left ventricle. Weak heart may result from any disease of the endo-, myo- or pericardium. Sahli explains certain cases by (*b*) *paralysis of the pulmonary vessels.* Under either (*a*) or (*b*) nephritic œdema or the toxic œdemas, such as those from the iodides, ether, adrenalin, pilocarpine or muscarin may be included. (*c*) *Inflammatory œdema,* such as that around a fibrinous pneumonia and numerous other pulmonary diseases, cannot be strictly brought under œdema, for it is an exudation, not a transudation. However, in this form the pulse is usually strong and there is often fever. (*d*) *Angioneurotic* œdema is sometimes described. It is also possible that (*e*) *occult changes in the blood and bloodvessels* are causal factors. These varied mechanisms, though generally stated to be most common in cardiac, renal and pulmonary lesions, may also operate in cachexia, anæmia, cerebral disease, old age and innumerable other conditions.

Pathology.—Pathologically œdema may be circumscribed or oftener diffuse. The lungs are large, heavy and spongy; they pit and on section, especially of the bases, a frothy serosanguineous fluid exudes.

Symptoms.—Symptoms usually come on *precipitately,* with hurried breathing, dyspnœa, cyanosis and carbon dioxide intoxication. The *sputum* is copious, foamy, serous, somewhat resembling soapsuds or at times tinged with blood; it is rich in albumin and poor in mucus and cells. The *bubbling breathing* indicates serum in the alveoli. The second pulmonic sound is accentuated, the pulse is weak, the lung note is slightly tympanitic from relaxation and coarse and

fine rales are heard everywhere. If the lower lobes are filled, the physical signs may closely resemble those of consolidation, *i. e.,* there may be increased fremitus, bronchial breathing and dulness. The *diagnosis* is determined from the sputum and lung findings.

Treatment.—Treatment may be beneficial in some cases, though too often œdema is the death agony itself. (*a*) *Prophylaxis* concerns the causal disease, for example, cardiac weakness or anasarca in nephritis. (*b*) Rapidly acting, *diffusive stimulants* are urgently indicated hypodermatically, as camphor, ether, strophanthus and strychnine, and coffee or champagne by mouth. (*c*) *Morphine* with atropine is at times almost specific. (*d*) *Venesection* relieves the right heart and therefore the lungs. (*e*) *Stimulating expectorants,* as senega, and *rapid purgation* by croton oil, ♏iij, on the tongue, are sometimes of value. The author has obtained no results from the acetate of lead, recommended by Traube in doses of gr. ss to i every hour and by Nothnagel in a single dose of gr. x to xx.

V. Hæmoptysis.—Hæmoptysis or hæmoptoë refers to "spitting of blood," while profuse hemorrhage is known as pneumorrhagia.

Etiology.—(*a*) Laryngeal ulceration or inflammation; (*b*) tracheal ulceration or aneurysm bursting into the trachea; (*c*) severe inflammation or ulceration of the bronchi, stasis from cardiac disease, bronchiectasis, putrid and fibrinous bronchitis, rupture into the air passages of aneurysm or anthracotic lymph glands, and thermal, chemical or mechanical irritation are etiological factors; hæmoptysis may also occur in the acute exanthemata, in such affections of the blood as hæmophilia, scurvy, purpura hæmorrhagica and leukæmia, and in gout, renal disease and cholæmia; hæmoptysis occurring as vicarious menstruation was mentioned by Hippocrates, and though rare, is an established variety; vicarious hæmoptysis after ovariotomy or suppression of hemorrhoidal bleeding is still more uncommon and its existence has been regarded with just scepticism. (*d*) Lung disease is the most important etiological factor, and the chief lesion is (i) tuberculosis (*q. v.*), which causes 80 to 90 per cent. of cases of blood-spitting. It is commonest between the fifteenth and thirtieth years, and very rarely occurs under six years of age, as children seldom develop cavities. As stated under tuberculosis, it occurs as capillary or profuse hemorrhage. It was noted long ago that hæmoptysis sometimes occurs rather epidemically among tuberculous cases; in such an outbreak, Flick, Ravanell and Erwin found the pneumococcus in the sputum. (ii) Infarcts are usually characterized by hæmoptysis. (iii) Pneumonia, abscess and gangrene; (iv) tumors; (v) parasites of the lung, trauma; and (vi) syphilis and leprosy, are more uncommon causes. (*e*) Hæmoptysis nervosa is most rare, though authentic cases have occurred in cerebral traumatism, hysteria (where it has caused death), epilepsy and in other neuroses. (*f*) A toxic hæmoptoë may occur from mercury or iodides, but it is very rare.

Symptoms.—Symptoms follow those of the causal tuberculosis, mi-

tral stenosis or aneurysm, or develop in apparent health. Hæmoptysis *is in itself only a symptom.* Blood poured out into a cavity or into the lung substance is not always voided, especially in very weak or moribund persons. Prodromes are unusual; *without warning,* a salty taste in the mouth, substernal warmth, a sense of something rising from the chest and coughing are followed at once by a few spoonfuls or more of blood. At times a previously mucous sputum may be accidentally noted to be blood-stained. The blood varies in amount from the merest stain to 5 quarts (Laennec); it may appear but once or may recur repeatedly. It is generally bright red and well mixed with air; the formation of rouleaux is uncommon. If the blood has clotted in the lung or air passages, the clot, too, is mixed with air and perhaps with mucus. Casts of the bronchioles may be voided. If there is gangrene or abscess, the blood may be brown or buff-colored and the red cells may be disintegrated. Brown hæmosiderin granules or, if the blood has remained as a clot two weeks, hæmatin crystals may occur. Asthma crystals are occasionally seen.

Physical Findings.—Physical findings, other than those of the fundamental condition, are frequently lacking; if the blood stagnates in a fairly large bronchus, there may be bronchial breathing, bronchophony, increased fremitus and dulness if the affected area measure 2 by 5 cm. In the bronchioles, blood gives rise to small rales, in the bronchi to coarser rales.

Complications.—Fever, aside from the causal disease, may result from resorption (ferment fever). Urobilin icterus, functional heart murmur, relaxation of the right heart, albuminuria and puffiness of the ankles are possible but not conspicuous complications.

Diagnosis.—Examination of the nose, gums, throat and larynx readily excludes hemorrhage which may simulate hæmoptoë. It is often difficult to distinguish at the time of bleeding between bronchial and pulmonary bleeding, but the question may be decided by the history, later physical examination and the clinical evolution.

	Hæmatemesis.	*vs.*	Hæmoptysis.
(*a*)	The blood is acid (unless a very large amount is voided).		Is alkaline.
(*b*)	The patient nearly always recognizes whether it is vomited or coughed up. (Blood from the lung is very rarely swallowed and then vomited.)		(Hæmoptysis, very rarely, causes consensual vomiting.)
(*c*)	Is often dark (though large amounts of bright red blood may be vomited, as in ulcer).		Nearly always bright (though when stagnating in a cavity it may become dark or buff).
(*d*)	Is mixed with food, HCl, ferments, etc.		Mixed with air, which is also found in the clots. (Not infrequently tubercle bacilli or heart disease cells are found.)
(*e*)	In 16 per cent. of cases blood is in the stools.		Is very rare in stools.

Prognosis and Course.—The duration is variable, but spontaneous cessation is the rule. The immediate prognosis is good, as early death from hemorrhage is rare, except in cases of profuse pneumorrhagia, resulting, for instance, from aneurysmal rupture, but even then the patient may live years after the aneurysm has broken into the lung substance. Drowning or suffocation of the patient in his own blood is rare. The final outcome depends on the etiology. In Ware's 386 cases 62 per cent. recovered; early death occurred in 3 per cent.

Treatment.—PREVENTION.—In tuberculosis (*q. v.*) the patient should be kept quiet, the cough suppressed and exertion and vocal efforts avoided; in heart disease and marantic thrombosis, rest is indicated to obviate detachment of the thrombi.

THERAPY.—*Absolute physical and mental quiet* should be enforced when hæmoptysis occurs. No movement should be allowed; coughing should be suppressed by hypodermatic injections of morphine, which also allay the attendant mental excitement (opiates are employed guardedly in very large hemorrhages, for bronchial occlusion may result). The patient should be assured of a favorable outcome; no physical examination should be made except auscultation of the chest in front, for immediate treatment takes precedence of diagnosis; if rales indicate the side of the hemorrhage, the patient should lie on that side to prevent aspiration into the contralateral bronchus.

Arterial pressure should be reduced by administration of amyl nitrite or aconite, which lessens tension in the pulmonary and arterial circulation. A liquid, simple, spare diet should be given. Alcohol, coffee and tea should be interdicted. Even if the patient collapses cardiants should be avoided, for syncope is nature's readiest hæmostatic; the old practice of venesection in this way promoted thrombosis. Cardiac excitement is lessened by placing a large ice-bag over the precordium. Active purgation by croton oil is very beneficial.

Hæmostatics are not proven to control the lesser blood circuit, which appears to be rather independent of drugs which operate on the arterial circuit. Ergot, if anything, rather raises the blood pressure in the lungs and should therefore not be given. Calcium chloride gr. v to x, and common salt on the tongue are of some value (salt acts reflexly, by way of the vagus, on the pulmonary vessels). Gelatin (2 per cent.) may be used in urgent cases and the beneficial effects of turpentine have long been known. Nature, as a rule, effects the cure; drugs do not, though opiates reinforce nature. Hippocrates employed ligature of the extremities, *i. e.*, binding tightly for a few minutes the vessels of the groin and axilla, in order to lessen the return flow of blood toward the point of hemorrhage. Artificial pneumothorax, to compress the bleeding lung, has been advocated in cases of desperate profuse and persistent hæmoptysis.

PARASITES OF THE LUNG.

Echinococcus of the Lung.—Man may become infected with the tinea echinococcus from dogs by their licking their master's hands or from fæces contaminating human food, especially vegetables. The unilocular form is much more common than the multilocular. Infection occurs either (*a*) primarily, the embryo skipping the liver, or (*b*) much more often secondarily, by metastasis or breaking through the diaphragm from the liver. In Neisser's 900 cases, 50 per cent. occurred in the liver, 9 per cent. in the kidneys, 8 per cent. in the cranium and 7 per cent. in the lungs. The small miliary white cysts may grow to the size of an adult's head. They may calcify, indurate, suppurate, putrify or rupture into contiguous parts (see ANIMAL PARASITES).

Symptoms.—Many cases produce no symptoms and are unexpected findings at the post mortem. Dieulafoy described three clinical stages:—(*a*) the initial, (*b*) the tumor stage and (*c*) the suppurative or rupture stage. (*a*) The *initial stage* often has no symptoms, but may be marked by scanty hæmoptysis, cough, emaciation and possibly fever, all of which may closely simulate incipient phthisis. (*b*) In the *tumor stage,* there are bronchial (or weak) breathing, increased fremitus and circumscribed dulness, which occur in the lower lobes and twice as frequently on the right side as on the left; it sometimes causes bulging of the chest wall. There is a sharp transition between it and the note of the sound contiguous lung. Dislocation of the heart and compression of the lung may occur. The *x*-rays often confirm the physical signs. The dulness curves upward, but is quite different from the curve of pleuritic effusion which is concave upward. Tumor of the lung gives a larger, harder flatness. Puncture (*v. i.*) reveals a clear, aseptic fluid of 1,009–15 specific gravity with little or no albumin, very rich in sodium chloride and the characteristic succinic acid; the inosite is of no practical importance; the hooklets are pathognomonic. (*c*) In the *stage of suppuration or rupture,* as in liver echinococcus, rupture may occur into the pleura (in which primary disease is 25 times as rare). Pneumothorax may result. Far oftener rupture into a bronchus occurs, with pain, dyspnœa, collapse and sudden "mouthful" expectoration of pus, small vesicles, lamellated cyst wall and hooklets. At the time of rupture a curious urticaria develops (Finsen) which is probably toxic, as the fluid from echinococcus cysts produces it when inoculated into animals. Profuse hæmoptoë is common. If it comes from the liver, the sputum is ochre-yellow from undissolved bilirubin, even when there is no direct connection with the bile vessels; cholesterin is often present. The vesicles or cyst wall may cause suffocation. Rupture may also occur into the pericardium, stomach or peritoneum. In this stage hectic fever is usual.

Diagnosis.—Diagnosis is determined only from the sputum, or from puncture which has decided dangers; sudden, probably toxic death was observed 11 times by Maydl.

Prognosis.—The course varies much. A cyst in two or three months becomes as large as a hazelnut, and in five months as large as an orange (Dieulafoy). Death may result from suffocation, hemorrhage, gangrene, hectic fever or toxæmia from rupture or puncture.

Treatment.—If puncture is made and is followed by severe symptoms, the case should be instantly operated upon. There is no medical treatment. Without surgical intervention 57 per cent. (Hearn) to 66 per cent. (Davaine) die. Tuffier and Garré report but 10 per cent. mortality with operation.

Distoma of the Lung.—This affection was first described by Bälz in Japan, Formosa and Corea. The worm is plump, wide, egg-shaped, measures 8 to 10 x 4 to 6 mm., is brownish red and is provided with two small suckers. Its eggs are oval, brown and measure 0.08 to 0.1 x 0.05 mm. Little is known of its origin. It is found in the bronchi and in the infarcts and cavities, which it produces. The cavities contain red disks, a mucoid substance and shreds of lung tissue, and are surrounded by fibrous tissue. The parasite is also found in the liver, diaphragm, mesentery and brain. The course of the disease is chronic, though most cases terminate favorably; it is marked by (*a*) bronchitic symptoms, (*b*) hæmoptysis and (*c*) a viscid clear sputum in which minute brownish-red dots, the distoma eggs, are seen. In exceptional cases death results from emaciation, exhausting hæmoptysis or brain embolism.

Tuberculosis, syphilis and *actinomycosis* of the lung are described under Infections.

DISEASES OF THE PLEURA.

PLEURISY.

Definition.—Pleurisy is an inflammation of the pleura, usually secondary to tuberculosis or other lung disease, general infections, septicæmic affections and disease in contiguous tissues. To Hippocrates, pleurisy meant pain in the side; though Boerhaave first confined the term to pleural disease, the first full description dates from Laennec.

Pleurisy constitutes 3 per cent. of internal maladies and is the most common of all post mortem findings (in over 50 per cent.). It is susceptible of division into infinite varieties, acute, subacute or chronic according to its course, dry or effusive according to its consistence, primary or secondary as to its cause, diffuse or circumscribed as to its distribution and serofibrinous, hemorrhagic or suppurative as to its nature. Any such classification is more or less artificial, whence it seems preferable to describe all its symptoms first and group special types later.

Etiology.—1. Tuberculosis.—So-called primary, spontaneous or cryptogenetic pleurisies are often tuberculous (Barrs, Netter, Lemoine, Vaillard, Coston and Dubville). Landouzy considers that

98 per cent. of all pleurisies are tuberculous and that 60 per cent. of all cases of phthisis pass through the so-called *pleuritis a frigore;* Netter holds that 70 to 80 per cent., and Ashoff that nearly all "primary" cases, are tuberculous. Le Damany in 55 "primary" pleurisies proved by inoculation that 92 per cent. were tuberculous. Lord states that two-thirds of pleuritics have no obvious disease in other organs and three-quarters respond to tuberculin. The causal lesion may be recognized at autopsy in calcified bronchial glands or in small lung foci. Tuberculous pleurisy is often bilateral. Its special relations will be discussed later.

2. Diseases of the Lung Substance.—Pneumonia, abscess, gangrene, infarcts, tuberculosis, tumors, pneumothorax, tertiary syphilis, actinomycosis, etc., are etiological factors; according to Koplik, 95 per cent. of pleurisies in children follow pneumonia. It is less frequent in diseases involving the bronchi, as in grippe or measles.

3. Acute Infections.—Rheumatism, sepsis, endo- and pericarditis, scarlatina, measles, diphtheria and other acute infections may result in pleurisy. It develops in about 2 per cent. of typhoid cases; it may develop in secondary syphilis without lung lesions.

4. Diseases of Adjacent Organs.—(*a*) *Intrathoracic* affections, involving the ribs, spine, œsophagus, mediastinum, lymph glands, diaphragm and pericardium; (*b*) *abdominal* affections, as of the stomach, spleen or liver (abscess, tumor or cirrhosis (in 16 per cent.)), subphrenic abscess, suppurative or tuberculous peritonitis (in 25 per cent.), perinephritic abscess, abdominal malignancy, etc., may promote pleurisy; pleural infection may occur by direct local invasion or by the lymph and blood streams; in neglected appendicitis, hepatic and diaphragmatic invasion are the steps to pleural infection.

5. Tumors.—Echinococcus and endothelioma (Wagner and Fränkel) are the most important types; endothelioma is an infectious process (Neelson), a lymphangitis proliferans (Schwenniger) or a lymphangitis carcinomatosa (Schottelius). Carcinoma, sarcoma, ecchondroma and rarely lipoma are also factors; pleural neoplasms are far more frequently secondary than primary.

6. Altered Conditions of the Blood (Dyscrasiæ).—Gout, nephritis, scurvy and tertiary syphilis are often causative of pleurisy and frequently bilateral pleurisy; reduced physiological resistance predisposes to infection.

7. Terminal Infections.—Various forms of sepsis are often fatal to subjects of chronic disease of the heart, liver and kidneys; bilateral pleurisy is frequent.

8. Age, Sex, Localization.—*Age*: most cases occur between the twentieth and fiftieth year, but no age is exempt. *Sex:* Eichhorst observed pleurisy in 3 per cent. of all his male and 2 per cent. of all his female cases, and found the following *localization*: right-sided in 54 per cent., left-sided in 42 per cent. and bilateral in 4 per cent. Cases following nervous disease, cold or exposure, venous thrombosis

or trauma can usually be brought within the above given classification.

Bacteriology.—The etiological classification above given does not correspond to the bacteriological division. Suppurative pleurisy may result from the streptococcus, pneumococcus, tubercle bacillus or less often from the staphylococcus or typhoid or colon bacillus; serous pleurisy may be due to the tubercle or pyogenic organisms. The frequently negative cultural results have favored the hypotheses that chemical products, as tuberculin in phthisis, may produce pleurisy or that bacteria produce pleurisy and then die. It must be remembered that cultures are made, not from the pleura directly, but from the fluid effused from it. The organisms found are most frequently the pneumococcus, tubercle bacillus and streptococcus; less often the staphylococcus, pneumobacillus of Friedländer, typhoid, colon, influenza and diphtheria bacilli, gonococcus, ray fungus or glanders bacillus. (*a*) *Purulent pleurisy* (empyema) is due most often to the streptococcus in adults and to the pneumococcus in children. About 10 per cent. of empyema is tuberculous; empyema giving no results culturally or by inoculation is usually tuberculous and may be regarded as a cold abscess of the pleura (Kelsch and Vaillard). The staphylococcus, Micrococcus tetragenus and pyogenes tenuis, pneumobacillus, typhoid and colon bacilli are infrequent factors. (*b*) *Serous pleurisy* is usually tuberculous, as shown by the constantly increasing percentage of cases in which inoculations are successful. Tubercle bacilli when found, suggest a direct communication with a tuberculous focus in the lung or lymph glands; negative results are due either to an insufficient amount of fluid (less than 10 to 20 c.c.) being inoculated or to the effusion being due to toxins rather than to the bacilli. Eichhorst found tubercle bacilli by direct examinations in but 15 per cent. of serous pleurisies, though his inoculations were positive in 62 per cent. Jousset recommended that the fluid be allowed to coagulate and the clot digested with pepsin, centrifuged and then stained; his method is termed inoscopy. (*c*) So-called primary pleurisy which is non-tuberculous is usually pneumococcic. (*d*) Meta- and para-pneumonic pleurisy is most frequently due to the pneumococcus, but also to the streptococcus and staphylococcus, even when no pus is formed, although mixed infections have a tendency to suppuration.

Symptoms.—Physical findings are absolutely essential for diagnosis and all subjective symptoms are ambiguous or may be wholly absent. In the main, all types have much the same signs which differ more quantitatively than qualitatively; serous pleurisy nearly always shows friction, and fibrinous pleurisy is almost always accompanied by some exudation.

Inspection.—(*a*) Inspiratory retraction of the intercostal spaces may result from imperfect ingress of air, pain or adhesions. Epigastric retraction during inspiration is due to contraction of the diaphragm. (*b*) The patient often lies during the dry stage on the sound side to avoid pain from pressure on the inflamed surfaces; later,

during the exudative stage, he lies on the diseased side in order to allow a freer expansion of the sound lung, especially when the exudate is large. Traube believed that continued lying on the diseased side aggravated pain by congesting the pleural and subpleural veins. The patient may sit up for breath, or assume the diagonal decubitus (Andral). (*c*) *Ectasia* (distention) of the affected side occurs especially in children, and is determined by mensuration and inspection. The interspaces are obliterated, the costal arch is immobile during respiration, the skin glistening, the spine curved toward the diseased side, the shoulder higher and the nipple and scapula are further from the median line. (The right chest is normally one or two centimeters larger than the left.) The sound side exceeds, by three centimeters, its normal dimensions on account of compensatory emphysema (Gerhardt and Kerlias). In some very large effusions the writer has observed an apparent retraction of the diseased side. (*d*) *Respiratory movement* of the affected side *is decreased* or absent both in dry and exudative pleurisy. Respiration is reflexly decreased on the diseased side, and the breathing lags on the affected side. The frequency of respiration is increased from lung compression, heart luxation, the low diaphragm, fever, pain or paresis of the intercostal muscles. Pain alone may reflexly decrease the respiratory expansion and variation on the same side may occur, there being more movement above and less below. (*e*) The *x*-rays often show a dark shadow. (*f*) Inspection also reveals cyanosis, dislocation of the apex beat and œdema of the chest wall in suppurative pleurisy and occasionally in other forms; (*g*) *absence of Litten's phenomenon* and (*h*) rarely pulsation of the effusion. (*i*) C. L. Greene describes a rhythmic lateral displacement of the heart peculiar to pleurisy: "Such movement is most marked in medium-sized effusions. The heart approaches the affected side in inspiration and moves outward in expiration. The extent of movement often amounts to two inches. It may be measured by fluoroscopic examination, auscultatory percussion, by simple deep percussion of the free cardiac border, or in some instances by mere inspection of the apex beat. Deep breathing and especially forced expiration are essential to the success of the maneuver, and to obtain this morphine must sometimes be administered. Marked rhythmical lateral movement has not been found by the writer in pneumonia, tuberculous infiltrations of the lung, malignant disease of the pleura or lung or in subdiaphragmatic abscess."

Palpation.—Palpation confirms the findings of inspection. (*a*) An up and down (less often horizontal) *friction rub is felt;* it increases on pressure, which also increases the pain and tenderness, and it is the more readily distinguished the coarser the rub and the more capable the chest wall of vibration. The friction develops before the effusion, is often present with it and after it is absorbed. The rub is due to roughening of the pleura, which loses its glistening appearance and looks like glass upon which one has breathed (Klebs); the two raw pleural surfaces scrape on each other. It may be very

transient, lasting a few hours or days, or even disappearing when the patient takes a deep breath; or it may last for months or even years. It is usually absent in obese individuals. (*b*) Pressure on a tender interspace during deep inspiration may cause a sudden contraction of the upper part of the rectus muscle on the side of lesion (Schmidt). (*c*) *Vocal fremitus,* normally greater on the right than on the left side, varies according to the level at which it is tested. (i) Above the effusion it is normal. (ii) Near the spine, over the compressed and relaxed lung it is increased. (iii) *Below, it is weak or absent,* from exudation of thick fibrin or fluid, though less change is observed in dry pleurisy than in the effusive form. Irregular findings are due to the presence of adhesions bringing the lung closer to the chest wall in some places than in others. The condition of the lung and the bronchial secretion are important in diagnosis, whence before testing, the patient should cough and take a few deep breaths. The results are irregular in cases of loculated effusion. (*d*) *By tapping with the finger tips the increased resistance* is clearly palpated. (*e*) Œdema of the chest wall on the diseased side may be inflammatory both in serous and purulent pleurisy, or mechanical and more intense from pressure on the azygos or hemiazygos veins. (*f*) Fluctuation in massive effusions is extremely rare. (*g*) Palpation determines the *dislocated apex beat,* the *dislocated liver or spleen,* the bulging of the diaphragm and sometimes a furrow between the diaphragm and liver (Stokes).

Percussion.—Percussion is negative in dry pleurisy, except when a very thick fibrinous exudate causes dulness and weak breathing; after an effusion has resorbed the residual plastic lymph causes dulness. When there is effusion (*a*) *dulness* is found between the fifth and eighth ribs in the left axilla or over the liver and posteriorly on the right side. Dulness also results from old adhesions, atelectasis of the lung or a very thick chest wall. As in pneumothorax, emphysema of the lungs and calcification of the ribs may somewhat obscure the dulness of an effusion. In doubtful cases the apparently sound side should be percussed first. According to Ferber's experiments on the cadaver, thirteen ounces (400 c.c.) of fluid are necessary in the adult for recognition and four ounces (120 c.c.) in children; post mortem experiments, however, do not exactly fulfill the conditions obtaining during life. The effusion must be one inch (2 c.c.) thick to produce dulness. Sometimes, however, even six to seven ounces (200 c.c.), which produce a dulness two fingers in height, may be recognized. In some doubtful cases smaller amounts may be detected, as proven by puncture, especially in persons having thin thoracic walls, as children and women. Light percussion is necessary when the effusion is small, lest other normal parts be brought into vibration. Before percussion the patient should breathe deeply several times to eliminate partial collapse of the lung and impaired ingress of air from accumulated bronchial secretion. *Percussion is absolutely essential to diagnosis.* Not merely the audible dulness

should be regarded, but also the *palpable sense of resistance,* on light percussion. The dulness usually amounts to *distinct flatness.* In larger effusions, the *flatness,* at first located posteriorly, increases along the spine and laterally in the axilla. In massive exudates, flatness may reach from the lower limit of the pleura to above the clavicles.

The dulness is highest posteriorly as the patient lies on the back. When he sits the line of dulness describes a parabolic curve (courbe de Damoiseau) which is lowest behind and due according to Gerhardt to the uneven thickness of the chest wall or according to Eichhorst to irregular adhesions. If the fluid should effuse with the patient out of bed, the upper level would be horizontal. Dulness is found one to two centimeters higher than the upper level of the fluid, due to plastic exudate. (*b*) Right-sided effusions merge with the liver dulness, but the lower limit of left-sided effusions can usually be determined, because they *invade Traube's semilunar space.* This "half moon" space lies between the fifth and sixth costal cartilages above and tenth rib below, representing the supplementary pleural space and is designated as the "half-moon" space because of the curve at its upper portion, produced by the apex of the heart; its tympanitic note is decreased or replaced by flatness in left-sided effusions (without adhesions). Dulness here may not be confused with that of the spleen, for there is no respiratory excursion in pleurisy, and the dulness lies more toward the heart than toward the splenic region. Scybala or food in the stomach or bowels may also impinge on Traube's space. (*c*) *Respiratory excursion and Litten's sign are absent* in effusions of any size. (*d*) *The change of the upper line of dulness with change of position* has been emphasized by most writers. According to personal experience, it is difficult to detect because adhesions are present at the level of the fluid; the patient must lie hours in another posture before any alteration occurs.

The following findings are rather less important: (*e*) *A vertical strip of tympany* along the spine, often measuring three centimeters wide, may be detected when there is a large exudate; it is due to compression of the lung. Tympany in the lower left chest results from gaseous distention of the stomach and intestines. (*f*) Tympany is observed over an upper lobe from compression or relaxation of the lung. It was first observed by Avenbrügger and Skoda, and is usually called the *Skodaic note.* (*g*) *"Cracked-pot" resonance* occasionally appears on short forcible percussion over the upper lobe, and results from sudden expulsion of air through the rima glottidis. (*h*) *William's tracheal note* is observed on the left more than on the right side and most clearly in the first and second interspaces. It is obtained chiefly when the entire side is dull and is due to transmission of percussion vibration from the fluid to the trachea, giving a tympanitic note. A change of pitch with opening and closing of the mouth, like that observed in lung cavities by Wintrich, may sometimes occur. (*i*) *Grocco's sign* is *a small triangular dulness on the*

sound side along the vertebræ, due to the interposition of fluid between the sound lung and the spine which thus cannot vibrate; it also occurs in subphrenic abscess, cysts or ascites; Korányi first described this sign in 1897 and Grocco independently in 1902. (*j*) *Dislocation of other organs.* There is no parallelism between the amount of exudate and the amount of luxation, which, of course, is absent if the organs are adherent. The *heart is usually dislocated in toto* but seldom suffers torsion or change of the direction of its axis. The right ventricle may be seen to pulsate to the right of the sternum, even at the right nipple line, when the effusion is left-sided. In right-sided effusions the apex is pushed to the left and downward, for the diaphragm is lower. The *liver* is *less easily displaced* than the heart, but in right-sided effusions is pushed down and the left lobe tipped somewhat upward, though both lobes may be dislocated downward in very extensive pleurisies (Traube and Fräntzel). In left-sided effusions the *spleen is dislocated downward* and forward, undergoes some torsion and may stand perpendicular to the costal arch.

Auscultation.—This is most important in dry and effusive pleurisy. (*a*) The *friction rub* (see Palpation) may be hard or soft, is superficial (*i. e.,* close to the ear) and resembles the sound made by scratching the finger upon the ear or the crunching of snow under the shoe; it often develops in the first twelve hours. It is most frequent during inspiration, but is very often detected in expiration; it seldom occurs during expiration alone. Inflammation of one leaf of the pleura is sufficient for its production. It may disappear after several long breaths which smooth the pleural roughening. The interrupted rub is due to pleural irregularities. It may be increased by pressure and is subject to no law as to its duration. Gerhardt heard a pleural rub which lasted six years. It is heard most frequently over the anterolateral portion of the chest, and is detected more clearly with the stethoscope than with the naked ear, but may sometimes be heard at a distance of several feet, as in a case seen by the writer with Dr. H. M. Richter. If it is apical, it is probably due to tuberculosis. It may rarely be present in miliary tuberculosis (v. Jürgensen) and in tumors of the lungs or pleura without actual pleurisy. It usually becomes less clear when exudation develops, and its reappearance indicates the beginning of absorption or extension of the pleuritis. (*b*) The *breath sounds* are modified not only by the effusion, but also by the condition of the lung, the strength of breathing and the conditions of conduction existing in the pleura and thoracic walls. In general, and especially in men and children, the *respiratory sounds are weakened,* even in fibrinous pleurisy, from repressed breathing and perhaps from pain alone; cog-wheel breathing is common. In moderate effusions three zones are observed: (i) Absence of breathing in the lower parts from compression of the lung by the exudate, which lessens the conduction of the vesicular sounds; (ii) bronchial breathing in the middle zone from moderate collapse of the alveoli of the lung, with fair conduction through the bronchi;

(iii) an upper zone of sharp or exaggerated vesicular breathing. In larger effusions the lower zone extends higher and bronchial breathing may be heard near the clavicle or between the scapula and spine. In the largest effusions the breathing is indistinct everywhere, with areas of metallic bronchial breathing when the lung is wholly collapsed. Amphoric breathing is exceptional. (*c*) The *voice sounds are not heard* over the fluid or over very thick, plastic exudate. They may persist in various places, when islets of the lung are adherent to the chest wall. (*d*) *Bronchial breathing* and increased fremitus may be present, especially in children with empyema, under the following conditions; if the chest wall is not the seat of œdema, if the bronchi are patent, if there is no great fibrin deposit on the pleura and if the lungs are retracted but not absolutely compressed. The bronchial breathing may sometimes be as intense as in pneumonia or tumor. In adults increased fremitus may be observed between the spine and scapula where the lung is compressed. (*e*) *Bronchophony* depends upon the same conditions as bronchial breathing, *viz.,* patent bronchi, moderate retraction of the lung and conditions favoring good conduction of sound. (*f*) *Ægophony,* which is an exaggerated bronchophony, was described by Laennec; it is found chiefly in middle-sized effusions, usually at the level of the fluid. It occurs less often in large cavities and in infiltrations of the lung. In large effusions it is rare, the compression being too great. It is caused by the voice sounds passing through the compressed smaller bronchi to the chest wall; it may be present when the exudate is increasing or decreasing at a given point. (*g*) *Baccelli's symptom* is the "whispering voice," which Baccelli thought was more frequent in serous effusions, but it also occurs in other forms of pleurisy, in chronic induration of the lungs, pneumonia, hydrothorax and cavities. (*h*) A *wide propagation of the heart tones* indicates consolidation rather than effusion; if consolidation is excluded, the hearing of the heart tones more widely than normal suggests dislocation of the heart by an effusion. Murmurs may result from bending of the large vessels by dislocation of the heart, as well as accidental murmurs resulting from a weakened heart, toxæmia or high temperature.

Diagnostic Puncture.—This determines: (*a*) the existence of pleurisy, excluding pneumonia, hypostasis, tumor or atelectasis; (*b*) the character of the fluid; and (*c*) the prognosis. It is indicated in all doubtful cases. Puncture with aspiration is also an important therapeutic measure. *Negative results* from puncture may result from a thick fibrinous exudate, from failure to reach an encapsulated effusion, as in interlobar pleurisy, and from the membrane being pushed in before the needle so that the fluid is not reached. In negative dry taps, after withdrawal of the needle, examination should be made to see that no plug of fibrin or cells has collected in the needle. If a plug is obtained it should be emptied upon a glass slide and examined under the microscope. Care should be taken that no carbolic acid or alcohol remain in the syringe or needle, which, by coagulating

the serum, may cause some dry punctures. The puncture should be made with a large needle. *Positive results:* when the needle is introduced, resistance may be felt, which is due to a callous pleurisy or an indurated lung. Often after introducing the needle, the point may be felt to be freely movable, as in a cavity. Sometimes the results differ according to the level of the puncture; in rare cases serum may be obtained from a high puncture and from a lower one a cellular deposit, the cells settling to the bottom. Again, puncture in different localities may show serum in one and hemorrhage or pus in other sacs, which Galliard calls polymorphous pleurisy. Bacteriological examinations made with 10–20 c.c. of the serum give frequent results. The special chemical, bacteriological, cellular and other characteristics will be discussed under individual forms.

Symptoms.—Symptoms may be absent (latent pleurisy); if present they are not distinctive and there is no fixed relation between the symptoms and physical findings. (*a*) The *temperature* is variable. Its rise is usually gradual and without a rigor. For weeks there may be no temperature, even in purulent exudates, especially in marantic and diabetic subjects and in terminal infections. When present it is more frequently subcontinuous or remittent than continuous. The average elevation is 101° to 102°. Temperature is no positive index as to the character of the fluid. Exacerbations of temperature are important prognostically, especially when the appetite is poor and no change occurs in the physical findings. Recovery is not expected until the temperature has been normal for some time. (*b*) The *pulse* is increased; it is sometimes small and is occasionally of the paradoxical type or unequal in the two radial vessels. (*c*) *Dyspnœa* seldom attends pleurisy, for effusions may attain enormous proportions without respiratory difficulty, particularly when they are gradual in development. Dyspnœa is usually referable to coincident bronchitis, pneumonia, lung congestion, pericarditis, arteriosclerosis, nephritis or bilateral pleuritis. (*d*) *Pain* is usually an initial symptom. It is in no way proportional to the physical findings. It may be absent when auscultation shows a marked rub. Its character is usually dull, tearing, stabbing and sometimes very severe; it may be crampy or colicky. The pain is pleural (Cruveilhier) and is present in the diseased side, well down over the ribs, over the liver, epigastrium or low in the back, and sometimes may radiate to the sound side (Laennec and Gerhardt), into the arm or spine or into the abdomen, especially in children, in which case it may simulate appendicitis. (*e*) *Coughing,* an early and frequent symptom, results from accidental bronchitis or more often from pleural irritation. It may occur after change of posture or after tapping. It is usually unproductive. (*f*) Other symptoms are anorexia, vomiting, headache, difficult swallowing due to involvement of the *foramen œsophageum* (Ferber) and marked sweating; the sweats of pleurisy rank third in importance after those of sepsis and rheumatism. The urine is scanty but increases during resorption, when peptonuria and transient

albuminuria are often found. The *skin* may be pale and cachectic from purulent accumulations, red from high temperature, cyanotic from poor oxygenation or suddenly pallid from hemorrhage into the pleura, when also there is a sudden increase of fluid. *Emaciation* is common in chronic tuberculous or purulent pleurisy.

Special Forms.—1. Character of the Inflammation.—In 790 cases Eichhorst found serous pleurisy in 81 per cent., fibrinous in 12 per cent., purulent in 5 per cent., ichorous and hemorrhagic, each in 1 per cent.

(*a*) *Serous pleurisy* is the prototype of pleuritis. As stated, most cases are tuberculous. The fluid is lemon-colored. Its specific gravity ranges between 1,015 and 1,023, which Méhu considers characteristic of inflammation. Albumin amounts to 3 to 7 per cent.; sugar is frequently present. The fluid clots after puncture. All pleuritic exudates, serous and otherwise, contain white blood cells, red blood cells and endothelial cells; the cells may show fatty degeneration.

Cytodiagnosis.—Much emphasis has been placed on the character of the leukocytes found in tuberculous pleuritis; Widal, Sicard and Revault (1900) especially insisted—and later writers confirm the statement—that *lymphocytes* are characteristic of primary pleural tuberculosis and afford early diagnostic data long before the results of inoculation are available; the fluid must be examined before it coagulates, to which end the fluid is received into a pint of salt solution; in other pleurisies the polymorphonuclears prevail; in other words, the "lymphocytic formula" indicates tuberculosis, the "polymorphonuclear formula" indicates pyogenic infection or pleurisy secondary to well-developed phthisis, and the "endothelial formula" indicates irritation, transudation or malignancy. The tuberculous nature of pleurisy is established by inoculation tests, which are positive in 66 to 94 per cent., by apical involvement or that of other serous membranes, by the finding of tuberculosis in cases of sudden death in the course of pleurisy (Lendouzy, Dujardin, Beaumetz, Dieulafoy, Kelsch, Binet), and by the fact that 25 to 33 per cent. of cases later become obviously tuberculous. It is well to suspect the tuberculous nature of all so-called rheumatic pleurisies (*pleuritis a frigore*). The sputum and lungs should be carefully watched in such cases for some time. For the tuberculin tests see page 319. The leukocytes are not increased in the blood count.

(*b*) *Fibrinous pleurisy* usually shows some fluid, serous or purulent, if systematic punctures are made.

(*c*) *Purulent or suppurative pleurisy* (empyema) usually begins as a serous exudate which soon becomes opaque and purulent. The sterile tuberculous form has been discussed (*v. s.* Bacteriology). In contrast to serous pleuritis two-thirds of this group are secondary. The most frequent types are (i) the streptococcic form, which is observed chiefly in adults, in which the fluid is thin and separates on standing into an upper serous layer and a lower denser layer, in which fibrin formation is slight; and (ii) the pneumococcic form, which is

more common in children, in which the fluid is more homogeneous and viscid, like the "laudable" pus of the early writers, in which the fibrin and membrane formation is abundant and the odor peculiar; it is more often accompanied by endocarditis, pericarditis and meningitis, and is more often febrile and more susceptible of spontaneous recovery than the streptococcic form. In Netter's series of empyema the streptococcus was causal in 47 per cent., the pneumococcus in 39 per cent., the tubercle bacillus in 11 per cent. and the staphylococcus in 3 per cent. Over 80 per cent. of juvenile empyemas are pneumococcic. The fact that the pneumococcus has far less pyogenic propensities in the lung than in the pleura has excited frequent comment. Microscopically, various bacteria are present (*v.* Bacteriology), polymorphonuclear and often fatty white cells, and sometimes cholesterin and Charcot-Leyden crystals. The distention of the chest may be enormous; Krause reported 22 pounds of pus in one case. Greater visceral luxation than in any other type may occur. Leukocytosis and hectic fever are mentioned under symptoms.

Empyema pulsans (Walshe); Sailer collated 95 cases in 1904; 95 per cent. of all pulsating effusions are purulent; 93 per cent. are left-sided. A pulsating tumor as large as the fist may be noticed, which usually decreases during inspiration, or the effusion may simply pulsate behind the ribs; in either case the pulsation may be expansile. The pulsating tumor is usually between the second and fourth ribs, anteriorly, but may be seen lower on the side or even well below in the back. Pulsation is probably propagated from the aorta; it has been attributed, though without satisfactory reason, to necrosis of the pleura, coincident pneumothorax (Féréol), atelectasis of the lung (Comby) or adhesions and paresis of the intercostal muscles (Traube, Keppler). The most apparent conditions for pulsation in an empyema are considerable cardiac energy, massive exudation and paresis of the intercostal muscles. Empyema pulsans may be confused with aneurysm, which latter is much slower in its evolution, is much oftener located above and to the right, and has its own signs, murmur and thrill; it may be confused with pulsating abscess, pneumonia, gangrene or tumor of the lung. The pulsation ceases after aspiration. Aneurysm may co-exist with pleurisy. Expansile pulsation of the chest wall has been observed twice without exudation, aneurysm or any organic change (Lafleur and Edwards).

Empyema may rupture externally, *empyema necessitatis.* Rupture is usually along the sternum or lower ribs. Gravitating abscesses may result, as pulsating tumors in the groin and loin, which may be confused with aneurysm or spinal caries. Rupture into the lung may cause suffocation during sleep; according to Netter, 25 per cent. of post-pneumonic empyemas rupture into the bronchi. Fistulæ, hectic fever and amyloidosis are frequent sequelæ. Hippocrates knew of the "mouth full" expectoration and also the fact, later described by Traube, that pus may penetrate the lung like a sponge without perforation or pneumothorax. Unverricht thinks, however, that perfora-

tion into the lung without pneumothorax is probably caused by a small opening which allows the escape of fluid into the lung but does not allow the escape of air into the pleura, because of the higher tension in the exudate. Hellin (1905) found 114 cases of *bilateral empyema* recorded in the literature; 67 per cent. occurred in males and 90 per cent. in young persons under fifteen years old; the mortality was 30 per cent.

(*d*) *Putrid pleurisy* is usually associated with gangrene of the lung, pneumothorax, œsophageal carcinoma, appendicular abscess and kindred lesions. The fluid is extremely fœtid; fat, leucin, tyrosin, cholesterin and sometimes hæmatoidin crystals are found; the colon bacillus and other gasogenic bacteria are frequently present.

(*e*) *Hemorrhagic pleurisy* is usually serofibrinous and the exudate is tinged with blood. Five to six thousand red cells per c.mm. are necessary to produce a reddish tinge. Its character is seldom suspected before puncture. (i) Most cases are *tuberculous.* Dieulafoy speaks of these cases as having "hæmoptysis into their pleuræ." After puncture more fluid is frequently formed but recovery is possible. (ii) Some cases are *cancerous;* Moutard-Martin has found hemorrhagic pleurisy in but 12 per cent. of pulmonopleural cancer; pain, dyspnœa, luxation and rapid pulse are frequent symptoms; the fluid is dark red, contains little fibrin and is usually formed rapidly; a case of Desnos was punctured 30 times in six months. (iii) Some cases occur in *liver cirrhosis* (of which Laennec published the first case in 1826), in nephritis and in hemorrhagic fevers or exanthemata; profuse hemorrhage into the pleura was called pleural hæmatoma by Wintrich and pachypleuritis hemorrhagica by Chouppe from its resemblance to hemorrhagic pachymeningitis.

(*f*) *Chyliform pleurisy* (*v. i.* Chylothorax).

2. Location of the Exudate.—(*a*) *Pleuritis diaphragmatica* produces notable subjective disturbance. Radiating pain is often experienced, which may resemble angina pectoris (Andral). Pain and tenderness in the epigastrium and between the tenth rib and the xiphoid is pathognomonic, according to Guéneau de Mussy.

Schmidt's respiratory abdominal reflex, an inspiratory spasm of the upper part of the rectus muscle, is indicative of phrenic involvement. Dysphagia from inflammation of the foramen œsophageum may result. If left-sided, vomiting and singultus may be noted, and if right-sided, icterus is not uncommon. Cough may occur spontaneously or on pressure. Pressure in the lower interspaces near the spine elicits tenderness; tenderness over the cervical portion of the phrenic nerve may be noted, together with pain reflected along the acromial branches of the cervical nerves to the shoulder. Dyspnœa is usually great, the temperature is generally normal, the lower chest moves but little, the respiratory murmur is very weak and friction may be heard over the liver or in the "half-moon" space of Traube. If there is no exudate, the diaphragm may remain high. Fluid, accumulating between the diaphragm and lung without reach-

ing the chest wall, causes no dulness. Fibrinous is more common than serous effusion.

(*b*) *Interlobar pleurisy* occurs most frequently after lobar pneumonia or in tuberculosis. Previous pleurisy may favor its localization by obliterating the general pleural space. As Laennec noted, it is almost always suppurative. Its early diagnosis is uncertain, for the fever, rales, cough and dyspnœa suggest a lesion of the lung; the symptoms may resemble those of lung abscess from which differentiation is sometimes impossible; later a zonular surface dulness is found conforming to an interlobar fissure; the dulness is bounded above and below by a normal or somewhat over-resonant note. The heart may be pushed to the right, when there is left-sided localization, but the liver is not luxated in right-sided localization. Interlobar pleuritis may be shown clearly by the *x*-rays. Large effusions may break internally, pus suddenly appearing in the sputum. Hæmoptysis may occur. Puncture is attended with some danger, as the lung may be infected by the needle, during its withdrawal. Spontaneous recovery is infrequent. The outlines of the lobes are shown in Figs. 33, 34 and 35.

(*c*) *Mediastinal pleurisy,* described by Laennec and Andral, is most often tuberculous or pneumococcic, and more frequently purulent than serous. The symptoms of lung compression are less distinctive than are those of compression of the mediastinal tissues. There may be wheezing, dyspnœa, dysphagia, intrathoracic oppression or pain, a pertussis-like cough, enlarged thoracic veins from azygos compression, deviation of the larynx or trachea, and dulness between the scapula and spine; the *x*-ray picture may be suggestive. The sudden onset and febrile course differentiate it from aneurysm, tumor and adenopathies. The outlook is unfavorable without operation.

(*d*) *Peri-* (*para-*) *pleuritis* (Wunderlich, Bartels, Billroth) is an inflammation outside of the parietal pleura, and is usually purulent. Some cases follow glanders, but most of them are due to *actinomycosis.* Fistulæ and the evacuation of the characteristic sulphur granules are common. The symptoms are more local and irregular than those of pleurisy; fluctuation is more frequent; heart dislocation and lung compression are rare; the respiratory excursion is free, and on heavy percussion lung resonance may be elicited beneath the dulness. Metastatic inflammation, perforation and burrowing are common.

3. Pleurisy in Children.—In children pleurisy is attended by more general symptoms, higher temperature, higher pulse and more rapid respiration. Bronchial breathing is more frequent, there is less dulness, less luxation and less friction. It often follows pneumonia, where, in the majority of cases, the empyema is more benign. Tuberculosis is less frequently causative in children than in adults. According to Lemoine, metapneumonic pleurisy is distinguished from the parapneumonic type, by its slower course.

Course.—The course of uncomplicated pleurisy averages three to six weeks; acute cases last two, subacute three to eight and chronic

eight or more weeks. No general rule can be laid down, and the onset, whether mild or stormy, is no index of the issue. No cycle is seen in any form of pleurisy. Pleuritis acutissima, with high fever, typhoid symptoms and early death is fortunately rare. Friction along the lowest limits of the pleura and in front rarely attends great effusions

FIG. 33.

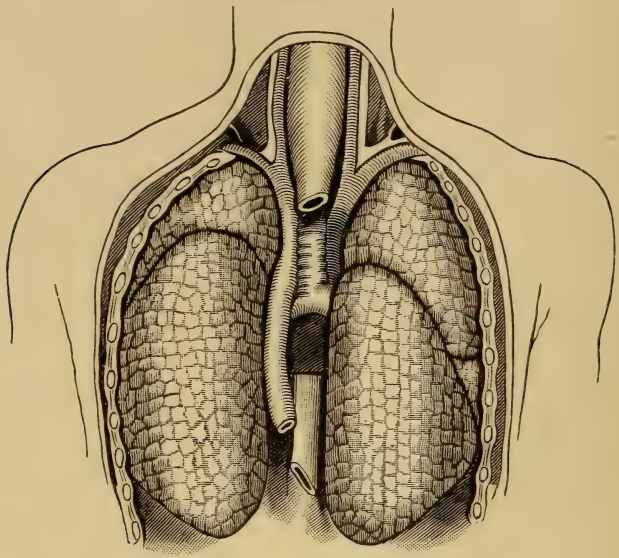
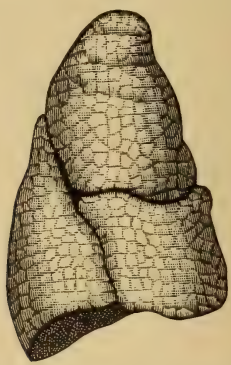


FIG. 34.

FIG. 35.



Cuts from Dieulafoy, showing: Fig. 33, the posterior surface of the lungs and their interlobar fissures; Fig. 34, the lateral aspect of the left lung; and Fig. 35, that of the right lung.

and therefore, after the exclusion of tuberculosis, carcinoma or pyæmia, the prognosis is relatively good. In the average case dry pleurisy precedes and succeeds exudation. Recurrence and exacerbations render the prognosis unfavorable. Partial absorption with persistence of temperature or with dyspnœa is indicative of tuberculous pleurisy. Rheumatic pleurisies heal more readily than tuberculous. The author has seen recovery of secondary carcinomatous pleurisy.

Issue.—1. Resorption.—Resorption may occur; even purulent pneumococcic pleurisy may, in exceptional cases, be absorbed in part without operation or rupture, leaving inspissated caseous accumulations. Serous pleurisies may be absorbed after three to six months, during which time persistent friction is common over the lower chest.

2. Adhesions.—The thickness of pleural adhesions is more important than their extent. Universal obliteration of the pleural sac by thin adhesions is of no consequence. Compensatory emphysema, pain, thoracic oppression, obliteration of the complementary pleural space, dulness, decreased vocal fremitus and breathing, failure of Litten's phenomenon, decreased respiratory excursion and stagnation of bronchial secretion may occur when the adhesions are thick. If there is exudate around the heart, the apex beat may increase during the inspiration; this also occurs in bronchial catarrh (Eichhorst). Failure of the right heart and stasis may ensue, though most extensive adhesions sometimes exist without cardiac embarrassment. Peritonitis, mediastinitis and obliterative pericarditis may complicate the case. In some instances the pleura calcifies or ossifies, leaving a deposit measuring three cm. or more in thickness.

3. Sudden Death.—Fatal syncope may result after exertion, coughing, urination or defecation; it results from paralysis of the left ventricle, cardiac thrombosis, pulmonary œdema, bending of the aorta, compression of the cava and rarely from pulmonary embolism caused by venous thrombosis, rupture of fluid into the lung or bronchi, which may drown the patient during sleep, and death from hemorrhage into the effusion, pneumothorax or brain embolism.

4. Retractio Thoracis.—Retraction is characterized by increased resistance, decreased fremitus, decreased circumference of the affected side and often by bronchiectasis. The percussion note is dull and the breathing is usually decreased, but sometimes is bronchial. The causative factors are atmospheric pressure, retraction of the pleural scars, extension of the inflammation into the lungs (pleurogenous interstitial pneumonia), which is also known as pleuritis deformans, and which sometimes divides the lung up into lobes or islets. Retraction is generally a slow process but the writer has seen marked deformity develop in three weeks.

5. Changes in Other Organs.—When retraction occurs, other organs may be dislocated. The liver may be higher when the right side is involved, and in left-sided retraction the lung may shrink away from the heart, the spleen and the heart may lie higher and Traube's space may be increased. Organs displaced by exudate may in rare cases remain luxated after the exudate is absorbed. Pleural callosities may produce recurrent laryngeal paralysis which may also follow the pressure of a fluid exudate or enlargement of the glands. Other complications are acute and chronic infiltration in the lung; inflammation and degeneration in the chest muscles; extension to other serous surfaces; septicopyæmia; stasis in the liver, stomach, extremities, etc.; hemorrhages into the skin; chronic nephritis;

amyloid degeneration and pulmonary, glandular, genito-urinary or miliary tuberculosis. Scagliari reports forty-seven cases of paralysis of the arm, probably due to brachial neuritis.

Diagnosis.—The physical findings only are final; symptoms are most unreliable. Diagnosis of the malady is the first step, and the second is the determination of its cause.

Differentiation.—1. The diagnosis of *pleuritis sicca* is established only by feeling or hearing the friction rub. (*a*) The *crepitant rale* is largely inspiratory, and is heard as a number of small crackles at the end of inspiration. Bronchitic rales or rhonchi are continuous. Coughing and deep inspiration alter or remove rales but the pleural rub is increased by pressure and deep inspiration and is less extensive. It may disappear after a number of deep inspirations, because this makes the two surfaces temporarily smooth. (*b*) It must be differentiated from *sacculated breathing* and from the physiological, bilateral cracking in the chest muscles on deep breathing. (*c*) *Intercostal neuralgia* is intermittent, occurs in typical attacks and is limited to the interspaces; Valleix's points are usually present at the angle of the ribs, in the middle of the axilla and anteriorly, and correspond to the points of exit of the posterior, lateral and anterior perforating branches of the intercostal nerves. Intercostal neuralgia is increased less on deep breathing than is pleuritic pain. (*d*) In *caries of the ribs* pain is localized directly over one rib and not between the ribs. (*e*) Differentiation from *rheumatism* of the thoracic muscles may be very difficult, for both affections may induce pain on breathing, tenderness and disturbed breathing; there may be a history of cold or exposure or of repeated attacks. There is usually no fever or cough. Faradization decreases the pain and movement increases it more than is the case in pleurisy. (*f*) The rub in *pericarditis* (*q. v.*) is more distinctly cardiac and depends upon the cardiac movements. In pleurisy along the border of the heart, on holding the breath the pleuritic rub is heard near or over the heart, and decreases after five or six heart beats. On very deep inspiration the pleural rub ceases and the pericardial rub is increased. Differentiation is most difficult when there is pleurisy over the lingual lobe and left border of the heart, though pericarditis most commonly produces a rub at the base of the heart, especially over the conus pulmonalis arteriosus.

2. *Pleuritis humida* (pleuritis effusiva) must be distinguished (*a*) from *infiltration* of the lung substance (see table in Diagnosis of Pneumonia). (*b*) *Tumors of the lung* produce cachexia, narrowing of the chest behind on one side and the characteristic exudate; this is often brown (Fränkel) and sometimes chyliform, and contains fat globules (Quincke) and degenerated large cells which may show mitoses; the glands above the clavicle may be enlarged, as well as those in the mediastinum, which produce recurrent laryngeal paralysis. Tumor particles are rarely obtained by puncture or in the sputum, and are only found in carcinoma, for sarcoma rarely ulcerates.

When tumors of the lung are suspected, a trocar rather than a needle should be used. There is more resistance to puncture in tumor than in pleuritis. Unverricht observed the development of carcinoma at the seat of puncture. In tumors there is less displacement, less invasion of Traube's space and less widening of the chest than in pleurisy, and the breathing is often bronchial, the flatness is intense and the fremitus is increased. (*c*) *Hydrothorax* (*q. v.*), usually described as bilateral, may be unilateral, especially in disease of the liver, kidneys and heart, where right-sided hydrothorax is observed. Unilateral hydrothorax is sometimes explained by obliteration of the other pleural cavity by adhesions. The fluid readily shifts its level on change of posture, which does not occur in pleurisy. The claim that salicylic acid, iodine and potassium iodide pass readily into transudates and but slowly and in small amounts into exudates has been disproven by Weintraub, Leuch and Feldmann. In hydrothorax, the specific gravity is below 1,014, the albumin is 1 to 3 per cent. and the sediment shows few white cells and some endothelial cells. Bacteriological tests are negative. (*d*) The presence of *hæmothorax* is determined only by puncture.

3. *Circumscribed pleurisy* requires differentiation (*a*) from *pericarditis;* pleurisy shows a more irregular outline, the apex beat is not altered and there is no cardiac insufficiency or pericarditic friction. (*b*) *Lung cavities* show dulness when full of secretion and resonance when the secretion is voided; the fluid obtained by puncture (which is made under a wrong diagnosis) has a higher specific gravity and a larger percentage of fat than that of pleurisy. (*c*) Differentiation from *splenic tumors* is determined by respiratory excursion and palpation of the lower edge of the spleen. (*d*) *Liver enlargements* rarely occur solely upward, save in echinococcus and in abscess of the convexity; these are prone to occur anteriorly, and pleurisy is more frequently detected posteriorly. In tumors of the liver there is usually respiratory excursion which is absent in pleurisy. (*e*) *Subphrenic abscess* (*v.* Localized Peritonitis) may be intraperitoneal (as from liver abscess) or extraperitoneal (*e. g.,* perinephritic). The liver stands higher in the chest because of the paretic diaphragm. There may be a history or physical signs of previous disease of an abdominal organ. The needle, when thrust toward the abscess, reaches the pus only when thrust deep in the lower interspaces. The pus has frequently a fæcal odor. The manometer shows increased pressure during inspiration and decreased pressure during expiration, the converse of the findings in pleural effusion. (*f*) *Abscess of the liver.* Pleurisy, serous or suppurative, may complicate abscess of the liver. Differentiation is easy when abscess of the convexity is located forward; when it is situated posteriorly, aspiration alone will distinguish it; the pus shows liver cells, amœbæ, cholesterin or bile. (*g*) 43 cases of *pleural echinococcus* are reported; 11 per cent. of hepatic cysts reach the lung and pleura.

Prognosis.—The *immediate mortality* is about 5 per cent., though

an early prognosis cannot be made. (*a*) The *etiology* is an important factor. (*b*) The *nature of the fluid* is of prognostic import. Fibrinous pleurisy (except when it occurs at the apex of the lung) is the most favorable type. Prince Ludwig Ferdinand considers the prognosis in the effusive forms as follows: (i) most favorable in metapneumonic serous or purulent pleurisy; (ii) next most favorable in serous pleurisy due to the staphylococcus, (iii) less favorable in staphylococcus empyema, (iv) least favorable in tuberculous, fœtid or putrid forms. The prognosis is usually poor in hemorrhagic, chyliform, peracute and bilateral (generally hemorrhagic or purulent) effusions. It is more favorable in effusions which are relatively rich in solid constituents (Méhu and Bernheim); effusions containing little solids are apt to recur. When the freezing point of the exudate is found by cryoscopy to be below that of the blood, spontaneous absorption is unlikely to occur (Rothschild). Since the time of Hippocrates, the belief has prevailed that right-sided are less favorable than left-sided effusions. (*c*) The persistence of temperature and sweats, anæmia, recurrences and external rupture are unfavorable. (*d*) *Sudden death* is more frequent than it is usually believed to be. The author saw three cases in one year.

Opinions vary as to the *ultimate prognosis.* Eighty-seven per cent. of cases become tuberculous in one to two years (Fiedler); 33 per cent die in five years (Bars); 6 per cent. are curable (Osler); 15 per cent. die of tuberculosis, 90 per cent. are in good health after two to five years and 80 per cent. are healthy after five years (Cabot).

Treatment.—*Etiological* treatment is applicable only to the rheumatic and syphilitic forms.

Treatment of *fibrinous pleurisy,* which remains fibrinous, or of the fibrinous or first stage of exudative pleurisy. *Pain* is the main indication; morphine may be given in severe cases, especially when coughing is severe; the ice-bag, poultices, blisters, cups and hot fomentations give little relief when the pain is acute; blisters may eventually cause as much pain and discomfort as the disease itself; the pleura should be splinted by three or four *strips of adhesive plaster* two inches wide, overlapping the median line before and behind by about three inches, and running around the chest horizontally and obliquely; these should be applied with considerable pressure during expiration, that the lung in its lower part and the pleural surfaces may be well immobilized; zinc oxide adhesive plaster is least irritating to the skin. Personal experience with the early use of salicylates, has not been satisfactory. The patient should be kept in bed.

In *serous pleurisy,* the patient should be kept in bed, and the cough and fever treated expectantly. *Laxatives* are given solely to obviate straining at stool, which may induce cardiac weakness; they do not reduce the effusion. If at the end of the second week the effusion shows no sign of being absorbed, various measures are indicated; Schroth's treatment consists in limiting the amount of fluid ingested to about one quart (1000 c.c.) daily, but the method gives no results

as exudation is an active, not a passive process; tincture of iodine is seldom beneficial, and when old is distinctly a local irritant; potassium iodide, digitalis, drastics to produce copious evacuations, pilocarpine (combined with cognac to support the heart), cotton jackets and sweats are often given. Of late a salt-free diet (dechlorination) is advised, as in nephritis (*q. v.*). The author cannot agree that any of these measures are helpful and believes pilocarpine is always dangerous. Transudates may be removed in this way but not exudates.

Aspiration.—Trousseau's indications were (*a*) the vital indication (when life is threatened, because of large exudates); (*b*) moderate effusions with slow resorption, and (*c*) persistent or residual exudates. The author believes that *early puncture* (thoracocentesis) and aspiration are indicated in every case, where two quarts or more (2,000 or 2,500 c.c.) of fluid are effused, whether the temperature is still high or not, and whether pressure symptoms or dyspnœa are present or not. This amount of effusion causes dislocation of the heart, liver or spleen, compression of the lung or ectasia of the chest; high intrathoracic pressure may cause sudden death, sometimes without any warning symptoms of cardiac insufficiency. Early puncture does not irritate the pleura or favor recurrence or suppuration. Early and *repeated punctures* obviate compression of the lung and atelectasis, as well as compression and torsion of the large arteries. Aspiration was first performed by Hippocrates and exploited by Schub and Skoda (1841) and Trousseau (1844).

Method of Aspiration.—After the usual surgical antisepsis, a small trocar or large aspirating needle is introduced, which has an elbow connecting with a rubber tube, to siphon the fluid to a vessel below, or connecting with a bottle which can be exhausted by a pump. There is no absolute rule as to the site of puncture. This must be governed solely by the physical signs; for instance, adhesions which hold the lung to the surface vitiate any set law; fluid is usually obtained between the scapular and post-axillary lines and low down in the chest. The trocar should be introduced perpendicularly to the chest wall and not obliquely, in order to avoid injuring the intercostal artery lying above the rib, as a result of which the author twice saw fatal hæmatoma and hæmothorax. Whiskey and hypodermics should be at hand, because of the possibility of syncope. The use of the pump is necessary in but 10 per cent. of cases, for the fluid can usually be siphoned readily; a tube is attached to the needle or trocar, filled with boric acid solution, clamped with an artery forceps and unclamped, when it is placed in a basin containing the same solution and at a lower level than the seat of puncture. The fluid, if pumped, should be withdrawn gradually; caution is necessary lest air be pumped the wrong way, *i. e.,* into the pleura. The amount removed need not be great, as moderate relief of intrapleural tension often promotes absorption. The blood pressure during aspiration should be controlled by the sphygmomanometer. The amount to be withdrawn is estimated arbitrarily at one quart (1,000 c.c.,

Naunyn and Dieulafoy) or three pints (1,500 or 1,600 c.c., Fränkel and Bowditch). Complete or nearly complete evacuation may cause the accidents enumerated below and is thought by Litten to induce miliary tuberculosis. Aspiration should be *discontinued* when the exudate becomes bloody (from lung injury), when friction develops, when pain is felt or when coughing (from congestion of the relaxed lung) begins. *Repetition of puncture;* a second puncture is necessary in 25 per cent. and a third in 5 per cent. of effusions. *Favorable results* are slowing and strengthening of the pulse, relief of oppression, increased diuresis, return of luxated organs to their place and improved appetite; but relief of pressure on the lung and absorption of the exudate are of the greatest importance. *Unfavorable results* are (*a*) syncope due to cerebral anæmia which results from reflux of blood to the expanding lung which contained little blood before it was relieved by the puncture, a relatively uncommon occurrence; (*b*) pulmonary œdema, the albuminous expectoration described by Terillon, Besnier, Johnson, Duffin and Scriba, which is due to the sudden withdrawal of too much fluid (two to five liters) at one tapping; it should take a half-hour to evacuate one and a half quarts or 1,500 c.c. (Fräntzel); œdema pulmonum is treated most successfully by hypodermics of morphine, gr. $\frac{1}{4}$ and some clinicians advise giving it before aspiration; Sears records 10 deaths from simple puncture; (*c*) sudden death from pulmonary embolism or cerebral embolism; the latter results from discharge of clots from vessels in the relaxing lung, and causes the so-called pleuritic hemiplegia; some reported cases are hysterical; (*d*) injury to the heart, liver or spleen; (*e*) pneumothorax and subcutaneous emphysema may occur though the author has never seen them. (*f*) The author twice witnessed an extensive hæmothorax and subcutaneous hæmatoma in a pleural puncture, in which the trocar was impatiently thrust in and out during an unsuccessful thoracocentesis. Naunyn writes of death following puncture of an atheromatous intercostal artery. (*g*) Fibrinous bronchitis has followed paracentesis. When repeated punctures fail to produce the desired results, the advisability of operation (as in empyema) should be considered.

Empyema in all cases necessitates operation (practiced by Euryphon and after him by Hippocrates). Some cases may recover spontaneously, but this cannot be depended upon. A day or two before operation, aspiration is indicated when the fluid is under high tension, thereby relieving the heart. Some surgeons advocate treating tuberculous empyema without operation, as Volkmann treated other cold abscesses; Krönlein observed, after operation on these cases, recovery in 36 per cent., improvement in 28 per cent. and death in 36 per cent. Results depend on the etiology and bacteriology. The mortality in Velpeau's cases was 100 per cent. and that of Dupuytren was 92 per cent.; Dupuytren himself "chose to die of empyema at the hands of God rather than to die at the hands of man under operation." In Schede's series the mortality of tuberculous empyema

was 77 per cent., of metastatic 32 per cent., of metapneumonic 13 per cent. and of primary, 8 per cent.

Hemorrhagic effusions may necessitate injections of gelatin subcutaneously; the amount aspirated should not exceed one pint (500 c.c.) and aspiration should be repeated as seldom as possible.

After-treatment is directed toward prevention of thoracic retraction, to which end gymnastics and deep breathing are essential. A patient having suffered from a "primary" effusive pleurisy should be treated as one with latent tuberculosis.

PNEUMOTHORAX.

Definition.—Pneumothorax is an accumulation of gas or air in the pleural sac. It was named by Itard of Paris (1803) and was described fully by Laennec (who in 1819 made the first diagnosis) and Skoda. Hippocrates described succussion. As it excites pleurisy in 90 per cent. of the cases, the following varieties are distinguished: *pyo-, sero-* and *hæmopneumothorax.*

Etiology.—1. *Diseases of the lung and pleura* cause about 95 per cent. of all cases. In Biach's collection of 918 cases in Vienna, pulmonary *tuberculosis* was causal in 77 per cent., gangrene in 7 per cent., empyema 5 per cent., trauma 3 per cent., bronchiectasis and abscess each 1 per cent., the etiology was unknown in 2 per cent. and the remaining 4 per cent. were due to emphysema, infarct, thoracocentesis, echinococcus and contiguous disease perforating into the pleura, as gastric or œsophageal ulceration or caries of the sternum. By other writers (*a*) tuberculosis is thought to cause 80 or over 90 per cent.; acute forms are more important than chronic tuberculosis in which there are protecting pleural adhesions; pneumothorax develops in two (Drasche) to six (Eichhorst) per cent. of tuberculous cases. (*b*) Ulcerative lesions of the lung (*v. s.*) may erode the pleura and admit air. (*c*) Trauma, rupturing of the lung directly or by *contra-coup,* thoracocentesis or stab wounds, occur far less frequently than one would expect. (*d*) Empyema may rupture into the lung without allowing ingress of air from the opposite direction. (*e*) Rupture of a healthy lung by tearing away of the lung as pleural adhesions give way in coughing, muscular straining or violent coughing in pertussis is very exceptional.

2. *Perforative lesions in the mediastinum,* œsophagus, peritoneum, stomach or colon and subphrenic abscess are uncommon causes (*v. s.*).

3. Pneumothorax may occur *spontaneously.* The view held by Oppolzer, Biermer and others that putrefaction may occasionally develop primarily (without rupture) has received confirmation by discovery of the *Bacillus aërogenes capsulatus* and other gasogenic organisms (Lewy, Welch and Fränkel). It is always possible, however, that a point of rupture in the lung is overlooked or has healed over, and the fact remains that air enters the pleura chiefly from the lung or from a gas-containing organ.

Pneumothorax occurs largely in *adults* and four to seven times as frequently in *males* as in females. Cozzolino (1906) found only 41 cases in children; of these but 40 per cent. were tuberculous. Eichhorst and Drasche find it more often on the right side.

Pathology.—When the chest is opened, air or gas escapes with a force sufficient to blow out a candle. The volume of air may exceed 2,000 c.c., and its tension, studied first by Wintrich and Weil, compresses and collapses the lung, dislocates the heart and mediastinum and depresses the diaphragm and subphrenic organs. The gas may be odorless or fœtid. The pathological and clinical findings vary (*a*) as the gas is encapsulated or occupies the entire pleural cavity; (*b*) as the air is aseptic (producing a pure pneumothorax, or one with slight exudation) or is infected by the organisms of the primal disease which excite serous, purulent or putrid pleurisy; (*c*) as the *air-containing* cavity is "closed," "open" (into a bronchus, or sometimes also open externally) or possesses a valve, thus allowing passage of air in one but not in the opposite direction. These points will be considered below.

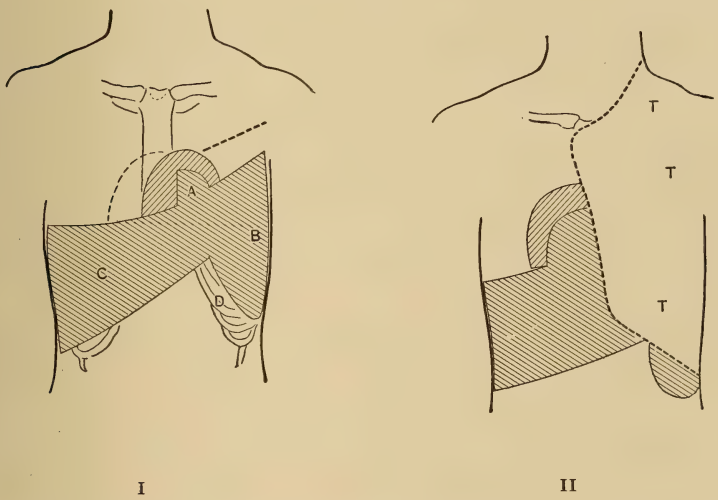
Symptoms.—The *onset* is usually *sudden,* occurring spontaneously or following coughing or muscular exertion; it is characterized by *sudden pain in the side, cyanosis, dyspnœa* and *collapse,* due to reflex action on the vagus; there may be sudden evacuation of pus from the breaking into the lung of an empyema; an urticaria occasionally develops (Gerhardt, Senator); decreased diuresis, albuminuria, stasis and subcutaneous emphysema are sometimes observed. The onset may resemble angina pectoris, pulmonary embolism or abdominal perforation. The onset may be insidious for pneumothorax sometimes constitutes an unexpected post mortem finding. In other instances the previous history or the course may justify a presumptive diagnosis, as in a case clearly tuberculous; and in still other cases, a latent lung lesion (a small caseous focus) may burst and thus produce its first symptom.

Physical Findings.—1. Inspection.—(*a*) The affected *side is distended* 2 to 4 inches (5 to 10 cm.) and *immobile,* and contrasts sharply with the widely-moving sound lung. (*b*) The *interspaces* are *obliterated.* (*c*) The *heart* and *liver* or spleen are *dislocated.* (*d*) There is *dyspnœa* and *cyanosis.* (*e*) The *attitude* is characteristic; the patient most often lies *on the affected side* to afford ample play to the sound lung; sometimes there is orthopnœa and if the point of rupture opens into the lung or bronchus, a peculiar decubitus may be assumed, in order to drain the pleura. (*f*) The *x*-rays show the *lung flattened* near the spine, the *diaphragm depressed* and *motionless,* and the shadow of the effusion.

2. Palpation.—(*a*) *Vocal fremitus is absent,* unless adhesions hold the lung to the chest wall. (*b*) The *chest wall feels resistant* and (*c*) sometimes *splashing,* fluctuation or a "pillow sensation" is noted. (*d*) The *dislocated apex beat* and the edge of the liver or spleen are felt.

3. Percussion.—Somewhat different results are obtained according to the tension of the gas and the chest walls. (*a*) In *open pneumothorax* the note is full, tympanitic and even metallic when percussion is made on a solid object, as a coin; this metallic reverberation, called *bruit d'arain* by Trousseau, is found in 75 per cent. of cases; cracked-pot resonance and Wintrich's change of note may be elicited when the mouth is opened. (*b*) In *closed pneumothorax* the note is full and loud; it is tympanitic when there is moderate pressure and the intercostal muscles are relaxed; again the note may be dull (Skoda) if the tension is great or if the chest muscles are spasmodically contracted, as in sudden pneumothorax (vagus reflex action). (*c*) *Flatness* over the lower thorax usually results from pleural effusion. This dulness may occur without coincident pleurisy, and conversely, the author has seen tympany prevail over the entire side, even where there was great pleural effusion. Skoda said that the fluid is twice as much as we anticipate. (*d*) On change of posture, a *changing level of the fluid* is readily elicited, the gas being found above and the fluid below, for the gas acts in this respect as

Fig. 36.



I II

I. Left-sided effusive pleurisy, showing relative heart dulness (A), effusion (B), liver flatness (C), and fluid impinging on Traube's half-moon space, of which but a small part remains resonant (D).

In larger effusions reaching, *e. g.*, up to the dotted black line, the relative heart dulness may be pushed over to the dotted line (over C).

II. Left-sided pneumothorax marked by tympany (T. T.), pushing over the mediastinum and also the heart to the right, and pushing down the left lobe of the liver and the spleen.

a spirit-level; this change is far more important than in simple pleurisy, as there are no adhesions. (*e*) The *pitch* over the gas-distended part *changes with change of posture* (Biermer's change of note). It becomes deeper as the patient sits up, because the vertical dimension of the chest is thus increased by the pushing down of the dia-

phragm. (*f*) The *heart dulness* is *replaced by resonance* in left-sided pneumothorax, and is found to the right of the sternum; in right-sided pneumothorax the luxation is less and towards the left axilla. (*g*) The *liver* or *spleen dulness* is *pushed* well *downward*.

4. Auscultation.—(*a*) The *breath sounds* are usually *absent* in the closed variety of pneumothorax, and this, with ectasia and hyperresonance, is most characteristic. In the open and sometimes in the closed form, the breath sounds are *bronchial*, amphoric or metallic, the voice sounds, especially under the clavicle, are metallic (metallic bronchophony), and coincident rales in the bronchi are also metallic. Amphoric sounds were referred by Laennec to a fistula but probably are due to the air acting as a sounding board. (*b*) Air passing through the point of rupture may produce curious *snapping or whistling sounds;* the point of rupture is oftenest between the second and fourth interspaces and between the mammary and axillary lines. (*c*) The *gutta cadens* (the metallic "falling drop") may be due to rales or dripping of fluid from fibrin shreds as the patient's posture is changed. (*d*) The Hippocratic splashing (*succussio Hippocratis*) was first carefully described by Laennec and is elicited by placing the ear to the chest and sharply shaking the patient; it is sometimes heard at a distance from the patient and in one of James' cases was heard over a large clinical amphitheatre; in three cases patients have spontaneously called the author's attention to a "splashing in the chest"; it is oftenest heard in the side or in the interscapular area and in three-quarters of the cases; succussion usually declares the fluid before dulness develops. (*e*) The *heart tones* may be *metallic*, and the heart's action may even cause splashing sounds. Hellin collected 54 cases of double pneumothorax; it is not incompatible with life.

Diagnosis.—The ectasia, the resonant note, suppressed or amphoric breathing, cardiac and other luxations and succussion are most characteristic.

Type.—Contrary to the usual statement, Weil proved that dislocation occurs in all types, though it varies in degree. (*a*) In the *open form* Wintrich's change of note on opening the mouth, metallic sounds, the fistula murmur, periodic expectoration of large amounts of pus, moderate cardiac and other dislocations and no manometric increase of the tension in the pleura are distinctive. Methyl blue injected into the pleura is coughed up. (*b*) The *closed type* is distinguished by increased manometric tension, absence of breath sounds, marked ectasia, change of note on sitting but not on opening the mouth and marked luxations. (*c*) In the *valvular form* air may enter the pleural cavity but cannot escape back and therefore pus cannot be voided. Some of the auscultatory findings of the open type are noted. The manometer shows increased pressure. The fistula may become permanently closed by organic change (organic type), so that aspiration of the air is not followed by further accumulation; or the fistula may be only mechanically closed, and air may reaccumulate after its aspiration.

DIFFERENTATION.—(*a*) *Cavities* may be simulated by circumscribed pneumothorax, but in cavities the interspaces are sunken, the fremitus is increased when they are empty, luxation is absent and the Hippocratic succussion and coin sound are extremely rare. (*b*) *Pleurisy* can be distinguished on careful examination. (*c*) *Meteorism* is at once excluded by filling the stomach or colon with water. (*d*) *Diaphragmatic hernia* is very frequently confused with pneumothorax; out of 433 cases collected by Grosser only 12 were recognized. In Dehio's recent case the *x*-rays determined the condition. Respiratory excursion is present and the tympany and metallic sounds are usually accompanied by such symptoms of marked incarceration as vomiting, colic, obstipation and indicanuria. The intestines push the heart to the right. Ninty per cent. of cases of diaphragmatic hernia are left-sided. (*e*) *Pyopneumothorax subphrenicus* (*q. v.* under PERITONITIS, LOCALIZED FORMS), described especially by Leyden, results from abdominal lesions, such as cancer, ulcer, appendicitis, disease of the gall-tracts, trauma and foreign bodies whose histories are at least suggestive. Thoracic symptoms, as cough, sputum and dyspnœa, are not conspicuous, at least early in the process and the lungs may show perfect excursion, though the diaphragm is sometimes paretic. The manometer shows that the pressure is increased during inspiration and decreased during expiration, the converse of which is true in pneumothorax. The *x*-rays show that the diaphragm is above the fluid, while in pneumothorax it is below the fluid.

Prognosis.—The prognosis varies with the cause, the condition of the lungs and the character of the fluid, but it is always serious. Cases in a healthy lung following exertion, trauma or infarct are relatively favorable. The closed and open are more auspicious than the valvular type. In tuberculosis, pneumothorax seems to arrest the tuberculous focus if it is small (Hérard, Toussant, Fräntzel) and if no mixed infection occurs, but even in these cases complete ultimate recovery is infrequent. Eichhorst observed one case which lasted for five years. Gabb records a case which recovered and relapsed four times, the attacks being two to four years apart. According to Weil 25 per cent. die within a week and 50 per cent. within a month; West observed a mortality of 70 per cent., of which three-quarters died within two weeks, nine-tenths in less than a month and one case in twenty minutes.

Treatment.—The results of treatment are disappointing. (*a*) Narcotics and stimulants are imperative at the time of the rupture, for collapse, dyspnœa and cyanosis. (*b*) Aspiration is of most value in the serous forms; early aspiration may open a healing fistule. (*c*) The radical operation is indicated in putrid or purulent pneumothorax. Though some prefer not to interfere in the tuberculous cases on the ground that the compression exerted upon the lung by the pneumothorax is semicurative, others advise operation; Leyden reported 66 per cent. and Richardière 50 per cent. of recoveries.

HYDROTHORAX.

Definition.—Hydrothorax is a serous transudation into the pleura.

Etiology.—(*a*) *Stasis,* from cardiac, pulmonary or vascular factors is a frequent cause. In failure of the right heart the superior cava is imperfectly drained, which congests the vena azygos and hemiazygos and this leads in turn to pleural transudation. It may occur alone or precede dropsy but is oftener a part of general dropsy. Local venous obstruction from mediastinal tumor or aneurysm may induce hydrothorax. (*b*) *Blood changes* resulting from multitudinous anæmic, marantic or cachectic states, as nephritis, cancer and blood diseases. Under these captions may be brought diseases which alter the *walls of the vessels.* The chief factors are cardiac and renal.

Symptoms and Diagnosis.—1. The *symptoms* of hydrothorax do not include pain or fever. The condition accentuates, sometimes considerably, the dyspnœa attending the causal stasis or cachexia. The amount of fluid varies from ounces to quarts (or even to two gallons).

2. The *physical signs* are essentially those of moderate effusive pleurisy, except that the rub is absent; the fluid shifts with much greater facility on change of posture and produces a moderate compression of the lungs and luxation of the heart, liver and spleen. The transudate is usually described as bilateral (in 83 per cent., according to J. Dutton Steele), but in over half the cases it is unilateral and often right-sided, in cardiac, renal and hepatic disease, or less often, when previous adhesions obliterate the opposite pleura. Stengel explains right-sided hydrothorax by pressure on the azygos by the dilated right heart or cava or the luxated root of the lung.

3. The *fluid* is clear, slightly green, possibly moderately blood-tinged if it develops during the death agony, and shows a specific gravity of about 1,010 to 1,015; it contains but little fibrin and at the most develops only light delicate flocculi; the albumin ranges between 1 and 3 per cent. Centrifuging brings down only a few leukocytes, endothelial cells and red disks, but no bacteria. Right hydrothorax may prove very obstinate, probably because a low grade of inflammation eventually supervenes.

The prognosis and treatment are those of the cause; aspiration often prolongs life.

HÆMOTHORAX.

Blood effusion into the pleura is a rare malady. It may be caused by trauma, scurvy or allied blood diseases, by aneurysmal rupture, by ulceration of the pulmonary or pleural veins and by various necrosing lung diseases. It may occur with pleurisy and pneumothorax and has resulted from thoracocentesis. Its symptoms are essentially those of hydrothorax; it is suspected only by a sudden onset with signs of acute anæmia, and is proven only by the exploring needle. Its treatment is symptomatic; for the occasional syncope, the usual stimulants should be given; ice should be applied to the chest or a

2 per cent. gelatin solution injected for hæmostasis; operation is invoked when indicated by the causal lesion.

CHYLOTHORAX.

A very rare affection, in which true chyle or chyliform fluid is found in the pleura. Its etiology is much disputed; (*a*) in the genuine chylous cases true chyle containing sugar is found, though of late years sugar has been found in very many forms of serositis and in most transudates; only 60 cases of this type are recorded. In 11 cases (of which 4 died and of which 9 were right-sided), traumatic rupture of the thoracic duct has been found. In others the duct was obstructed by thrombosis of the subclavian vein or by glands and tumors. (*b*) In a second group, the pseudochylous (chyliform or adipose), there is no chyle and the milky appearance is due to fatty or minute albuminous granules held in suspension. This variety is chiefly tuberculous or carcinomatous. It has been thought that old empyemas may become chyliform. The characters of both forms of fluids will be considered under chylous and adipose ascites, with which they are frequently associated. The signs are those of transudation or exudation.

PLEURAL TUMORS.

Pleural tumors are nearly always secondary to pulmonary, mediastinal or extrathoracic growths. Carcinoma and sarcoma are the most frequent forms; enchondroma, lipoma, hypernephroma and dermoids are very uncommon. The pleura is involved by contiguity or metastasis. Wagner, Fränkel and Riedel described a primary endothelioma of the pleura, of which 50 cases are reported, but, as stated under the etiology of pleurisy, it is considered by some as solely inflammatory; clinically it presents the picture of chronic pleurisy plus occasional metastatic deposits in the liver, lungs, kidneys, muscles and lymph glands. The signs are those of lung tumor plus those of pleurisy; the clinical diagnosis of primary tumor is at the best but probable, even if tumor particles are obtained by puncture.

DISEASES OF THE MEDIASTINUM.

MEDIASTINAL TUMORS.

Mediastinal tumors are very rare. They are usually primary. Sarcoma is the most common form and then carcinoma; lipoma (7 reported cases), substernal struma, thymic growths, fibroma, myoma,

osteoma, enchondroma, echinococcus, dermoid (64 cases collected by Christian, 1908), teratoma, gumma, tubercle, leukæmic, pseudoleukæmic and chloromatous adenopathies are rarer forms. Sixty per cent. occur in males between twenty and thirty years of age.

Symptoms.—Symptoms develop gradually. (1) *Local signs:*—(*a*) Bulging may be present in the jugulum or under and near the sternum, especially in lymphosarcoma, or it may show in the anterior mediastinum and cause stretching of the skin. (*b*) There may be pulsation, imparted by the heart, aorta or its branches; expansile pulsation is rare. (*c*) The tumor or secondarily involved glands may be palpable deep in the jugular notch. The vocal fremitus varies. (*d*) The sternum may be exquisitely tender from erosion. (*e*) Dulness may be elicited under or near the sternum, or, if the posterior mediastinum is involved, in the back; the dulness may blend with the heart, suggesting aneurysm or effusive pericarditis, though it is more irregular than in either of these affections. (*f*) Auscultation gives variable results; sometimes there is bronchial breathing, or if the bronchi are closed the breath sounds may be absent.

2. *Compression symptoms,* as in aneurysm, may predominate in tumors of the posterior and middle mediastinum, though local physical findings may be slight or absent. (*a*) The vagus and its branches may be affected. Paroxysmal asthma and orthopnœa are common. Dyspnœa is the earliest and most frequent symptom of mediastinal tumor, is partly due to compression of the vagus, and partly to tracheal, cardiac and pleuritic involvement. Other vagus symptoms are brazen cough from paralysis of the vocal cords, tachy- or bradycardia, hiccough, vomiting or œsophageal spasm. (*b*) The trachea or bronchi may be compressed from without or occluded by growth into their lumina, in which case the symptoms of tracheal or bronchial stenosis may be pronounced. Tracheal tugging and shrinking of one side of the chest (instead of bulging) may be noted. The sputum may contain blood, tumor tissue or hair. The larynx may show lateral deviation. (*c*) The arteries of the neck may show a systolic stenotic murmur, but the veins are more often compressed, especially the innominates or the superior cava, which cause bilateral or unilateral œdema and, less often, cyanosis of the face, neck and arms; enlargement of the veins on the thorax indicate the collateral circulation. (*d*) The heart may be compressed and dislocated, downward and outward if the growth emanates from the anterior mediastinum, forward if from the posterior mediastinum. Dislocation of the liver and spleen are less common. (*e*) The œsophagus may be so compressed and dysphagia may be so extreme that death from inanition results. (*f*) Inequality in the pupils is uncommon. (*g*) Neuralgia, muscular paralysis or even spinal paraplegia may result. The boring pain of aneurysm is rare.

Diagnosis.—A diagnosis is positive from the signs, or presumptive from the symptoms. Dyspnœa, ectasia, irregular substernal dulness,

venous stasis, bronchial stenosis, heart luxation and the *x*-rays picture make the diagnosis probable; puncture may both eliminate the possibility of aneurysm and withdraw tissue or dermoid material (Türck); the sputum sometimes voids tumor particles, hooklets and hair. Deep aneurysm (*q. v.*) is most difficult to eliminate. The irregular dulness and the apex beat at its extreme limit to the left exclude pericarditis. Tumors of the lung and pleura produce less pressure symptoms, though such differentiation is rather academic.

Prognosis and Treatment.—The prognosis is unfavorable, as malignant growths cause emaciation and death from inanition, compression, exhaustion or pneumonia results within a few months. Benign tumors run a longer course, sometimes five to seven years, or even forty in Lebert's dermoid case. Treatment is palliative. A few successful operations are reported: in twenty operated cases of dermoid, 70 per cent. recovered (R. S. Morris).

MEDIASTINITIS.

Acute inflammation is uncommon, but may follow various local lesions, as pleurisy, pericarditis, pneumonia, acute lymphadenitis and kindred processes (*v.* Mediastinal Pleurisy) by contiguity or by metastasis. (*a*) Acute nonsuppurative cases begin with chill, fever, pain in the mediastinum and tenderness anteriorly over the sternum or high in the back; there may be œdema over the sternum; dysphagia, dyspnœa and vague cardiac symptoms may develop. A presumptive diagnosis is made from acute inflammatory symptoms with mediastinal localization. The prognosis is grave and the treatment is symptomatic: an ice-bag should be applied over the sternum and opiates should be given for pain. (*b*) Of *mediastinal abscess,* Hare (1899) collected 115 cases in most of which the anterior mediastinum was affected. They occurred chiefly in males. Acute cases, most often traumatic or sequential to the eruptive diseases, were more frequent than chronic cases, which were usually tuberculous (*v.* Mediastinal Pleurisy).

Symptoms.—The symptoms are mediastinal, as in simple acute mediastinitis, and general or septic. A throbbing retrosternal pain is common and dyspnœa may result from pressure by large pus pockets. Œdema, fluctuation and pointing in the upper interspaces or in the jugular notch may be noted. Fatal hemorrhage may follow simultaneous erosion of the aorta and bronchus. External rupture may occur in the second interspace (Daudé) or internal rupture into the air passages, bloodvessels, œsophagus or pleura; deep burrowing to the abdomen has occurred. The exploring needle is of great diagnostic aid in doubtful cases. Operation is indicated: Auvray (1904) found in the literature 36 operations with 33 recoveries.

(*c*) *Chronic mediastinitis* is discussed under adhesive pericarditis.

MEDIASTINAL HEMORRHAGE.

Small hemorrhages of no clinical importance may result from "blood dissolution," as in icterus, hemorrhagic blood diseases or acute infections. Larger hemorrhages may follow trauma or erosion of the large vessels, both of which usually overshadow the mediastinal incident.

INTERSTITIAL EMPHYSEMA.

(See DIFFERENTIATION OF EMPHYSEMA.)

SECTION IV.

DISEASES OF THE DIGESTIVE TRACT.

DISEASES OF THE MOUTH.

CATARRHAL STOMATITIS.

Etiology.—Simple, acute or erythematous stomatitis is the most frequent form. In children it may develop with dentition, improper feeding, rude cleansing of the mouth and digestive disorders, especially in those who are poorly nourished; it is part of various infections, notably of the exanthemata and syphilis. In adults it results from thermal, mechanical and chemical irritants; hot food, alcohol, tobacco, various chemicals (iodine, mercury, arsenic, copper, etc.), acidulous vomiting, inflammatory extension from the salivary glands, carious teeth, nose and throat disease, uncleanliness, anæmias and stasis are among its causal factors.

Symptoms.—Symptoms are chiefly redness, swelling and increased secretion, and in the acute forms burning pain which makes eating difficult. The increased saliva is usually acid and irritates the lips or chin if it flows over them. The gums are turgid (*gingivitis*) and the tongue broadened, lax and therefore indented by the teeth, as is also the buccal mucosa. The tongue is generally coated. Vesicles sometimes form and break down into small erosions which heal with cicatrices. The whitish areas sometimes seen are epithelial thickening. Fever, thirst and general symptoms are absent, except in some infantile cases. Acute stomatitis lasts but a week or two, but may become chronic. Transient stomatitis is physiological in the newborn.

Treatment.—In children the mouth should be gently washed after each nursing. Cold water or ice mitigates pain. Boracic acid is excellent and safe. In adults the teeth should be cared for. Tincture of myrrh and tincture of rhatany—equal parts—are excellent astringents. Stronger antiseptics may be used in adults but are dangerous in children, who may swallow them; for instance, 2 per cent. sodium salicylate, 2 per cent. chlorate of potash, 1 per cent. carbolic solution; erosions may be touched with 1 per cent. silver nitrate.

STOMATITIS ULCEROSA.

Etiology.—Though probably a mycotic affection, its bacteriology is unknown. In some cases the same microörganisms are found as in

Vincent's angina. Sporadic cases are more common than epidemics, which may occur in asylums or prisons. It may be induced by malnutrition, uncleanliness, oral or dental disease, phthisis, leukæmia, diabetes, toxic factors (principally mercury), and occurs especially at dentition and more in city than in country children. (See Diagnosis of Diphtheria and Acute Pharyngitis.)

Symptoms.—The symptoms are those of catarrhal stomatitis plus necrosis and then ulceration. The gums, especially about the incisors and canines, are red, swollen and spongy; they bleed readily, secrete pus and ulcerate; over the ulcers a membrane develops. The teeth are exposed, loosened and perhaps fall out. Necrosis of the jaw sometimes results. The tongue and cheek ulcerate less often and the pharynx is nearly exempt. The breath is fœtid, the saliva is increased, the submaxillary lymph glands and the salivary glands are swollen and mastication is almost impossible. Fever, prostration and signs of sepsis, as erythema, chills and rapid pulse, may mark the severest cases. Acute cases last one to two weeks and chronic cases weeks or months.

Varieties.—(*a*) *Mercurial stomatitis* may occur in workers in the metals or from clinical administration. The author saw a hospital case of nephritis in which one dose of five grains of calomel caused necrosis of the entire tongue, which rotted off at its root. (See Intoxications.) Mercurial and other stomatitis may cause "erosions" of the enamel in the developing teeth of children; they become pitted, discolored and transversely furrowed. (*b*) *Riga's disease,* described by Riga (1881) in south Italy, is an affection in which there is a pearly induration which occurs only between the tip of the tongue and the frænum, appears with the first dentition, sometimes ulcerates, is occasionally epidemic, is often observed with the status lymphaticus and is invariably fatal. (*c*) In *nursing women,* ulcers, 3 to 4 mm. in diameter, may develop from the lymph follicles of the lip and cheek; they usually indicate malnutrition, sometimes are quite painful and respond to general tonics and hygiene. (*d*) *Herpetic or pemphigoid stomatitis* is seen in neurotics and is frequently recurrent (Jacobi's stomatitis chronica neurotica). (*e*) Bednar described (aphthous) *white plaques* on the posterior part of the hard palate near the alveolar border of the upper jaw, which may ulcerate; they occur in the first three months of life, and are apparently due to sucking, are not syphilitic, and are ominous only in marantic children. (*f*) *Parrot's ulcers* occur in marantic new-born children on the hard palate near the median line; they seldom heal.

Treatment.—Treatment includes that of catarrhal stomatitis. Potassium permanganate 1 to 1,000 relieves the *fœtor ex ore.* Necrotic areas may be touched with silver nitrate solution or equal parts of tincture of the chloride of iron and glycerine. Potassium chlorate gargles may be combined in children with gr. x to xx of the drug every day internally and twice the amount for adults, but it must be remembered that toxic effects may result. Opium and belladonna are used in severe cases for pain and salivation.

APHTHOUS STOMATITIS.

Etiology.—It may occur alone or with other diseases, sporadically or epidemically. It is probably contagious but its nature is still undetermined; when due to milk it ceases on withdrawal of the milk. Besides the general causes of stomatitis (*v. s.*), it occurs chiefly in children between ten and thirty months old.

Symptoms.—Symptoms begin with restlessness, slight fever, thirst, salivation and pain in the mouth. Round, yellow-white and slightly elevated areas as large as a pea appear on the surface and edges of the tongue and less often on the lip and buccal mucosa. The mucosa is slightly reddened about them. Attempts to scrape them off produce bleeding. Pathologically they are areas of superficial necrosis, into which exudation of fibrin and round cells occurs (stomatitis fibrinosa disseminata). Vesicles seem to be no part of the process, though they may occur on the skin near the mouth. Occasionally there is tumefaction of the lymphatic and salivary glands. Their clinical evolution is rapid; they last one to two weeks, and recurrence is frequent. The *treatment* is that of stomatitis.

PARASITIC STOMATITIS.

Etiology.—*Thrush, muguet, soor* or *stomatomycosis oïdica* is due to the Oïdium albicans, closely allied to the saccharomyces, which consists of branching mycelia and ovoid granular and refracting spores. It occurs chiefly in very young or marantic children whose mouths have been improperly cared for, or who have stomatitis. It prevails especially among the poor and in institutions. Dirty nipples are the most frequent mode of inoculation. Thrush may develop in weakly convalescents or in tuberculous and diabetic adults.

Symptoms.—A few thin, punctate, whitish spots develop on the tongue, cheek and soft palate; they become yellowish as the areas grow or fuse. The fungus is at first deposited on the epithelium, between the layers of which it proliferates and penetrates. The mouth is dry or, with diffuse stomatitis, is moist from increased salivary flow. In the more severe cases the fungus may reach the pharynx, larynx, œsophagus and even the stomach and intestine. Its deposit is generally limited to areas with flat epithelium, and epithelia of the cylindrical and ciliated order usually escape. It may, in exceptional cases, cause œsophageal occlusion or bronchopneumonia; or by unusually deep penetration to the submucous vessels it may cause cerebral, renal and other embolism. The *outlook* is good except in marantic subjects.

Treatment.—*Prophylaxis* is most essential in regard to cleansing of the mouth, sterilization of bottles and nipples and maintenance of the general health by fresh air and early treatment of dyspeptic disorders. In some cases the stomatitis persists until a wet-nurse is substituted for the feeding with cow's milk. Cleansing the mouth

with *borax,* boric acid, sodium bicarbonate and lime water is most effective. If unusual extension occurs a teaspoonful of a resorcin solution (1 to 100 or 300 parts) may be given every two hours.

Stomatitis may in rare cases result from *other parasites,* as the sarcina or leptothrix, and from such bacteria as the pneumococcus, gonococcus, Friedländer's bacillus, etc.

GANGRENOUS STOMATITIS.

Etiology.—"Gangrene of the cheek," *noma* or *cancrum oris,* is a rare affection. *Age:* of Brun's 415 cases, only 11 occurred in persons over fifteen years of age and only 6 in infants. The author recently saw noma in a man forty years old, which was fatal on the fifth day. *Sex:* more cases occur in girls than in boys. *Malnutrition* and neglect cause some cases; 50 per cent. follow measles; typhoid, scarlatina, etc., are less frequent factors. A bacillus resembling that of diphtheria has been isolated (Bac. necroseos). Organisms similar to those in Vincent's angina may be seen.

Symptoms.—Symptoms usually begin with ulceration of the gums or buccal mucosa near the angle of the mouth. Rapid necrosis and gangrene follow with extensive brawny œdema of the face. The fœtor is intense and much tissue is frequently sloughed off. Perforation of the cheek, necrosis of the jaw and extension to the throat, orbit or ears are not uncommon. Seventy per cent. of cases die within a week, with such septic symptoms as high fever, weak heart, prostration, diarrhœa, colitis and meningitis or lobular pneumonia. The mortality was 92 per cent. in Springer's collation.

Treatment.—Treatment is ineffectual. A full diet and free exhibition of alcohol are indicated as in sepsis, and disinfectants should be applied to the necrotic focus; local measures as excision, the live cautery and caustics are employed, usually without success and sometimes with aggravation of the necrosis.

DISEASES OF THE TONGUE.

ECZEMA.

In eczema the epithelium thickens and desquamates, leaving reddish, smooth and circular, serpiginous or geographical areas, which heal centrally and extend peripherally. It may cause local irritation or worry the patient, who may think he has syphilis or cancer. Again, it is an accidental finding. Its cause is unknown, its course is chronic and its treatment by silver nitrate is quite inadequate.

LEUKOPLAKIA.

Leukoplakia (psoriasis, ichthyosis, keratosis) is an affection closely resembling the "geographical tongue," in connection with which it is often described. Irregularly-shaped, smooth, pale plaques measuring one to two centimeters, develop on the tongue and less often on the lips, cheeks or tonsils; they consist of thickened epithelium. Eighty per cent. of cases occur in men (98 per cent., Fournier); the use of tobacco and alcohol, and gout and gastric disease are probable factors; Fournier's experience with 324 cases convinced him that 80 per cent. came from syphilis and 20 per cent. from tobacco. Its symptoms are not marked, its course is very chronic and its treatment is nearly always without results. Ten per cent. solution of sodium salicylate; chromic acid, 5 per cent.; strong silver nitrate applications, especially to fissured places; corrosive sublimate (1 to 500 parts) and the galvanocautery are recommended. Excision is necessary for papillomatous or epitheliomatous outgrowths, which are said to develop in one-third to one-half the cases.

ACUTE GLOSSITIS.

Acute glossitis may be localized or diffuse, and ends in suppuration in 33 per cent. It follows stings, burns, erosions, trauma or the entrance of pyogenic cocci through the lymph follicles. The tongue is swollen, sometimes so greatly that it protrudes beyond the lips, and is painful. In 145 cases the mortality was 3 per cent (Bennett, 1906). Eating and even speaking or oral breathing are impossible. Early scarification and later incision of purulent foci are indicated.

GLOSSITIS DESICCANS.

Glossitis desiccans is a rare, chronic, intractable affection in which deep indentations of the margins of the tongue and consequent lobulation occur. *Melanoglossia* (*nigrities linguæ*) results from proliferation of the filiform papillæ at the base of the tongue, which appear like hair (*melanotrichia linguæ,* black-haired tongue); they are removed by a 10 per cent. salicylic acid solution or by curetting. The *mucor niger* may cause a black coating on the tongue.

DISEASES OF THE SALIVARY GLANDS.

PTYALISM, SALIVATION, HYPERSECRETION, SIALORRHŒA.

Etiology.—The chief causes are (*a*) disease of the mouth, stomatitis, dentition; (*b*) toxic substances, as jaborandi, tobacco, digitalis, mercury, iodides and various metals; (*c*) nervous affections, as trigemi-

nal neuralgia, diseases of the pons and medulla, paretic dementia, hypochondriasis and hydrophobia; a close distinction should be drawn between oversecretion and lesions in which, like bulbar paralysis, the saliva cannot be swallowed; (*d*) reflex salivation, which may also occur from diseases of the tympanum through the chorda tympani, from gastro-intestinal disease (gastric ulcer, pancreatic lesions, etc.) by way of the vagus and sympathetic nerves and from genital conditions, as pregnancy.

Symptoms.—The physiological salivary flow measures seven ounces to three pints; a pathological flow of over twelve quarts has been recorded. The salivary glands are tense, swollen and hard. The reaction of the saliva may become neutral or acid; it is chiefly thin and fluid, but may contain some ropy mucus or scattered pus cells; the ptyalin disappears together with the potassium sulphocyanide. The saliva may trickle into the larynx during sleep, and thus cause violent coughing; it may flow into the stomach and produce vomiting, of an alkaline or neutral vomitus, especially in the morning; it may stream from the lips and redden the adjacent skin.

Prognosis and Treatment.—The prognosis depends wholly on the causal affections, whose operation may be transient, intermittent or permanent. Treatment is likewise causal. Any toxic cause should be removed. In nervous and reflex types potassium bromide is often valuable and the iodides are sometimes efficacious in pregnancy. The more severe cases necessitate opium and belladonna.

XEROSTOMIA.

Hyposecretion or arrested secretion was first described by Jonathan Hutchinson (1888). Besides its occurrence, as a result of atropine and allied remedies, it occurs in diabetes insipidus and mellitus, in some cases of contracted kidney and finally as an independent neurosis or a neurotic symptom. Women (80 per cent.) are affected more often than men. The tongue and mouth are dry, red and glistening, which may render speaking, chewing and swallowing most difficult. The tongue may cleave to the roof of the mouth. The digestion of carbohydrates is impaired. *Treatment* consists in painting the mouth with iodine, potassium iodide and glycerine (1, 10, 100 parts respectively), the administration of pilocarpine hydrochloride $\frac{1}{12}$ to $\frac{1}{8}$ of a grain and galvanic applications to the parotid.

PAROTITIS.

Besides acute specific parotitis (mumps), infection may reach the parotid or other salivary glands by way of the blood stream or by infection entering their ducts. *Acute parotitis* may be simple but is oftener suppurative, the staphylococcus being the most common pyogenic organism. (*a*) Any of the acute infections may be complicated by parotitis, of which typhoid is the most common. (*b*) Affections

of the abdominal or pelvic structures are sometimes complicated by acute parotitis, to which Paget drew special attention; they include diseases of or operations on the alimentary and genito-urinary tracts, peritoneal disease, pancreatitis, abdominal trauma, etc.; many of these cases are clearly attenuated sepsis. Wagner, in 1904, was able to collect 61 cases of postoperative parotitis; 31 were operations upon the genitalia; he believes the cause is not hæmatogenous infection, but an infection ascending along the duct from the mouth. (*c*) A rare acute parotitis in facial neuritis is described by Gowers.

Chronic parotitis sometimes follows epidemic or symptomatic parotitis, poisoning by mercury or lead, syphilis or chronic nephritis. The parotid is usually painless, though it is sometimes both painful and tender. Mikulicz has described a "chronic symmetrical hypertrophy of the salivary and lachrymal glands."

Treatment.—In acute parotitis, local derivants as leeches, the early application of cold and the later use of hot fomentations, and finally surgical incision, if there is pus formation, are indicated. Chronic forms respond slowly to treatment; iodine or mercurial ointment should be employed. Iodide is useful in Mikulicz's disease.

ANGINA LUDOVICI.

Ludwig's angina is generally due to a streptococcic infection beginning in the submaxillary gland, and extending to the floor of the mouth and the cervical cellular tissue. In some cases no promoting cause is found, or trauma may be its immediate antecedent, but most cases are secondary to typhoid, diphtheria and kindred acute infections. It is the analogue of acute phlegmon of the pharynx and larynx. The inflammation spreads rapidly, causing fever, redness, pain and swelling under the tongue, cellulitis and sometimes gangrene in the neck (*cynanche gangræneuse*). Mastication and deglutition may be impossible and glottis œdema is not infrequent. The parotids are sometimes invaded. Resolution is very exceptional and external pointing of pus is not likely to occur, whence, without early surgical intervention, septic and pyæmic complications or laryngeal and pharyngeal œdema or phlegmon very frequently develop. The mortality is 40 per cent.

SIALODOCHITIS FIBRINOSA.

Inflammation of the salivary ducts with formation of membrane (Kussmaul) results from infection ascending from the oral cavity and is characterized by fever and tenderness, pain and tumefaction of the salivary gland, due to retained secretion and infection. The duct is kept patent by pressure or sounding.

SIALOLITHIASIS.

Stones of calcium phosphate or carbonate occur more frequently in Wharton's duct than in the other salivary ducts. Their usual cause is stagnation of the salivary secretion by foreign bodies, bacterial or fungus invasion or mechanical stricture following ulceration. Roberg (1904) found stones in Wharton's duct fifty times, in the submaxillary gland twenty-eight times, in Stenson's duct six times and in the parotid gland twice. Bendixen collected 216 cases (1908).

DISEASES OF THE PHARYNX.

ACUTE PHARYNGITIS.

Etiology.—Acute, superficial, simple angina may be caused by (*a*) rheumatism, cold or exposure; (*b*) infections, either acute (measles, scarlatina, variola) or chronic (malaria or syphilis); (*c*) gout, dyspepsia or smoking; (*d*) in many cases the cause is obscure; though probably always mycotic, no single virus is found. It is most common in youth.

Symptoms.—(*a*) *Local:*—The throat is red, glazed and streaked with mucopus which is easily wiped away. The soft palate and uvula are sometimes swollen; superficial erosions, small vesicles on the palate and swollen mucous follicles are occasionally seen. In some cases the local findings, especially when hidden in the nasopharynx, are very slight in proportion to the local pain and general symptoms. There is a scratching, tickling sensation in the throat which may radiate into the posterior nares or into the ear. The patient hawks, but raises little, unless there is coincident rhinitis or laryngitis. Swallowing is rather painful, slight deafness is quite common, the speech is sometimes slightly nasal and the angular lymph nodes are painful and tender. (*b*) *Constitutional symptoms* are usually slight. At the onset there are chilliness, moderate fever, aching in the muscles and in some individuals extreme cutaneous hyperæsthesia, which, on superficial examination, may be falsely attributed to changes in the deeper parts of the body. With or without coincident tonsillitis, there may be high fever, and in labile individuals, marked nervous intoxication. Herpes facialis occasionally develops. Complications, as acute endocarditis or nephritis, are very uncommon. In a few days the process is complete and convalescence is rapid.

Treatment.—(*a*) *Local:*—Painting the entire pharynx with a 20 per cent. silver nitrate solution usually affords local and general relief. (*b*) *General:*—Dover's powder and aspirin (acetylsalicylic acid), each ten grains, may be exhibited for unusual pain, aconite for

high fever, calomel, followed by salines, for elimination and a 1 per cent. solution of carbolic acid for the irritated throat.

The *pneumococcic sore throat* may be suppurative, pseudodiphtheritic, follicular (like tonsillitis), erythematous or herpetiform; its onset is severe, like that of pneumonia, it produces a thicker, denser and more adherent membrane than does the streptococcus and the prognosis is generally poor.

CHRONIC PHARYNGITIS.

Etiology.—The causes are: (*a*) repeated acute pharyngitis; (*b*) smoking or alcohol; (*c*) abuse of the voice, as in clergymen, venders, etc.; (*d*) extension from chronic nasopharyngitis or laryngitis; (*e*) chronic nephritis, syphilis, arthritism, etc. These factors are most common in adolescence and middle life.

Symptoms.—The mucosa of the nasopharynx is red and lined with turgid venules; the pillars and uvula are relaxed and the drooping uvula may tickle the tongue or epiglottis, causing chronic coughing. The sides of the pharynx may show isolated whitish epithelial thickening or more often small, reddish, hypertrophied lymph follicles (*pharyngitis granulosa*), sometimes in linear distribution. The pharynx is dry and burning, exciting hawking efforts which are unproductive and irritating and may cause morning vomiting. The mucous glands are at times hyperplastic. Slight bleeding occasionally causes unnecessary worry. Middle ear involvement is common, through the Eustachian tube. *Pharyngitis sicca* is an atrophic type, analogous to rhinitis atrophica and marked by a pallid glazing of the posterior pharyngeal wall and by formation of crusts.

Treatment.—(*a*) The *causal factors*, as straining or tension of the voice in speaking, alcoholism or smoking, etc., should be corrected respectively, by rest and avoiding irritant foods or stimulants. Alkaline laxatives are frequently beneficial. (*b*) *Galvano-puncture* of the turgid veins or hyperplastic follicles is indicated; marked improvement is probably more frequent than aggravation of the condition by over-zealous treatment. (*c*) *Local medical treatment* is less efficacious, as 2 to 20 per cent. solution of tannin in glycerine, equal parts of tr. ferri chloridi and chlorate of potash, 1 to 5 per cent. solution of chloride of zinc or 2 to 20 per cent. nitrate of silver solution.

℞ Phenolis ..... gr. xx.
Acidi tannici ..... ʒss.
Aluminis ..... ʒj.
Glycerini ..... ℥ij.
Aquæ rosæ ..... q.s. ad ʒv.

M. et ft. gargarismus.
S.—As gargle, properly diluted.

℞ Tincturæ ferri chloridi ..... ʒijss.
Potassii chloratis ..... ʒiij.
Aquæ ..... q.s. f℥vj.

M. et S.—One teaspoonful in one-half glass of water as gargle.

(*d*) For *atrophic* pharyngitis, iodine, potassium iodide and glycerine massaged thoroughly into the pharynx, is the best measure:

℞ Iodi ..... gr. iij.
Potassii iodidi ..... ʒss.
Glycerini ..... ʒj.
M. et S.—Locally.

PHLEGMONOUS PHARYNGITIS.

Angina phlegmonosa, especially described by Senator, is not superficial but invades the submucosa. It is usually primary but may be metastatic from distant suppuration. The throat is painful, swollen and œdematous, the neck enlarges and suppuration occurs with high fever, septic symptoms and difficulty in swallowing and breathing, especially when the subject lies down. The inflammation may be hemorrhagic or necrotic. Phlegmonous angina is the analogue of suppurative submucous laryngitis and angina Ludovici. Without surgical intervention or early spontaneous rupture into the throat, death occurs from septicopyæmia, mediastinitis or glottis œdema.

RETROPHARYNGEAL ABSCESS.

Various forms are described. (*a*) The *primary* form occurs in children under two years of age; the early symptoms are dysphagia, a nasal voice and retropharyngeal suppuration, as a visible protrusion or a palpable fluctuation on the posterior pharyngeal wall; dyspnœa, cyanosis and inspiratory retraction of the chest develop. It is a primary lymphadenitis. (*b*) Secondary types are less common in (*i*) caries of the cervical spine, (*ii*) diphtheria, erysipelas, scarlatina and (*iii*) suppurative inflammation in the retropharyngeal lymph glands. The secondary type is caused by infection travelling along the lymph vessels, or rarely by metastasis. Unless surgical measures are instituted or spontaneous rupture occurs early, death occurs from asphyxia, mediastinitis, glottis stenosis, inhalation pneumonia or sepsis. After incision the head should be promptly thrown well forward to obviate occlusion of the air passages.

PHARYNGEAL ULCERATION.

Syphilitic and tuberculous ulceration have been considered. Follicular ulcers are superficial and small and, like all ulcers in the mucous membrane only, heal without a cicatrix. Ulceration also occurs in diphtheria and the diphtheroid group, due chiefly to the streptococcus. Malignancy too, causes ulceration. Cancer and tuberculosis are usually diagnosticated with ease, and in doubtful cases syphilis is excluded *ex juvantibus* by the use of mercury and iodides.

Vincent's angina is discussed under Diagnosis of Diphtheria; according to Vincent, it constitutes two per cent. of all anginas in-

cluding diphtheria. It occurs especially in children between six and ten years of age and in medical students and servants in anatomical laboratories, between the years of eighteen and thirty. Tobacco, scurvy, anæmia, tuberculosis, syphilis, scarlatina, measles and the eruption of the wisdom teeth are predisposing factors. The two causative organisms were first described by Rauchfus (1893); (*a*) the *fusiform bacillus* has pointed, sometimes rounded ends, is sometimes bent, measures six to twelve μ in length and may be flagellated; (*b*) the *spirillum* or spirochæte is long, delicate, twisted and always occurs in severe forms. They stain with carbol-fuchsin but not by Gram's method.

Symptoms.—(1) In the first period, there is *congestion* and œdema. (2) In the second stage, a grayish yellow *membrane* forms which is thick, friable and cheesy and usually removable. (3) Under the membrane, an *ulcer* develops; it is usually single, round or oval and has an irregular edge, an uneven granulating floor and seldom any extension laterally.

The general symptoms are lassitude, indigestion, vomiting or epistaxis; fever is uncommon and complications rare (albuminuria, enteritis or noma). Syphilis and diphtheria require differentiation. The affection heals under applications of iodine, pencilling with silver nitrate and gargles of hydrogen peroxide or chlorate of potash.

DISEASES OF THE TONSILS.

ACUTE FOLLICULAR OR LACUNAR TONSILLITIS.

Definition.—An acute mycotic parenchymatous inflammation of the tonsils, usually marked by decided constitutional reaction.

Etiology.—(*a*) *Bacteriologically,* the streptococcus is the most frequent cause, though the staphylococcus and Fränkel's diplococcus are also encountered; it is claimed that somewhat attenuated diphtheria bacilli are found, particularly at the time of diphtheria epidemics, but in such cases it is more correct to consider it as a sub-type of diphtheria (*q. v.*). (*b*) The still unknown virus of *rheumatism* is thought to gain access to the blood by way of the tonsils or pharynx; affections commonly regarded as associated with rheumatism, as erythema nodosum, chorea and some forms of endocarditis, may follow acute tonsillitis (Packard). Tonsillitis may begin the rheumatic cycle, as described under rheumatism in children, or it may appear later in the rheumatic process, with endocarditis, pericarditis, arthritis and chorea. Endemics and the spread of the disease through a household strengthen the conception that tonsillitis is contagious. (*c*) *Cold* and *dampness* are directly predisposing causes; these conditions obtain largely in the spring. (*d*) Tonsillitis occurs chiefly in youth

and adolescence. (*e*) *Individual predisposition* depends on some unknown factor and one attack may induce recurrence. (*f*) *Poor hygiene* and possibly sexual activity, especially in young married people, may be conducive to tonsillitis (Shepard).

Symptoms.—(*a*) The *onset* occurs with chilliness or rigor, severe *pains in the back* and limbs, *fever,* rising within a day to 103 or even to 106 degrees in children or susceptible adults, *pain in the throat,* particularly on swallowing, and tenderness of the glands at the angle of the jaw. (*b*) *Local findings:*—The *tonsils are swollen* and soon show on the deeply injected surface three to ten yellowish-white or creamy points, corresponding to the crypts or lacunæ; the crypts contain bacteria, epithelium, leukocytes and sometimes cholesterin or fatty needles and are essentially small abscesses; the tonsil is the seat of serocellular infiltration. Occasionally there are small superficial areas of necrosis, slight erosions or even formation of membrane. The voice may acquire a somewhat nasal twang, the pain increases for a few days and the angular glands remain painful and tender, whence the head is often held rigidly forward and the jaws are opened only with difficulty. Pain in the ear is common. (*c*) The chief *constitutional symptoms* have been considered above. Anorexia, heavily coated tongue, feverish breath, hyperæsthetic skin, marked depression, accelerated pulse and respiration are common. In some cases the systemic symptoms are disproportionately severe and children, as well as some adults, may exhibit delirium; in two adult cases seen by the writer, retraction of the neck with headache, delirium and vomiting was mistaken for meningitis. Herpes facialis is quite frequent.

Complications.—Complications are not common. Acute pericarditis, endocarditis or nephritis may develop. Functional heart murmurs, febrile albuminuria and erythema (from the toxins or from medication) are not very infrequent. As a rule convalescence is complete within a week, though marked prostration and considerable residual swelling remain for some time. Repeated attacks may cause nephritis or endocarditis.

Diagnosis.—The typical case cannot be confused. (*a*) *Diphtheria* produces a membrane, more whitish in color, which has an almost pathognomonic tendency to spread to the uvula, soft palate or pharynx; it leaves bleeding raw points when it is stripped off; it recurs after removal; it sometimes extends to the larynx; it is followed not infrequently by paralysis, which almost never results from tonsillitis; it responds promptly to serotherapy and shows the Klebs-Loeffler bacillus. (*b*) *Tonsillitis necrotica* leaves an ulcer; its bacteriology is disputed, some cases showing the fusiform bacillus and spirillum of Vincent and others are alleged to be due to the Bacillus diphtheriæ; in some cases there is much fever; in others none; the general reaction is severe. (*c*) The possibility of incipient *scarlatina* should always be kept in mind.

Treatment.—1. Prevention.—The subject should be hardened by

cool baths or sponges, and tonsillotomy should be performed. Douching of the nose and gargling with Dobell's solution should be part of the daily toilet.

2. Local Measures.—The best of the many local remedies in the author's experience is *silver nitrate;* after cocainizing the throat, a small probe, bent at right angles near its end, is introduced into each swollen crypt to puncture it and secure free drainage; then Dobell's solution is applied, undiluted, on small bits of cotton in each crypt; this is followed by 20 per cent. silver nitrate solution; the results are often immediate, sometimes aborting the attack. Squeezing out the crypts with a spatula is extremely painful but relieves the patient when the physician has no probe at hand. Hot water or hot fomentations may be applied to the neck.

3. Constitutional Treatment.—In a relatively small number of cases sodium salicylate, given as in rheumatism, affords relief. Hydrargyri biniodide gr. $\frac{1}{500}$ given every fifteen minutes for ten doses often aborts the process. Guaiac, though generally used, has produced unsatisfactory results in personal experience; the dose is tr. guaiaci ammoniat. ♏x to xl every two hours. Aspirin (ac. acetylsalicylicum) and Dover's powder āā gr. x, relieve pain in the throat, body and limbs. Aconite and belladonna are often beneficial in children with high fever, and rather less so in adults; they are given as in coryza or influenza.

SUPPURATIVE TONSILLITIS.

Etiology.—The etiology is almost identical with that of the follicular type. Suppurative tonsillitis is most common in adolescence, and for unknown reasons individual disposition is an important factor.

Symptoms.—The early local and general symptoms resemble those of the follicular type but are often more severe from the onset; one, or less often both, tonsils are greatly swollen, tense, œdematous, deep red and may show distended lacunæ. The pain often radiates to the ears. The swollen tonsils may come in contact, or if but one tonsil is swollen it may reach over to the sound side. There is often coincident stomatitis and a free salivary flow. The palate and uvula are frequently œdematous and covered with muco-pus. Swallowing causes great pain in the throat, the jaw cannot be depressed because of the swollen, painful angular glands, and the speech is nasal. The toxæmic prostration is often profound, the fever ranges between 103 and 105 degrees and the pulse between 100 and 140.

Incision in the first two or three days evacuates no pus, only blood and serum escaping. In a few days fluctuation is felt in the tonsil, or anterior to and above the tonsil (peritonsillar abscess). Introduction of a probe into the early and often unsuccessful incision is followed by escape of pus and by immediate relief. The abscess may break spontaneously, usually forward and rarely toward the throat, when, in rare cases, especially when rupture occurs during

sleep, suffocation or aspiration pneumonia may follow. In equally rare instances ulceration may reach the carotid with sudden and fatal hemorrhage. Minute suppurative foci may cause septicæmia (Kretz).

Treatment.—(*a*) For prevention *tonsillectomy* by the galvanic snare is indicated after an individual tendency is manifested. (*b*) In *local treatment* an ice-bag placed over the neck is beneficial. Local applications to the tonsils are at the best uncertain remedies. It is good practice, early in the course, to introduce a knife, *with extreme care,* covered with adhesive plaster to within half an inch of its point, above and inside the tonsil; blood and serum escape, tension, œdema and pain are relieved and the later abscess more readily points toward the cut; the wound is carefully opened with a blunt probe every day. (*c*) *Salicylates* apparently help and possibly hasten maturation. *Opium* and *coal tar products* relieve the pain to some extent. (*d*) In exceptional cases, intubation or tracheotomy are indicated, because of great swelling or collateral œdema. (*e*) The diet is necessarily liquid. (*f*) In *convalescence*, iron and strychnine overcome the resulting prostration and anæmia.

CHRONIC TONSILLITIS.

Symptoms.—Chronic inflammation; tonsillar hypertrophy; hyperplasia of the pharyngeal or lingual tonsils; adenoids; aprosexia.

Etiology.—(*a*) Repeated *acute attacks* of tonsillitis cause chronic hypertrophy in some cases, and in others the tonsils gradually indurate and shrink. (*b*) Waldeyer's "Schlundring" of *lymphadenoid* tissue consists of the *two tonsils,* the adenoid tissue in the nasopharyngeal vault (Köllicker's pharyngeal tonsil, or "*adenoids*" in the common acceptation) and the so-called *lingual tonsil.* Variously combined hypertrophies of these structures may be noted, occasionally as congenital growths, but most frequently in young children. They generally begin during the third or fourth year and may develop until puberty. Adenoids are found in 1 per cent. of children (Meyer), though other statistics give 3 per cent. or higher figures. Rickets, scrofula, the lymphatic constitution and various acute infections involving the throat, as diphtheria, are cited as predisposing conditions.

Symptoms.—1. Local Changes.—(*a*) The *enlarged tonsil* may contain in its crypts cheesy plugs, which occasion an extreme *fœtor ex ore.* The plugs may be expressed; calculi may result from a deposit of lime salts. The pharyngeal mucus is increased, is sometimes tinged with blood and is expectorated with difficulty. (*b*) *Adenoids* in the nasopharyngeal vault occur alone or with tonsillar hyperplasia. They are most often papillomatous, reddish, vascular and range up to the size of a bean. (*c*) Hypertrophy of the "*lingual tonsil*" often disturbs deglutition. Enlarged tonsils and adenoids produce the following changes by stenosing the posterior nares.

PLATE XIII

FIG. 1

FIG. 2

Types of the "Adenoid" Facies. (Posey and Wright.)

2. Sequences.—(*a*) *Oral breathing* is the earliest symptom and is first noticed during the night, when sleep is disturbed by night terrors (*pavor nocturnus*) and by disordered and sometimes irregular breathing, for a time stertorous and then interrupted. Lying down may occasion asthmatic seizures or Balne's paroxysmal cough. (*b*) After some time *changes in the mouth* are noted. The *palatal vault* is *high* from atmospheric pressure in the mouth, and the transverse measurement between the upper teeth is proportionately diminished; there is less room for the teeth to erupt. (*c*) In the nose, the *anterior nares are small* and retracted and the voice is nasal (*rhinolalia claustra*); the consonants *n* and *m* are pronounced with difficulty. Bloch associated oral breathing with stuttering. The sense of smell or taste is obtunded. Hearing is impaired by pressure of adenoids on the Eustachian tubes, inflammation in the tubes or retraction of the tympana from low atmospheric pressure in the nasopharynx. (*d*) The *facies* is characteristic; it is *vacuous* and *apathetic,* the nose is pinched, the mouth open and the lips swollen. The so-called "adenoid habitus" may be observed without adenoids, as a stigma of degeneracy. (*e*) The *mentality* is altered in chronic cases; the cerebral reaction is slow, the memory tardy, the disposition sullen and the power of concentration diminished; this latter condition was named aprosexia by Guye. Woodward refers the physical changes to some cytotoxin elaborated in the hyperplastic lymphoid tissue. (*f*) *Nervous symptoms.* Headache is common. Adenoids cause some cases of habit-spasm. Night terrors have been mentioned. Enuresis nocturna results from carbon dioxide operating on the brain during sleep (Schech). (*g*) *Development* is sometimes retarded. (*h*) Changes in the *contour of the thorax* were noted by Dupuytren (1828), J. M. Warren (1839) and Lambron (1861). The pigeon- or *chicken-breast* is the most common deformity. The sternum juts forward, from which the ribs slant sharply backward; at the level of the diaphragm there is a circular depression, and inspiratory retraction is caused by the contraction of the diaphragm. The *funnel breast,* marked by a deep depression of the lower part of the sternum, is less commonly due to adenoids, and like the chicken-breast also results from other causes. The "emphysema chest" may result from adenoids. (*i*) Scheir finds that the cervical lymph glands are also enlarged and the blood evidences decreased hæmoglobin, increased white cells and distinct lymphocytosis. (Plate XIII.)

Diagnosis.—The oral breathing, adenoid facies, mental disturbance, palpation of the adenoids in young children and their rhinoscopic detection in older ones, and the mere inspection of hyperplastic tonsils determine the diagnosis. In the *prognosis* of untreated cases, the possibility of deafness, mental deficiency, retarded growth, asthma and emphysema should be borne in mind. Colds, acute recurrent tonsillitis, diphtheria and other infections are more likely to develop and produce disproportionate symptoms. In some cases the hyperplastic tissues may indurate and atrophy, but this spontaneous issue should not influence their management.

Treatment.—Palliative treatment is useless. The general health should be maintained by out-door life, an ample diet, hydrotherapy and by cod-liver oil, iron and other measures employed in scrofula and tuberculosis. Hypertrophied tonsils should be removed by the *galvanic cautery*, for details of which the reader is referred to special treatises. Adenoids large enough to cause symptoms should always be removed early under anæsthesia, by the curette or finger; ether is far less dangerous than chloroform, which frequently causes death in disease of the lymphadenoid tissues (the *status thymicus* or *lymphoid constitution*). The sequences described usually subside after operation, though oral breathing is a habit hard to break. At night a bandage should be used to keep the mouth closed.

DISEASES OF THE ŒSOPHAGUS.

INFLAMMATION, NECROSIS, ULCERATION.

I. Œsophagitis.—This is an uncommon finding at autopsy. Its *causes* are (*a*) mechanical, as from foreign bodies, strictures or dilatation of the œsophagus; (*b*) chemical, as from alcohol, tobacco, corrosive poisons or acid vomitus; (*c*) hypostatic, in cardiac or pulmonary affections; (*d*) rarely extension from pharyngeal or laryngeal inflammation; (*e*) acute infections, as smallpox and diphtheria. Œsophagitis is physiological in the new-born after the ingestion of the first meals of life. Its pathology is that of pharyngeal or nasal inflammation; it is marked by local or diffuse, acute or chronic redness, swelling and hypersecretion. Special *pathological forms* are follicular catarrh, sometimes resulting in follicular ulcers or retention cysts in the submucosa; exfoliative œsophagitis (o. desiccans superficialis); the fibrinous form, observed in acute infections, uræmia and rarely in hysteria; genuine diphtheria; the pustular eruption of smallpox; the toxic form from caustics or acids; only 12 cases are on record of the phlegmonous or purulent form which occurs as a single bulging submucous abscess or as diffuse submucous suppuration and often perforates in many places into the œsophagus which then presents a sieve-like aspect; forms due to the oïdium albicans or ray fungus, of which primary cases are reported. In chronic cases the epithelium may thicken (leukoplakia) or hypertrophy (papillomatous forms).

Symptoms.—Symptoms are absent in slight inflammation. In marked cases there is *pain*, continuous and substernal or elicited by speaking, or pressure on or movement of the spine. *Dysphagia* is constant in marked inflammations. Mucus, blood, pus or eschars, depending on the nature of the inflammation, are brought up by gagging efforts. Fever is common in the more severe types, in which the

local symptoms may be wholly subordinate to the causal diphtheria, smallpox, etc. In inflammation due to acids or alkalies, the history, local symptoms and eschars in the mouth and pharynx are distinctive.

Treatment.—Treatment is usually only palliative. Narcotics are given for pain, demulcents as acacia and ice, ice-cream and a liquid diet for the local irritation, and if these cause œsophageal irritation, nutrient enemata are given (*v.* page 602).

II. Necrosis.—This may be due to corrosive poisoning; pressure from struma, aneurysm, foreign bodies or sounds left in the œsophagus to dilate strictures; or "bed sores of the gullet," first described by Dittrich (1850) in laryngeal perichondritis, due to pressure of the larynx upon the gullet. *Œsophagomalacia* is softening of the part by regurgitation into it of the gastric acid; it occurs chiefly after death, but some cases appear to develop during the death agony.

III. Ulceration.—Aside from cancer (*v. i.*), follicular ulceration, caustic ulcers, syphilis and tuberculosis, there is the peptic or round ulcer. *Peptic ulcer* was first described by Cruveilhier, who correctly identified it with round ulcer of the stomach. Forty cases were collected by Tileston (1908). It appears in four main conditions (*a*) by extension from the round ulcer of the cardiac orifice, (*b*) in ulcers at the pylorus or duodenum, which produce gastric dilatation, (*c*) in multiple hemorrhagic infiltrations of the œsophagus, stomach and duodenum and (*d*) in alcoholism, arteriosclerosis and other imperfectly investigated conditions. Its chief symptom is *dysphagia* (50 per cent.); its pathology and complications, as hemorrhage, perforation and stenosis, as well as its treatment, are practically identical with those of gastric ulcer, with which it is often associated.

STENOSIS OR STRICTURE OF THE ŒSOPHAGUS.

Stenosis is the most important and frequent of œsophageal diseases. In this connection von Mikulicz's conclusion is important, *viz.* that the œsophagus is a closed tube only in its cervical segment and that its cardiac orifice is not physiologically closed.

Etiology.—(*a*) *Interstitial* factors, *i. e.*, in the wall of the œsophagus. are the most important; *cancer* (*v. i.*) causes 90 per cent. of all stenoses (*v.* Leyden, Kraus, Rosenbach); *ulceration* by caustics and acids comes next; then comes stenosis by diverticula or pouches (*v. i.*) which, bulging out of and beside the œsophagus, exert upon it a lateral compression; other rarer ulcerative stenoses result from healing gummata, tuberculous lesions, smallpox and other suppurative affections, and from peptic and diphtheritic ulcers; muscular spasm causes a few cases; congenital stenosis or atresia, of which 15 cases are recorded, occurs chiefly as a *cul de sac,* incomplete development or fistulous communications with the trachea or bronchi. (*b*) *Extra-œsophageal* factors, compressing the tube from without, include *aortic aneurysm* or abnormal branches from the aorta; vertebral tumor, cold abscess, exostosis or deformation; anthracotic lymphadenitis and

malignant lymphoma; thyroid or thymic inflammation or hypertrophy; mediastinitis callosa, pericarditis effusiva and pulmonary neoplasms. (*c*) *Intra-œsophageal* factors comprise the fewest causal agents, as foreign bodies, pedunculated polyps from the lower pharynx and oïdium albicans.

Apart from the special *pathology* of these causes, stenosis occurs chiefly (*a*) at the lower third of the œsophagus, (*b*) near the level of the bifurcation of the trachea or (*c*) high up in the œsophagus. The stenosis is more often single than multiple and more often annular than longitudinal; it varies from slight stenosis to complete atresia, in which the tube may be seen as an impervious cord. Above the stenosis the tube is dilated and its musculature is vicariously hypertrophied to force food through the narrowed point.

Symptoms.—Symptoms vary with the cause and its extent; thus mechanical *dysphagia* may be quite secondary to aortic aneurysm and may vary from time to time with the pressure exerted upon it; in the cicatrizing ulcers dysphagia is the only symptom. In advanced stenosis the food and later the fluids taken *regurgitate* after a short time if the stenosis is low, or at once if the stenosis lies just below the pharynx; as a rule, the subjective localization described by the patient is unreliable. The *regurgitated food is neutral,* macerated, fermented and often contains round cells, mucus or blood, but never HCl nor pepsin. Œsophageal *pain* is common, but its occurrence depends on the causal lesion. *Singultus* is rather frequent when the lesion lies near the diaphragm. Dyspnœa may result from pressure upon the air passages by the dilated portion of the œsophagus above the stricture.

Physical Examination and Diagnosis.—The chest should be examined for aneurysm, retrosternal struma and lung tumor and the finger should be introduced as far as possible down the throat, to exclude the possibility of lesions situated high in the œsophagus or larynx. Illumination of the œsophagus (*œsophagoscopy*) may give excellent results if done by an expert, but its general application is very limited. Local signs are elicited chiefly in three ways—by sounding, by auscultation and by the *x*-rays.

1. Sounding.—A soft sound is far less dangerous than a hard one, at least for the first examination and does the least damage in cases of acute œsophagitis, aneurysmal compression and ulcerating cancer. It is not sufficient to pass one stricture, as there may be others lower down, whence the sound should be passed into the stomach. The length of the œsophagus is 10 inches or 25 cm.; from the upper incisor teeth to the œsophagus 6 inches or 15 cm.; from the teeth to the cardia 16 inches or 40 cm. Recently it is maintained that the last measurement should be 19.7 inches or 50 cm. The length of the cervical portion is 2 inches or 5 cm., of the thoracic $6\frac{3}{4}$ inches or 17 cm., and of the abdominal part $1\frac{1}{4}$ inches or 3 cm. From the teeth to the level of the tracheal bifurcation is 9 inches or 23 cm.

2. Auscultation.—Deglutition is marked, physiologically, by two murmurs, heard with the stethoscope six to eight seconds after the be-

ginning of the act, first a short murmur and then a longer one. In cases of marked stenosis the swallowing sound is heard at a point just above the lesion; this is a valuable help when aneurysm is suspected and sounding is feared. In the cervical part of the œsophagus the noise results chiefly from the movements of the pharynx; in the thoracic segment the stenotic murmur is heard to the left of the spine from the first to ninth dorsal vertebra. Stenosis near the tracheal bifurcation often gives a delayed first murmur, and a gurgling, spurting character in both murmurs; in the lowest segment the murmurs are heard just above the cardia, at the ninth or tenth dorsal vertebra.

3. X-RAYS.—The *x*-rays may show the outline of the dilatation above the stricture (*v. i.* DILATATION OF ŒSOPHAGUS).

Prognosis.—The prognosis depends on the cause. Cancerous stricture may ulcerate, thus relieving the local symptoms, though the cachexia increases. Strictures resulting from caustics often improve. In cases where the cause cannot be relieved, the same symptoms result as in œsophageal cancer (*q. v.*). Perforation into the lungs, pleuræ or air passages, or into the mediastinum or vessels, may follow. Aspiration pneumonia, which is often gangrenous, is not infrequent.

Treatment.—The remediable etiological factors are few, except that iodides may be given in syphilis or aneurysm. Foreign bodies can sometimes be removed by long forceps, with the direction of the œsophagoscope. In cancerous and aneurysmal strictures, probing and dilatation are dangerous. Dilatation by sounds is attended by the risk of perforating the œsophagus. Sounds should be introduced beside the index finger, with the patient's head thrown slightly backward; severe gagging may be overcome by painting the pharynx with cocaine, though it is often as much psychical as œsophageal, and assurance is a better preventive than drugs. The subject should first swallow some olive oil to facilitate passage of the sound. In passing the sound it should be held like a pen and pushed with exceeding gentleness. Trousseau's tapering ivory olive points are excellent, but care is necessary, and successively larger sizes should be used. Sounds may be left in the œsophagus for some minutes or an hour, or even permanently, but pressure necrosis may then result. It is at times necessary to introduce filiform bougies, such as are used in passing narrow urethral strictures. Silver balls (2 to 7 mm. in diameter) with threads attached, may be swallowed; they should be carefully pulled out after remaining in over night. When the obstruction is high, external rather than internal œsophagotomy is advisable. As first practiced by von Bergmann, after opening the stomach, dilatation may be employed from below. Impermeable strictures call for gastrostomy and rectal alimentation. (See TREATMENT OF INTESTINAL OBSTRUCTION, thiosinamin.)

DILATATION. DIVERTICULUM.

Etiology.—*Dilatation involves the entire circumference; diverticulum involves but part of it.* Secondary *dilatation* may occur above stenosis of the œsophagus or above the cardiac end of the stomach. It seldom extends more than an inch and a half (4 cm.) above the stricture; it rarely follows pyloric stenosis. Primary (idiopathic) dilatation may be local or total. Marked dilatation above the cardia, Luschka's so-called "fore-stomach" is an example of the local form, which is usually congenital. The diffuse idiopathic dilatation results without organic stenosis and is seen chiefly between the years of twenty and forty. Its actual cause is disputed. Most clinicians incline to the view advanced in 1888 by Meltzer, of New York, that its cause is a *cardiospasm* due to abolition of cerebral inhibition on the cardia; Kraus reports a case with great atrophy of both vagi; Rosenheim maintains that atony and ectasia are primary and cardiospasm secondary. *Cardiospasm* produces a great hypertrophy of the cardiac muscle, even six times its normal thickness, and diffuse total dilatation so that the œsophagus may measure 18 inches (46 cm.) instead of 10 inches (25 cm.) in length or 12 inches (30 cm.) in circumference (Luschka). Though Zenker collected but 18 cases in 1878, the number is increasing; nearly a dozen were reported in 1904 (Sippy, Tyson, Kinnicutt and Sossen), and up to 1905 over 90 are recorded. The mucosa is catarrhal, thickened or eroded, and the muscularis is hypertrophied or fatty.

Symptoms.—The symptoms are gradual or sudden in onset. There is *dysphagia,* which may often be overcome by effort; the patient feels that the food does not reach the stomach. *Regurgitation* of the food, especially the portion last swallowed, usually occurs soon after eating or when the patient lies down; it may measure a quart, is neutral, decomposed if retained for some time, and contains lactic and butyric acids but no hydrochloric acid, pepsin or rennet. The regurgitated food may again be swallowed (rumination). Vomiting is impossible. The pressure of the stagnant food or fluid in the gullet may produce vagus symptoms, such as dyspnœa or palpitation. The *sound* moves freely from side to side, but meets temporary resistance at the cardia, though pressure usually overcomes the obstructing cardiospasm. One sound, introduced into the stomach, may recover HCl, pepsin, etc., but another, introduced *to* but not *beyond* the cardia, recovers fluid or food with a neutral reaction; methylene blue solution poured through the first tube returns blue; that recovered by the second tube is colorless. Distention of the œsophagus with carbon dioxide gives *tympany,* or with water *dulness,* to the left of the sixth to the ninth dorsal vertebræ, sometimes above the left costal arch, or even in the epigastrium. The œsophagoscope may reveal the dilatation; a distinct shadow may often, though not always, be seen by using the *x*-rays after the patient has eaten potatoes or bread mixed with 20 per cent. bismuth. The *second swallowing bruit* may be absent or

altered or sometimes sounds like fluid running into fluid. Marasmus, constipation and often death result, but the clinical course may cover years or decades.

Treatment.—Treatment is rather unsatisfactory. Olive oil ℥ss before meals may help the fluid (or the sound) to pass the cardia. Continued use of the stomach tube may be effective, but in some instances causes œsophagitis; its life-long use is often required. Lockwood's pneumatic bag may be introduced empty on a sound into the stomach, then inflated and withdrawn slowly and gently through the cardia. Gastrostomy and rectal feeding may be indicated. Operation should not be delayed too long. Mikulicz operated six times—with five cures—by entering the stomach, forcibly distending the cardia and rupturing its fibers submucously.

Diverticulum.—This involves only an arc of the œsophageal circumference. Two types are distinguished: 1. *Zenker's pressure diverticulum* (pulsion-diverticulum) of which Rosenthal (1902) collected 180 cases. It is found chiefly in men (77 per cent.), and is a pouch which forms very gradually on the posterior œsophageal wall, usually near the junction of the pharynx and œsophagus, and nearly always consists of only the mucosa and submucosa,—a species of bottle-shaped œsophageal hernia. Its onset is very gradual, for the muscle, probably congenitally weak, yields before intra-œsophageal pressure, trauma, foreign bodies and the stagnating food. The early *symptoms* are signs of pharyngo-œsophagitis, hawking and dysphagia, and food entering the sac rather than the œsophagus; the axis of the sac, which dips between the gullet and the spine, is roughly parallel with that of the œsophagus; many of its symptoms are those of a high œsophageal stenosis. The food stagnates and regurgitates with a fœtid odor. In 33 per cent. of cases a tumor appears in the supraclavicular region, which is dull when filled with food or tympanitic when distended with carbon dioxide; it sometimes can be smoothed out, whereon the food regurgitates. The murmurs on swallowing are protracted (in 50 per cent.). A sound usually enters the sac, and another, if slightly bent, enters the gullet; this also differentiates a diverticulum lower in the gullet, which is even less common. Examination by the œsophagoscope is generally negative, but the *x*-rays may show a characteristic shadow. There may be pressure on the heart, trachea, vagus, recurrent laryngeal nerve and brachial plexus. The clinical course is protracted, even to forty-nine years, and the spontaneous evolution is inanition in over 50 per cent. of the cases, gangrene (15 per cent.) or perforation with cervical phlegmon. *Treatment* includes feeding by means of the sound, rectal nutrition, gastrostomy and surgical resection; this was first performed by von Bergmann (1890), 24 cases being recorded with 20 per cent. mortality. Diverticula low in the œsophagus can only be treated by long-continued sounding.

2. *Rokitansky's traction-diverticulum* is frequent; according to Schmorl it is found in 3.5 per cent. of autopsies. It is due to extra-

œsophageal traction, which draws a small part of the gullet out into a funnel-like form. Over 80 per cent. are due to indurative periadenitis around caseous or anthracotic lymph glands. Cicatricial mediastinitis from pleuritis, pericarditis or vertebral disease may produce the same effects. It usually, therefore, occurs at the level of the tracheal bifurcation. Diverticula may result from both traction and pressure. Traction-diverticulum begins early in life, is usually single, is less marked on the anterior or lateral wall, barely exceeds five to ten mm. in depth and is rarely discovered during life, though some have been seen with the œsophagoscope. Its chief danger is ulceration at its apex and perforation into the trachea, bronchi, vessels, pericardium, pleuræ or mediastinum, with resulting gangrene of the lungs, fatal hemorrhage, pneumothorax or phlegmonous mediastinitis. There is no treatment.

CANCER OF THE ŒSOPHAGUS.

Etiology.—Its frequency is rated at $\frac{1}{4}$ to 1 per cent. of autopsies, and it is said by Petersen-Borstell to rank third among cancers (stomach 33 per cent., uterus 15 per cent. and œsophagus 6 per cent.). Its etiology is unknown. (*a*) Scars, trauma, ulceration and traction diverticulum are promoting causes; (*b*) 75 per cent. occur in males and 80 per cent. between the fortieth and sixtieth years; 8 per cent. occur before the fortieth year; Stewart records a case aged twenty-three and Heimann one aged nineteen.

Pathology.—Cancer of the œsophagus is almost always single and primary, though cancer of the stomach, pharynx or mediastinum may rarely involve the œsophagus secondarily. Most cases are squamous *epithelioma,* arising from its pavement epithelium; scirrhus, colloid and medullary forms are rare. *Localization;* most cases (57 per cent.) develop in the lowest third of the œsophagus; many (33 per cent.) in the middle, and the least (10 per cent.) in the upper third; its favorite sites are near the cardia, at the tracheal bifurcation level and just back of the cricoid cartilage. The muscularis sometimes hypertrophies above the cancer, where some dilatation may occur.

Symptoms.—The onset is usually insidious. Signs of *œsophagus stenosis* (*v. s.*) develop in varying grades; stenosis generally advances slowly but is occasionally sudden. It may lessen as the cancer ulcerates. Its symptoms are those of *stenosis,* as *dysphagia,* rather indefinitely located pain, regurgitation of food and perhaps also of bloody mucus or necrotic cancer tissue. Its signs are also identical, as obstruction on sounding, alteration of the murmurs, sometimes the detection by the œsophagoscope of a papillomatous or ulcerated neoplasm, and quite rarely tumor particles found in the fenestræ of the tube. *Cachexia* develops; constipation, indicanuria and peptonuria are usual; acetonuria is frequent. Extension, by contiguity, occurs to the spine, mediastinum and cardia, or in 60 per cent. of cases by *metastasis;* the mediastinal and peribronchial lymph nodes are usually invaded, the supraclavicular often and the upper cervical seldom.

Complications.—There may be pressure on the recurrent laryngeal (14 per cent.), vagus, mediastinal veins and arteries, on the sympathetic ganglia (13 per cent.) or brachial plexus. *Perforation* occurs in over 50 per cent., most often into the air passages, when aspiration pneumonia or gangrene may follow; into the lung, pleura (ichorous pleurisy or pneumothorax resulting); into the aorta (fatal hemorrhage), heart, pericardium and mediastinum. Pyæmia, pulmonary tuberculosis and marantic thrombosis, occur in some cases. *Death* is inevitable, usually within thirteen months, with emaciation, subnormal temperature, small slow pulse, slow shallow breathing, flattened abdomen and perhaps intoxication coma and fever.

Diagnosis.—The diagnosis is based (*a*) on a stricture of the œsophagus, which in 90 per cent. of all cases is carcinomatous; (*b*) on evidences of neoplasm, as age, cachexia, cancerous nodes, metastases and perhaps tumor particles on the sound. (*c*) Other tumors, as lipoma, myxoma, myoma (11 cases, Bryant, 1906), fibroma or sarcoma are of greater pathological than clinical importance, except accessible fibropapillomata of the upper gullet or lower pharynx.

Treatment.—The treatment is that of stricture or stenosis; nutritive enemata, feeding by the stomach tube, gastrostomy and for pain, narcotics are indicated. Œsophagectomy is seldom advisable, as less than 10 per cent. occur in the cervical portion, accessible to surgical interference. As to gastrostomy, Schmidt in 1897 reviewed 236 cases, in which 45 per cent. did not live longer than a week and nearly 90 per cent. not more than one hundred days.

PERFORATION; RUPTURE; HEMORRHAGE OF THE ŒSOPHAGUS.

1. *Perforation* may result from causes within or without the œsophagus. Internal causes include ulcers, foreign bodies, diverticula, cancer and use of the sound; external causes are aneurysm, gravitating abscess, lung cavities or gangrene, suppurating lymph glands or trauma. It is marked clinically by mediastinitis and cervical cellulitis, which are usually phlegmonous. Œsophageal fistulæ may communicate with the air passages, from which by the laryngeal mirror fluid or food is seen to be expectorated; a communicating tuberculous cavity may become flat when the patient drinks or eats, and the flatness changes to tympany when the food or drink is expectorated; this is tested by giving the patient milk, which will be brought up from the larynx. Aspiration gangrene, pneumopericardium, pneumothorax and fatal hemorrhage are common causes of death.

2. *Spontaneous rupture* (so-called) is a very rare affection, there being only 39 cases on record (Cohn, 1908). It is often confused with œsophagomalacia, in which the rupture is not longitudinal or clean-cut as in genuine rupture. The first case, and one of the clearest, was reported by Boerhaave (1714); in all cases, except Boerhaave's, the rupture was longitudinal. It follows over-eating or severe vomiting, and most victims have been alcoholics. There is

great and sudden pain over the lower œsophagus (where most ruptures occur) and over the xiphoid, severe vomiting, hæmatemesis, collapse and subcutaneous emphysema. Death is the rule from collapse, phlegmonous or ichorous mediastinitis, pleuritis or pneumothorax. Treatment is wholly expectant.

3. *Hemorrhage* may follow œsophageal ulceration, external or internal trauma, toxic œsophagitis, softening and aneurysmal or other perforations. Varicose veins in the lower œsophagus, first described by Le Derberden (1837), are most often complications of liver cirrhosis, less often of syphilis, senile atrophy and pylethrombosis. The diagnosis and localization of the bleeding point is at best only suggested by the history and previous physical findings. *Treatment* consists chiefly of keeping the patient absolutely quiet and giving morphine and rectal feeding, as in gastric hemorrhage.

MOTOR AND SENSORY DISTURBANCES OF THE ŒSOPHAGUS.

These will be considered under AFFECTIONS OF THE VAGUS (*q. v.*, under NERVOUS DISEASES).

DISEASES OF THE STOMACH.

ACUTE GASTRITIS (ACUTE CATARRH, ACUTE DYSPEPSIA).

Acute gastritis is a better term than catarrh, which implies a superficial inflammation of the mucosa.

I. Simple Gastritis.—Etiology.—It is a very common primary and secondary affection. (*a*) Its most important *mechanical* cause is improper food or improper eating; *rich food,* an excessive amount, *partial mastication,* washing down the food with water, hurried eating when very hungry, idiosyncrasies to certain kinds of food, decomposed articles containing bacteria or ptomaines and excessive ingestion of cold water and fruit are accessory factors. (*b*) *Thermal* factors include too hot or too cold foods, especially when taken on an empty stomach. (*c*) *Chemical* causes; aside from acids and caustics (see TOXIC GASTRITIS), *alcohol* ranks foremost; autotoxins may excite acute gastritis, as in chronic nephritis, cholæmia or gout. (*d*) Some cases are clearly *infectious;* the condition may occur in variola, measles, typhoid, grippe and pneumonia, as a secondary manifestation; occasionally it is an independent febrile affection, as in the Christiana epidemic reported by Hausemann (1880), in which 6,000 persons were affected within three weeks. (*e*) Certain conditions are somewhat *predisposing* to acute gastritis; for example, stasis from diseases of the heart, lung or liver, tuberculosis, cancer, syphilis, chlorosis or affections of the higher alimentary mu-

cosa. In some cases a familial or even hereditary tendency is noted, known as "the delicate stomach" by the laity. Nervous factors, as anger or excitement, are sometimes apparently causative, though they may be confused with the digestive neuroses.

Symptoms.—1. Digestive Symptoms.—These are sometimes absent, as demonstrated by Beaumont in the celebrated St. Martin case of gastric fistula. The *appetite* is usually *lost* or perverted. The *tongue is thickly coated* with white fur and dry mucus. The *mouth is dry* and there is a disagreeable taste and great *thirst,* even though the salivary flow is often increased. Hiccough, *eructations,* a sense of gastric distention, pressure or *pain* and tenderness over the stomach, are common. Nausea and *vomiting* characterize the more marked cases; the vomitus is pale yellow, bitter and contains little or no hydrochloric acid, though lactic, butyric and fatty acids are common; there is much mucus, which is frequently flecked with blood. Gastric intolerance, pain, nausea and vomiting summarize the situation. Corresponding anatomical changes are found, though few uncomplicated cases come to autopsy; the mucosa, particularly near the pylorus, is red, swollen, smeared with adherent mucus, sometimes slightly eroded or dotted with punctate ecchymoses; the dilated racemous vessels, as seen by the gastroscope or through a gastric fistula, are less conspicuous at autopsy for the blood in great part leaves the organ after death; microscopically the main findings are leukocyte emigration, granular degeneration and swelling of the mucous and other glandular cells.

2. General and Other Symptoms.—Herpes is an occasional finding. *Constipation* is the rule, though diarrhœa or icterus sometimes occurs in gastro-enteritis. The *urine* is *scanty* because of the vomiting; it deposits a lateritious sediment and sometimes contains indican or acetone. Nervous symptoms—as frontal *headache,* vertigo, intercostal neuralgia and depression—are common, but convulsions (acetonæmia) and other marked nervous manifestations occur infrequently in young and sensitive individuals. Fever is quite uncommon; when present, it is usually irregular and ephemeral.

Diagnosis.—The *diagnosis* is not difficult when the course is short and afebrile, as is usually the case. With fever or some constitutional symptoms it is an excellent practice to regard acute gastritis as a symptom, until, at least, typhoid, exanthematous disease or nephritis can be excluded by the later clinical evolution. Confusion is possible with a tabetic crisis or with the vomiting of pregnancy and hysteria.

Treatment.—(*a*) *Prophylaxis* embraces the etiological factors. (*b*) *Complete rest of the stomach* for one or more days is the first active indication; no rectal feeding is required for twenty-four to forty-eight hours, by which time the gastric equilibrium is in great part restored; warm water may be given by rectum to unload the bowels and to allay thirst. (*c*) If *gastric irritation* persists, the stomach should be thoroughly washed out by the *stomach tube,* using warm water with

a teaspoonful of sodium bicarbonate to the quart. Gastric lavage is a better measure than the use of calomel and salines, which irritate the inflamed, perhaps eroded mucosa. For the same reason emetics (apomorphine hypodermics or ipecac by mouth) are avoided. *Epigastric fomentations* alleviate pain. Obstinate irritation and vomiting usually respond to Bartholow's prescription, modified as follows:

℞ Phenolis ..... gr. iv.
Ac. hydrocyanici dil. ..... ʒss.
Bismuthi subnitratis .....
Cerii oxalatis .....āā ʒiss.
Mucilaginis acaciæ .....q.s.
Aquæ menthæ piperitæ .....q.s. ad ʒij.

M. et fac emulsum. S.—One teaspoonful without dilution every half to one hour for six to ten doses.

Opiates give immediate relief, but secondary nausea is an insuperable obstacle to their use. (*d*) *In convalescence,* dilute hydrochloric acid ♏ v to xv and bitters are given after meals, to stimulate and supplement the gastric secretion. The food should be simple, moderate in bulk and thoroughly chewed.

II. Gastritis Toxica (Venenata).—Etiology and Pathology.—Acute toxic gastritis is caused by various alkalies, acids and metallic poisons. Sulphuric acid produces a gray eschar, nitric acid a yellow one, alkalies a brown, copper a greenish blue, silver a deep black eschar, and phosphorus produces an opaque milky stain. The pathological changes are usually severe, particularly in places with which the corrosives remain longest in contact, as the pylorus and posterior wall; these changes range from simple epithelial desquamation and catarrh, to hemorrhagic infiltration, diphtheroid patches, suppuration and ulceration.

Symptoms.—The symptoms are those of gastritis simplex, but more severe; pain is intense in the mouth, throat, œsophagus and stomach, and is not relieved by the repeated vomiting. The vomitus is often bloody, the epigastrium tender and the abdomen tympanitic.

Prognosis.—The *outcome* varies; (*a*) collapse after a few hours or days is frequent, with small pulse, shallow breathing, subnormal temperature, bloody albuminous urine and other symptoms which vary with the poison ingested. (*b*) The stomach symptoms may subside and death may occur later from blood or visceral changes. (*c*) Strictures may develop, or (*d*) there may be atrophy of the secretory glands (achylia gastrica).

III. Phlegmonous Gastritis.—Etiology.—Only 100 cases are recorded (1908). It is an acute suppurative inflammation of the stomach, usually caused by the streptococcus; it begins in the submucosa and infiltrates the other coats diffusely or forms a circumscribed mural abscess. It most often occurs secondarily (75 per cent.) in sepsis, the acute infections, anthrax or in local diseases as gastric cancer. The writer has seen only one case, which followed erysipelas. Eighty-five per cent. occur in adult males. Alcoholism and traumatism are predisposing factors.

Symptoms.—The symptoms are gastric and septic. Besides the symptoms of acute gastritis, the vomitus sometimes contains pus, the epigastric pain is intense, meteorism is well marked and there are many symptoms of peritonitis, which is a most common complication. In a few cases an epigastric tumor marks the circumscribed variety. Sepsis, usually in a severe form, causes the high fever, the pronounced status typhosus, the small pulse, occasional icterus and petechiæ.

Diagnosis and Treatment.—A *diagnosis* is almost impossible, the *outcome* is fatal in 96 per cent. during the second week and the *treatment* is symptomatic, *viz.*, rectal nutrition and stimulation, narcotics for pain and vomiting and hydrotherapy for fever. Some cases heal by rupture into the stomach with cicatrization and, if the affection is suspected, operation can be considered.

IV. Diphtheritic Gastritis.—This form, due to the Bacillus diphtheriæ, is very rare. Diphtheroid gastritis, caused by streptococcic and other infections, is more common; it may develop after gastritis toxica, smallpox, pneumonia and kindred maladies.

V. Parasitic Gastritis.—This form is, in rare instances, due to the oïdium albicans, favus parasite (Kundrat), anthrax bacillus, larvæ of insects and tineæ.

CHRONIC GASTRITIS.

Chronic gastritis (dyspepsia, catarrh) is characterized pathologically by degeneration of the parenchymatous and infiltration of the interstitial tissue, and clinically by mucus formation and decreased secretion.

Etiology.—(*a*) The factors are the same as in acute gastritis, from repeated attacks of which chronic inflammation may result. Abuse of drugs, especially cathartics, is an important factor. *Alcoholism* and improper eating are most important. *Eating hurriedly* or at irregular hours, inadequate mastication, washing down the food with much fluid, excessive use of coffee, tea, ice water, carbohydrates, pastries or fried foods are salient factors. Chewing and smoking tobacco may cause chronic gastritis; stomatitis, carious teeth and pyorrhœa alveolaris are important causes. Most cases occur in adult males. (*b*) It often accompanies other gastric diseases, as cancer or ectasia. (*c*) It is frequent in tuberculosis, the anæmias, stasis, nephritis, gout, diabetes, syphilis, amyloidosis and Addison's disease.

The acid form is usually caused by excesses in eating, drinking and smoking; the subacid form by the abuse of spirits.

Pathology.—The pathological changes are observed chiefly in the *pyloric* region. The vessels are dilated, imparting a reddish-brown color, which later changes to gray. The mucosa is swollen, lax and covered with tenacious mucus. Sometimes punctate hemorrhages or erosions are observed. In a few cases the mucosa is rough, even papillomatous (*gastritis polyposa, état mamelloné*). The muscularis is not essentially changed, as a rule, but is sometimes hypertrophied near the pylorus. Histologically, the glandular cells are granular and fatty, or show a mucoid degeneration which extends

from the surface toward the deeper layers; the parietal cells cannot be distinguished from the principal cells, the glands are often large, sacculated or cystic and the tubules show irregular ramifications; the glandular structures may completely atrophy (see Achylia). The interstitial tissues show leukocytic infiltration.

Symptoms.—The symptoms begin *insidiously.*

1. Oral Symptoms.—The *tongue* is generally *coated* with moist white fur, reddish at its tip and indented on its edges, which may also result from alcoholic or nicotine stomatitis. There is frequently a bitter, sour, *salty taste* in the mouth and sometimes a marked *fœtor ex ore.* Increase in the saliva results from the coincident stomatitis.

2. Gastric Symptoms.—(*a*) Gastric pain is less frequent than a sense of *distention, discomfort* or *pressure after eating.* Soup and fluids, unless they are cold, generally cause no distress; *coarse or hard foods,* as beefsteak, hard bread, smoked meat, cabbage, hard-boiled eggs or cheese, cause distress. In nervous dyspepsia, any kind of food, hard or soft, may cause discomfort. In some cases of gastritis there is distress when the stomach is empty. Tenderness is much less than in cancer or ulcer and responds readily to treatment. (*b*) *Belching* of gas is common; eructations may be odorless or may contain fatty acids and even inflammable gas. Eructations of hydrochloric acid are rare. (*c*) *Nausea* is more frequent than vomiting, which may result from other conditions, notably chronic pharyngitis. The vomitus shows the same findings as the test meal (see Gastric Chemism). In alcoholics morning vomiting is common (*vomitus matutinus potatorum*), chiefly of saliva and mucus swallowed during the night.

3. Gastric Chemism.—Our intimate knowledge of the chemical changes in the stomach dates from the introduction of the stomach tube by Leube and the vast amount of work done by many workers, especially Riegel, Ewald and Boas. After washing out the stomach the Ewald test breakfast is given, consisting of a piece of dry bread and a cup of weak tea without sugar or cream. In an hour the stomach tube is again introduced and the gastric contents withdrawn by expression (the patient compressing the abdomen and straining), or by Ewald's aspiration bag. Innumerable *errors result from confidence in a single examination* and assigning undue importance to the gastric chemism alone. It is best to examine the contents chemically without filtration.

(*a*) The *gross examination* of the stomach contents shows coarse particles of food when the hydrochloric acid is absent or greatly reduced, or fine subdivision when it is present in fair amounts. A rancid odor denotes fatty acids, and a foaming appearance is caused by fermentation. *Mucus* is seen in practically all cases, and most clinicians do not recognize a simple gastritis without mucus formation, though Ewald distinguishes (i) simple gastritis in which the acid and ferments are decreased; (ii) a mucous gastritis, in which the acid is greatly decreased, the ferments are present and there is much mucus; and (iii) an atrophic gastritis which will be considered

separately. Mucus usually indicates a reduction in the hydrochloric acid. Normally there is little gastric mucus, though there is rather more with an amylaceous than with a proteid diet. Mucus of gastric origin is intimately mixed with the food and is not yellow, nummular, pigmented or mixed with air or cells, as in mucus from the mouth or respiratory tract.

(*b*) The *HCl is reduced.* It is recognized by the Günzburg test (2 gm. phloroglucin and 1 gm. vanillin dissolved in 30 c.c. of absolute alcohol); a few drops of the solution are added to an equal amount of the filtered gastric contents and slightly heated, without boiling, in a porcelain dish, when a carmine-red color develops, indicating free hydrochloric acid even in dilution of 1 to 20,000 parts, and not reacting to lactic or butyric acid; the solution should be kept in a dark bottle, for it degenerates in the sunlight. The solution should not be kept over two months. Boas recommends the following test, which gives the same reaction and is less expensive: resorcin 0.5, sugar 0.3, dilute alcohol q.s. ad 10 gm. HCl exists in a combined form or as free acid, which latter probably serves as an antiseptic.

The *total free HCl.* To 10 c.c. of gastric contents is added a few drops of the indicator (0.5 per cent. solution of dimethylamidoazobenzol in strong alcohol); it is placed in a beaker and, if free HCl is present, the yellow color imparted by the indicator will change to bright red; a decinormal solution of caustic soda (4 gm. to 1,000 c.c. of distilled water) is then added drop by drop to the gastric contents, until the color of the indicator has been changed from red to bright yellow; the number of c.c. of decinormal solution used is multiplied by 0.00365 and again by 10 to give the amount of free HCl. Very often the result is expressed in terms of the number of c.c. of decinormal solution used; *i. e.,* if 4 c.c. were employed, $4 \times 10 = 40$, the free HCl. The usual amount is 30 to 40.

The *total acidity* (HCl, lactic and other organic acids, acid phosphates and albumins, albumoses and peptones) is estimated by adding two drops of a 1 per cent. alcoholic solution of phenolphthalein (as an indicator) to 10 c.c. of the gastric contents; to this is added, drop by drop, a decinormal solution of caustic soda (4 gm. to 1,000 c.c. of distilled water) until the red color, due to the alkali acting upon the phenolphthalein, is no longer even deepened in hue by the alkali. As 4 to 8 c.c. of the decinormal solution are usually required, $4 \text{ to } 8 \times 10 =$ normal total acidity; *i. e.,* 40 to 80 expressed in terms of the decinormal solution (corresponding to 0.15 to 0.25 per cent. HCl). In most cases determination of the total acidity is sufficient. A total acidity under 20 gives no reaction with Congo paper; *i. e.,* there is no free HCl. The bread of the test breakfast (phosphates chiefly) accounts for a total acidity of from 5 to 8; this indicates that the gastric glands are atrophied.

In chronic gastritis the *hydrochloric acid* is *found decreased* or absent. A few cases, described by Riegel and Boas, show a total acidity of 40 to 50; this form is called gastritis acida, though its

separation as a type is scarcely justifiable; these are usually light, incipient or neurotic cases.

(*c*) *Rennet and pepsin* are rarely absent, though usually *decreased.* Pepsin is tested by mixing the gastric juice with egg albumin; 25 c.c. of normal gastric juice will dissolve 0.05 gm. of serum albumin in one hour, the same amount of fibrin in one and one-half hours and of egg albumin in three hours. Pepsinogen is tested in the same way with the addition of a few drops of hydrochloric acid, which liberates the enzyme (pepsin) from the pro-enzyme (pepsinogen). Boas insisted on the importance of the rennet ferment, which coagulates milk; if no rennet is present the filtrate will not coagulate milk.

Lactic acid is seldom found. (See CANCER OF STOMACH.) Leukocytes and epithelial cells are found at times, and in rare cases shreds of gastric mucosa (gastritis exfoliativa, Parisier).

4. OTHER SYMPTOMS.—(*a*) *Vertigo e stomacho læso* (Trousseau), gastric vertigo, is probably neurasthenic rather than gastric. The so-called dyspeptic asthma is not really asthma, but a dyspeptic dyspnœa and probably results from pressure of the distended stomach on the heart. Headache, physical and mental lassitude, a degree of melancholia, intercostal neuralgia, tender points between the shoulders or over the lower cervical spines and palpitation are fairly common. (*b*) The bowels are generally *constipated;* diarrhœa and tympany at times result from marked gastric fermentation or extension of the catarrh to the gut, when catarrhal icterus may result. Boas and Schmidt find undigested connective tissue in the movements. (*c*) The *urine* is frequently reduced and shows a lateritious deposit, phosphates and oxalates. (*d*) The general nutrition is usually well maintained if the intestines functionate well. (*e*) The motive and absorptive power of the stomach may be impaired, normal or even exaggerated, the food sometimes being hurried into the intestine.

Diagnosis.—Leube insists that chronic gastritis should never be diagnosticated until cancer, ulcer and functional disease have been finally excluded. Its cardinal points are (*a*) its steady, slow, chronic course; (*b*) the presence of mucus and (*c*) decreased secretion. (For its differentiation *v. i.* ULCER.)

Treatment.—1. PROPHYLAXIS.—Recurrence of the trouble is prevented by treatment of the causal conditions; digitalis is indicated in gastric stasis of cardiac origin and iron in anæmia.

2. HYGIENE.—Hygiene concerns regularity in living, moderation, recreation, massage, rational exercise and avoidance of haste in eating.

3. DIET.—F. A. Hoffman justly remarks that every one "has the stomach he deserves." Slowness of eating, drinking only after the solid part of the meal is eaten and thorough mastication are frequently curative without medicinal treatment. The importance of thorough insalivation of the food is manifest when we remember that the ptyalin operates for nearly an hour in the unchurned contents of the cardiac end of the stomach. As Ewald states, "die Diät der Magen-

Kranker fängt im Munde an." The *teeth* should receive care, as the treatment of pyorrhœa, extraction of carious teeth and fitting of proper plates. Food should be taken in moderate amounts, and the stomach should be given sufficient rest. Pawlow has shown that the relish of food excites the flow of the gastric juice; he names it the "appetite juice."

Kind of Food.—The term "easily digestible food" is extremely relative, but the kind of food is less important than careful mastication and insalivation. In severe or alcoholic cases an absolute milk diet may be tried, giving equal parts of milk and Apollinaris water, to which grains x of sodium bicarbonate, grains x of sodium chloride and grains v of magnesium oxide are added. Predigested milk soon becomes distasteful to the patient and is indicated chiefly in severe catarrh with much mucus formation or with atrophy. Generally speaking, the diet should be mixed, but proteids are superior to carbohydrates, which are prone to ferment. The *proteids* given are chopped lean meats, scraped beef, boiled mutton, veal, roasted steak, broiled tenderloin, pigeon, chicken and eggs boiled one minute. Chittenden has shown that we eat twice as much meat as is necessary. Beef tea, heavy fresh beef, hard-boiled eggs, pork, game, fish, warmed-over (therefore hardened) meat, turkey, goose, duck, smoked or cured meat (except bacon) and cheese must be interdicted. *Carbohydrates* are withheld when there is obstinate fermentation. Those allowed are arrowroot, well cooked sago, tapioca or rice, white bread, zwieback and toast; those occasionally allowed or tried later are mealy potatoes, spinach, pea or bean soups; those forbidden are substances containing much cellulose, as hard or brown bread, oatmeal, coarse vegetables, uncooked green vegetables, most fruits, pies and pastries. It is interesting to note that most of the carbohydrates ingested reach the bowel very rapidly. Carbohydrates, therefore, should be eaten before the proteids, that they may the earlier leave the stomach. *Fats* are a necessary part of the diet, especially moderate amounts of butter and cream. Other forms of fat as greasy soups are injurious.

Accessories.—No seasoning is necessary, but liberal quantities of salt may be used. Liebreich insists that condiments in moderate amounts are helpful. Tea, coffee and alcoholic beverages are usually prohibited. Smoking should be interdicted or restricted.

4. Gastric Lavage.—Lavage is indicated when there is formation of mucus, fermentation, deficient secretion and delayed digestion. It is generally considered the most helpful measure but in the writer's experience is seldom indicated as there is no stagnation of food. Morning lavage is best for removal of mucus, and lavage before the evening meal for delayed digestion or fermentation. Warm water is used, to which sodium bicarbonate, two teaspoonfuls to the quart, is added to dissolve the mucus, or salicylic acid (1 to 1,000) to modify fermentation. The stomach should be washed until the water returns clear. When the use of the stomach tube is resisted or contra-indicated, as by exceptional nervousness, modified lavage may be em-

ployed, the patient drinking before breakfast two glasses of warm water to which a teaspoonful each of sodium chloride and sodium bicarbonate has been added. This process is essentially the same as drinking the Kissingen, Wiesbaden and other waters. The sodium bicarbonate acts locally and the sodium chloride acts both locally and systemically, for small doses increase the hydrochloric acid and large doses reduce it by increasing the alkalinity of the blood. Carbonated waters may be used, which stimulate secretion and absorption and neutralize abnormal acids.

5. Medical Treatment.—(*a*) The value of hydrochloric acid is disputed. Its first indication is to convert pepsinogen into pepsin, whence it is useless in atrophic gastritis save as it stimulates the flow of bile. Its second indication is as a digestant; we employ 20 drops of the dilute acid every thirty minutes until ℥j to ij have been given. Its third indication is as an antizymotic, which Boas holds is its sole indication; it should not be given until about half an hour after meals, during which period the saliva is acting upon the carbohydrates; Pawlow has shown that it increases the flow of pancreatic juice and perhaps of bile. (*b*) Boas takes the extreme view that *pepsin* is useless. It should be given as a powder, for solutions rapidly deteriorate and are usually strongly alcoholic; many forms offered on the market are inert. Pancreatin is very frequently ineffective. Taka-diastase is beneficial in some cases. (*c*) *Bitters* are best exhibited before meals; though their mode of action is disputed, they surely act upon the appetite or on the nervous structures and increase the flow of saliva and gastric juice and probably also the pancreatic juice. Strychnine or nux vomica is the best of this group of drugs and is especially valuable in alcoholic cases. All bitters should be given in solution, as their bitter taste is their chief mode of action.

℞ Strychninæ sulphatis ..... gr. j.
Acidi nitrohydrochlorici ..... ℥j.
Tr. gentianæ comp. ..... ℥iv.
M. et S.—One teaspoonful half an hour after meals in half a glass of water.

Condurango bark (fluidextract ℥ss) is prepared by macerating the bark in cold water. Some bitters, as gentian, may irritate the stomach and induce diarrhœa. (*d*) *Antizymotics* (antifermentatives), as creosote or betanaphthol, often cause irritation. Phenol gr. j, thymol gr. ss to j, resorcinol gr. ij to v, after meals, act on the stomach and salol gr. x and betanaphthol gr. x (in capsule because of its bitterness) are intestinal antiseptics. (*e*) *Gastric sedatives* are seldom indicated except in the hyperacid type. Bartholow's combination (see page 578) meets all indications. Silver nitrate is sedative, astringent and alterative, but may cause nephritis or argyria; it is given in pill form, half an hour before meals, in doses of one-fourth of a grain (perhaps with quite small doses of opium or belladonna). Zinc oxide, gr. iij to v, given before meals, is valuable in alcoholic gastritis. Extract of belladonna in one quarter of a grain doses is help-

ful in pain, especially in hyperacid gastritis. Pyrosis and spasm may be considered under this heading. In pyrosis of fatty and other organic acids, alkalies may be employed, though sparingly; Trousseau gave sodium bicarbonate grains v, creta preparata grains x and magnesium carbonate grains v after meals. Spasm from gas or overloading of the stomach is modified by spirits of chloroform ♏xx and tincture of asafœtida 3j; spasm suggests erosions near the pylorus, when extract of belladonna gr. ¼ and subnitrate of bismuth gr. x are distinctly indicated, after meals. (*f*) *Other measures,* as faradization, massage and hydrotherapy, are less valuable than in gastric atony and sensory neuroses of the stomach. In constipation cathartics should be avoided, as they cause irritation and constipation recurs. Hot water, gastric lavage, colonic flushings and perhaps the mildest saline aperients are indicated.

Achylia Gastrica.—Etiology.—The term was introduced by Einhorn in 1892 to designate total suppression of gastric digestion. Atrophy of the stomach was first described by Austin Flint (1860) and by Fenwick (1877), Ewald, Quincke and Einhorn. Achylia frequently occurs secondarily in cancer of the stomach and in toxic gastritis, but also in other gastritides, cancer of distant organs (mammæ, uterus, intestine, œsophagus), diabetes, pernicious anæmia, tabes and in neuroses. In rare cases it develops as a seemingly primary affection, possibly as a congenital condition (Martius). It occurs oftenest in middle or advanced life, though isolated cases are reported at eighteen to twenty-five years of age.

Pathology.—Pathologically two main forms exist, (*a*) one, *cirrhosis ventriculi,* in which the stomach is very small and thickened so that its walls may measure several centimeters in thickness, and (*b*) another, *phthisis ventriculi,* in which the walls are thin or "wasted" and the stomach is lax or dilated. In some cases atrophy has been found in Meissner's and Auerbach's plexuses. The glandular structures are wasted (anadenia). In neurotic achylia there is no anatomical change.

Symptoms.—Symptoms may be absent if gastric motility is good and the intestinal glands are normal. If the same process exists in the gut diarrhœa, marasmus and death result.

1. Gastric Symptoms—There are no pathognomonic gastric symptoms. Pain is variable; it may be absent or moderate, though in some cases lancinating pain and other symptoms like those of hyperchlorhydria may exist (Einhorn). The appetite is also variable. Vomiting does not always occur but when present the vomitus always contains coarse, poorly digested particles.

2. Test Meal.—The recovered food is coarse; the total acidity corresponds to the amount in the food ingested. No HCl is found (achlorhydria), no pepsin (therefore no peptones), no rennet (therefore no coagulation of milk), no blood and according to most writers no mucus; according to Riegel and Schmidt, some mucus may be

present, due to metaplasia of the gastric mucosa to one resembling that of the intestine, the cells of which secrete mucus; as they in turn atrophy the mucus disappears. Lactic acid is found but rarely. The gastric *motor power* is usually *increased* and the stomach is emptied in less than an hour. In the contracted cirrhotic form, the stomach may hold but a few ounces; in the phthisic form ectasia may occur. In some scleroses of the stomach a sound is introduced into the stomach with difficulty, hydrochloric acid is present, the stomach cannot be distended with gas and its capacity is enormously decreased.

3. Other Symptoms.—Headache and vertigo are common. Constipation is the rule; in some cases diarrhœa may be the sole salient symptom, so that it apparently indicates an intestinal lesion (Oppler). Symptoms like those of pernicious anæmia (Henry and Osler) or like gastric cancer (author) mark some cases. The increased acidity of the urine, at the height of digestion, is naturally absent. The test meal establishes a clear diagnosis.

Prolonged observation may be necessary in order to exclude the secondary atrophic gastritis of cancer and amyloid gastric disease. In one instance the autopsy showed only atrophic gastritis, whereas clinically hæmatemesis, achylia, lactic acid, œdema, ascites and extreme emaciation had led to a suspicion of cancer.

Prognosis.—Life may be prolonged for years by compensating intestinal digestion, but recovery is impossible after atrophy develops.

Treatment.—Treatment is necessarily palliative. Thorough mastication of food is imperative. Cellulose is to be avoided and thick, plain, leguminous purées should be given. Butter is well tolerated. The meals should be small and frequent (every three or four hours). HCl should be given in full doses (ɱxx to xxx), Pawlow having demonstrated its stimulating action on the pancreatic flow and its control of the pylorus (thus averting diarrhœa). Water relieves the pain, which resembles that of hyperchlorhydria (Einhorn).

DILATATION OF THE STOMACH; MOTOR INSUFFICIENCY.

The nomenclature of this and allied conditions is a matter of dispute; dilatation of the stomach (gastrectasia) is only a symptom, and is understood as a more or less lasting dilatation with motor insufficiency.

I. Acute Dilatation.—Acute dilatation was first fully described by Fagge (1883); 217 cases of acute dilatation are recorded (Laffer, 1908).

Etiology.—(1) Most cases follow abdominal trauma or laparotomy (36 per cent.); gall-bladder operations are the most frequent type. The two cases seen by the writer were in women over fifty years of age who had been operated on for fibroids of the uterus. (2) It may follow acute infections as pneumonia or typhoid; or (3) overloading of the stomach; in one of Kolisko's autopsies in Vienna the author

saw an enormously dilated stomach in a man who had, on a wager, taken a large number of charged siphons and died soon afterward from gastric distention and reflex paralysis of the heart. (4) Most cases occur between twenty and thirty years of age. (5) Traction or obstruction of the superior mesenteric artery in the duodenal mesentery is the most favored etiological factor, though no proof exists that it is not a result rather than cause.

Symptoms.—The stomach is enormously distended; there is vomiting of brown-gray to black material which often runs out of the mouth, may be very copious and sometimes is offensive. There is usually no free HCl; sometimes there is lactic acid; succussion sounds are fairly frequent. The abdomen is greatly distended. There is much collapse and often abdominal pain.

Prognosis and Treatment.—Sixty-four per cent. die, usually within five days. The indications are: early gastric lavage; rectal feeding; strychnine in large doses and a few doses of atropine (under which the writer observed one recovery); salt solution under the skin, and other measures for shock; and in appropriate cases gastrostomy or gastro-enterostomy.

II. Chronic (Permanent) Dilatation and Motor Insufficiency.—In the following discussion it must be borne in mind that various grades of distention and atony may occur, that dilatation may be associated with increased power of the gastric musculature or with atony, and that motor insufficiency (inability of the stomach to propel food into the intestine) is not necessarily, though very often, associated with dilatation of the stomach. Dilatation and motor insufficiency are most prevalent in middle or advanced life.

Etiology.—1. Pyloric Stenosis.—Its causes are twofold, *malignant and benign.* It is most often *cancerous,* but may follow round or corrosive ulcers, chronic gastritis leading to hypertrophy of the pyloric musculature (stenosing gastritis), benign tumors of the stomach, as pedunculated polyps occluding the pylorus, and congenital stenosis, of which Shaw and Elting (1905) collected 145 cases; under one year of age 45 cases of congenital hypertrophic stenosis are reported, most of which were less than four months old. It may also result from congenital duplicature of the pyloric mucosa, volvulus of the stomach (of which 10 cases are reported), angular bending of the fixed pylorus by sinking of the stomach (gastroptosis) or from adhesions due to cholecystitis. Ricard and Chevrier collected nineteen cases of tuberculous pyloric obstruction. Stenosis of the duodenum produces essentially the same results as pyloric stenosis and may follow duodenal ulcer, diverticulum or twisting. The pylorus may suffer compression stenosis from tumors of the liver, colon, kidney or pancreas. Payr in 1904 collected 58 cases in which pyloric stenosis was associated with intestinal stenosis (chiefly from round ulcer). Temporary or permanent pyloric spasm (*v.* Gastric Neuroses) from fissure, erosion, ulcer or hyperchlorhydria may cause

the obstruction. The pathological sequence of pyloric stenosis is retention of food within the stomach and hypertrophy of its musculature in attempts, sometimes partially successful and at other times vain, to force the food through the narrow or closed pylorus. The analogy is close between this change and that observed in hypertrophied and dilated bladder from prostatic enlargement or hypertrophy with dilatation of the heart in obstructive valvular disease. In all three instances the hypertrophied muscle eventually degenerates. Dilatation of the stomach may be considered, then, as a disturbed compensation.

2. Muscular Weakness.—Muscular weakness (motor insufficiency, insufficiency of the stomach, atony, myasthenia gastrica) constitutes, in contrast with the first group of causes, a functional and, generally speaking, a more reparable type. (*a*) *Atony dependent on general causes* may occur in acute infections, anæmia and neuroses. The atonic tendency may be acquired or congenital. Rickets is thought to cause certain juvenile cases. (*b*) *Atony may result from gastric conditions,* as cancer, gastritis, hypersecretion, peritoneal adhesions, cholecystitis with pyloric adhesions, diaphragmatic pleurisy and diastasis of the musculi recti abdominales. *Overloading the stomach* with heavy meals, eating indigestible foods or excess of vegetables and carbohydrates, beer drinking, milk in large quantities, much water ingested with heavy meals, as in diabetics, swallowing of foreign bodies by insane subjects, etc., may develop muscular insufficiency. The stomach is usually enlarged, contains residual food and shows a lax, often wasted and thinned musculature.

Symptoms.—The symptoms of dilatation (again emphasized to be a symptom only) differ greatly in degree, ranging from absolute pyloric stenosis to the least motor weakness. The symptoms are partly mechanical, partly chemical.

1. Gastric.—(*a*) The appetite is variable; the tongue is sometimes coated, sometimes clean. (*b*) There is usually thirst and dryness of the throat, more frequently in hypersecretion than in cancer. (*c*) Singultus, *pyrosis* of fatty acids, oppression over the stomach, a cramped or uneasy feeling and eructation of gas are frequent. Gases are never formed without motor insufficiency; the kind of gas is of more scientific than clinical interest, but includes hydrogen sulphide, hydrogen, oxygen, nitrogen, carbon dioxide and even inflammable gases. (*e*) *Vomiting* is *always present in pyloric obstruction* and in marked motor insufficiency. It occurs easily and is often rather belched up than vomited. It occurs when the stomach is full, frequently during the night or at intervals of days; five quarts may be evacuated. *Large amounts argue directly for stagnation and dilatation.* The vomitus is acid, sour and often greatly decomposed. Three layers are noted, a brownish foam, a gray-brown fluid and a sediment of food, stones and seeds, which may have lain for days in the stomach. Gas often bubbles up as the vomitus stands. If gastric juice only is found, it indicates hypersecretion with

motor insufficiency. Chemically, butyric acid, peptones and, in cancer, lactic acid, are found; when the obstruction lies in the duodenum below the papilla of the common duct, bile is also present. Microscopically undigested meat or starch, in benign stenosis sarcinæ and in cancer the Oppler-Boas (lactic acid) bacilli, yeast and other bacteria are found. HCl is absent in cancer. (*f*) *Pain* is frequent; it is cramp-like, boring or burning.

2. Test Meal, Motility and Resorption.—(*a*) The *chemism* depends on the causal affection, particularly if it is gastric (see Ulcer, Cancer, Hyperchlorhydria, Hypersecretion). Hyperchlorhydria and hypersecretion are usually present except in malignancy and are sequential rather than causative. (*b*) *Motility* is best tested by *Leube's test dinner,* consisting of 13 ounces beef soup, 7 ounces beefsteak, one to two ounces bread and 7 ounces water, which should leave the stomach physiologically in not more than seven hours. If food is found later, stagnation is present; if found in the morning when Leube's meal has been given at night, there is great stagnation. The long stay of food in the stomach shows practically what the vomitus shows, *i. e.,* fermentation. Fluid is not absorbed by the stomach, whence the measure of its residual amount is important. There is more fluid in the stomach than was ingested, which is accounted for by hypersecretion of the gastric juice, actual transudation, mucus and perhaps regurgitation from the intestine or hemorrhage. Other tests are less reliable;—the *oil test* consists of administering three ounces (100 c.c.) of olive oil, 70 to 80 per cent. of which should leave the stomach in two hours; if more than 20 to 30 c.c. remain after this time it is indicative of motor insufficiency. *Salol,* grains xv, given just after eating, should normally reach the intestine within an hour, where it is split up into salicylic and carbolic acids; in testing the urine passed in forty-five to sixty minutes after eating, salicyluric acid should be found (reacting violet to tincture of the chloride of iron); salol is normally eliminated in twenty-four hours, and its presence in the urine for a longer time also indicates delayed absorption and dilatation; two errors are possible in this test, *viz.,* its possible decomposition by alkaline mucus in the stomach and its failure to decompose in the gut when there is acid intestinal fermentation (Brunner and Huber). Resorption may be tested by giving five grains of potassium iodide after eating. It should be absorbed and excreted by the saliva in seven to fifteen minutes; this is tested by adding nitric acid to the saliva and touching with starch paper, which will show the blue iodine reaction; a possible error is the prompt absorption of the KI when food is not absorbed. In general a homogeneous chyme means that the motor function is normal; coarse particles occur when there is motor insufficiency.

3. Physical Signs.—(*a*) *Inspection* in a good light is very important. A visible prominence of the dilated organ may be seen below the navel, sometimes showing clearly the abnormally low upper and lower curvatures, the upper curvature never being seen normally.

In obstruction a peristaltic wave passing from left to right or an antiperistaltic wave in the opposite direction often tells the whole story. "Stiffening" of the stomach is an infallible sign of pyloric stenosis. Peristaltic waves may be elicited by applying ether or ice to the epigastrium. The *x*-rays show the outline of the viscus after ingestion of considerable bismuth. (*b*) *Palpation* confirms the above findings and also elicits splashing (*clapotage*) of gas and water in the stomach, a sign on which no great reliance is placed, for splashing occurs in healthy stomachs. The hand may palpate the final gush of gastric contents into the gut. (*c*) *Percussion*—The normal vertical dimension of the stomach is 4 to 5.5 inches (10 to 14 cm.) and its maximum capacity three pints (1,600 c.c.). Filling the stomach with water and percussing, with the patient standing, outlines both the lesser and *greater curvatures,* which may even reach to the pubes. Siphoning the water and then distending the stomach with air pumped in or with carbon dioxide gas, replaces the flatness by tympany. (The carbon dioxide is produced by giving sodium bicarbonate 3j to ij dissolved in water, followed by an equal amount of tartaric acid in water.) When doubtful, the colon may be distended with gas or fluid. The greatest distention is at the fundus. With both gas and water in the stomach the note changes with change of posture, the fluid being in the lower and the gas in the upper parts, as in ascites; the most experienced acknowledge that they have confused enormously dilated stomachs with ascites and ovarian cysts and have introduced a trochar; Jadon published a case in which the stomach contained 45 quarts. Leube was the first to palpate the lower end of the stomach by means of a rigid sound; this has justly been practiced less frequently of late years; the normal distance from the upper teeth to the greater curvature is 23.5 inches (60 cm.). (*d*) *Auscultation* elicits the splashing which may be heard by the patient, and sometimes metallic heart tones, a fine crepitation or hissing noise as gas is generated or a splashing or dripping sound as the patient drinks.

Complications.—(*a*) *Constipation* is the rule, and the stools are hard because less fluid passes the pylorus. (*b*) The *urine* is often *alkaline,* its chlorides are lessened and its amount is reduced to a pint or less as a result of acid vomiting; triple phosphates are common and acetone and diacetic acid are occasional. (*c*) The skin is often dry and (*d*) emaciation is common, even in benign stenosis or muscular insufficiency, because food is imperfectly absorbed; some of it is lost to the organism by fermentation and possibly by tissue-destroying toxins. (*e*) Auto-intoxication from fermentation may develop, causing headache, vertigo, drowsiness, psychoses, polyneuritis, tetany, slow pulse and nervous dyspnœa. Tetany (Neumann and Kussmaul) occurs oftenest in hypersecretion or stenosis from cancer or ulcer; its symptoms are considered under TETANY, and it may be merely noted here that 73 per cent. of cases die (101 cases combined from Riegel, Albu, Fränkel-Hochwart, etc.).

Diagnosis.—The *vomiting, impaired motility,* as shown by Leube's

test dinner, the *visible outlines* confirmed by percussion and the low lesser curvature which is not seen normally, distinguish gastrectasia easily; its cause may be determined with difficulty only. The stomach may be physiologically large (*megalogastria*), but then there is no motor insufficiency, no vomiting and no stagnation. *Gastroptosis* will be considered later. The *degree* of *stasis* is determined by washing out the stomach at varying times after the test dinner. In *pyloric stenosis* there is greater dilatation than in simple motor insufficiency (or atony) and marked peristalsis is always indicative of obstruction. When a diagnosis of pyloric obstruction is established the benign must be differentiated from the malignant type; *benign pyloric obstruction* runs a more remittent longer course of over two years, even of fifteen years duration; HCl and the ferments are usually increased and are always present, sarcinæ are often and lactic acid seldom present; in the writer's experience stenosis due to round ulcer is, in fully half the cases, attended by subacidity or anacidity; while in *cancer* (*v. i.*) the march of symptoms is unfortunately progressive and achlorhydria, cachexia, severe anæmia with leukocytosis, hydrops, pyloric tumor, the Oppler-Boas bacillus and metastases develop with relative rapidity. The *spasmodic* form runs an intermittent course. The *prognosis* depends on the etiology and amenability to treatment; spasmodic types are curable; relative stenosis may somewhat improve. *In infants* with spastic obstruction or hypertrophied pylorus the onset within the first week or month of life is characteristic, with vomiting of even the mother's milk, emaciation, visible peristalsis and sometimes a palpable pylorus.

Treatment.—1. *Etiological factors* should be treated.

2. *General measures,* tending to tone the musculature, are (*a*) *abdominal massage,* practiced only when ulceration and inflammation are certainly excluded; (*b*) *drugs,* especially strychnine, which is often combined with atropine, and the application of an ice-bag to the epigastrium; (*c*) *hydrotherapy;* (*d*) *faradization* of the stomach; external application is seemingly as efficacious and certainly less difficult than intragastric application; and (*e*) *abdominal binders or supports.* This treatment avails only in atony.

3. *Gastric therapy.* (*a*) *Gastric lavage* removes the fermenting contents, relieves the pressure exerted by accumulated food and fluid, cleans off the mucus, lessens vomiting and auto-intoxication, and may permanently help atony and temporarily mitigates pyloric stenosis. The stomach should be washed with luke-warm water until it returns clear of food and mucus; salicylic acid and sodium bicarbonate may be added, as in chronic gastritis: the patient soon becomes accustomed to the tube and welcomes the relief afforded; it is generally said that morning lavage is best, but washing two hours before the evening meal is preferable, as the organ then suffers less stagnation through the night. (*b*) *The food* should be well divided, nourishing and not voluminous; it should be given with very moderate quantities of water, in small amounts and at intervals of three to four hours. The

average diet consists of meat, eggs, leguminous purées, thoroughly cooked rice and small amounts of butter; generally speaking, fats and carbohydrates are more prone to fermentation. P. Cohnheim advises an ounce or two of olive oil in pylorospasm and in moderate benign stenosis. A dry diet was early advised by van Swieten, but moderate amounts of fluid aid in propulsion of food into the gut. Of course much depends on the causal disease; a fluid diet is indicated in great pyloric stenosis and when the hydrochloric acid is decreased; meat should be given if the acid is not decreased and the pylorus is not greatly stenosed. Recently there has been a reversion in favor of one or two fair-sized meals a day, thus giving the stomach ample time to empty itself and to rest. Water is not absorbed by the stomach, as proven by von Mering, who also found that the absorption from the stomach of sugar, peptone, dextrin and alcohol is attended by actual excretion of water into the stomach. In pronounced cases peptonized milk and water are given by rectum to supplement gastric feeding. In all cases careful individualization is indicated; the exact degree of insufficiency, *i. e.,* the time required for absorption, should be tested by Leube's meal and the patient fed accordingly. (*c*) *Posture*—The seriously affected patient should lie on his right side one to two hours after eating, thereby relieving pressure upon the greater curvature of the stomach and directing the food toward the pylorus. (*d*) *Gastric medication*—(See CHRONIC GASTRITIS, ULCER, CANCER, HYPERCHLORHYDRIA). (*e*) *Thiosinamin*—(see page 650).

4. *Surgical intervention* is imperative in severe or malignant stenosis and when medical treatment fails. It embraces (*a*) pylorectomy, of which Dryendorff collected 188 cases with a 57 per cent. mortality; (*b*) pyloroplasty (29 cases with a 21 per cent. mortality); (*c*) gastroenterostomy (215 cases with a 43 per cent. mortality). In 153 operations for benign stenosis Moynihan had only two deaths. As shown in ulcer and cancer (*v. i.*) the earlier recorded mortality has been much reduced. According to Shaw and Elting, in the pyloric stenosis of infancy the mortality under medical treatment is 72 per cent., under surgical 56 per cent. In eleven operations for tetany with pyloric stenosis eight cases recovered (Jonnesco).

CHANGES IN FORM, SIZE AND LOCATION OF THE STOMACH.

Form.—The antrum cardiacum and fore-stomach are œsophageal rather than gastric anomalies. The *antrum cardiacum* is a congenital bulging of the œsophagus just below the diaphragm and the fore-stomach is a congenital dilatation above the diaphragm. There also exists a congenital narrowness of the pylorus as well as hour-glass stomach.

The acquired *hour-glass deformity* is more common and may result from spastic hypertrophic muscle, cancer, ulcer or adhesions around the middle of the organ, abdominal tumors, peritoneal tuberculosis, twisting of the stomach or hernia of it through the mesocolon. Mayo

Robson (1904) in 23 cases found ulcer as the cause in 15, tumor in 4 and perigastritis in 4. It may co-exist with pyloric stenosis. Randolph and Thomas observed 2 cases in one family.

Symptoms may be lacking in the lesser degrees of deformity; in the higher grades they are those of stenosis. (1) Only a part of a given amount of water introduced into the stomach may be recovered (Wölfler's first symptom). (2) After the stomach has been washed till the water returns clear the fluid may suddenly become clouded (Wölfler's second symptom). (3) The stomach is apparently emptied by the stomach tube and yet splashing is obtained (from the cavity nearest the pylorus); Jaboulay called this paradoxical dilatation. (4) Inflation by carbon dioxide produces distention first of the left and later of the right half of the epigastrium (von Eiselsberg's first symptom). (5) Normally when the stomach is distended and the gas passes through the pylorus into the bowel, there is a sharp gurgle over the pylorus; in the hour-glass stomach the gurgle is heard two to four inches to the left of the median line (von Eiselsberg's second symptom). (6) On gastric distention by carbon dioxide Moynihan found tympany only over the upper part of the stomach and not over its lower half. (7) When the stomach is distended a furrow may appear (Eichhorst and Schmid-Monard); Moynihan saw the furrow in 10 cases. (8) Filling the stomach with water and illuminating it by a light introduced into the stomach, the cardiac portion appears translucent and the pyloric part dark (Ewald). (9) A rubber balloon, introduced into the stomach and then inflated through the stomach tube, fills or outlines a part only of the stomach (Hemmeter).

Treatment is surgical. Moynihan successfully operated on 23 cases by single or double gastroenterostomy, gastroplasty, gastrogastrostomy, partial gastrectomy and dilatation of the stenosis.

Situs viscerum inversus (see page 421).

Size.—Megalogastria and microgastria are usually accidental findings if not associated with other gastric disease.

Location.—The stomach at birth is vertical and in adult life is generally held to lie so that three-quarters of it is to the left of the median line. Rosenfeld insisted that the whole stomach is vertically disposed to the left of the mid-line.

Gastroptosis.—(See Enteroptosis, page 654.)

ROUND ULCER OF THE STOMACH.

Definition.—Ulceration of the stomach is characterized in the majority of cases (*a*) etiologically by its occurrence chiefly in young women or in chloranæmic subjects; (*b*) pathologically by a "punched out" funnel-like ulcer, oftenest near the pylorus, and (*c*) clinically by localized pain and tenderness, hæmatemesis and hyperacidity.

Frequency.—It is found in 2.5 to 5 per cent. of all autopsies (W. H. Welch, Orth and von Jaksch). It occurs four times less frequently in America than in Europe.

Etiology.—The causes of ulcer are not exactly known. (*a*) *Age and sex.*—Most cases (70 per cent., Lebert) occur between *twenty and thirty* years of age; in children E. C. Cutler collected 29 cases; 60 per cent. occur in women. In men ulcer develops most frequently between thirty and fifty years of age; the author has seen five cases in men over sixty. (*b*) *Anæmia* and chlorosis are strongly predisposing factors. Anæmia and (*c*) *hyperacidity* are usually regarded as important factors; the writer believes Rosenheim and Cohnheim are correct in stating that hyperacidity is only a result; 73 per cent. of Leube's cases of chlorosis showed hyperacidity. (*d*) Changes in the *vessels.* As a normal access to the stomach of arterial blood prevents its autodigestion, it is thought by some that interruption in the local circulation is the cause of ulceration; Virchow believed that embolism was causal and Klebs held that arterial spasm was an etiological factor. Embolism and ulceration follow burns. It will be noted, however, that ulcer often occurs at an age when arterial changes are rare, though syphilis, alcoholism, arteriosclerosis, cardiac and hepatic diseases apparently cause some of the cases in later life. (*e*) *Occupation* is a factor. Many cases occur in servant girls and in cooks; (*f*) trauma in cooks, shoemakers and tailors is an accessory factor, for the tasting of hot foods and pressure against the stomach promote the development of ulcer. The pressure of corsets, the swallowing in certain trades of particles of iron, porcelain and glass; and imperfectly masticated or improper food, may be brought under this caption. External trauma is a distinct factor. Letulle and Martin hold that (*g*) *bacterial necrosis* causes ulcer, which is clearly shown by Dieulafoy's cases of pneumococcic hemorrhagic ulceration of the stomach.

Pathology.—Scars indicating healed ulcers are three times as frequent at autopsy as open ulcers. The ulcer is known as *ulcus simplex* to distinguish it from malignant or syphilitic ulceration, *ulcus rotundum,* as the smaller ones especially are round, *ulcus pepticum* and *perforans.* The peptic ulcer occurs almost exclusively in the stomach, lower œsophagus and upper duodenum. (See Duodenal Ulcer.) Its general morphology was first thoroughly described by Cruveilhier (1829) and Rokitansky (1839); it has a "punched out," clean-cut appearance; in contour it is round or oval in small, and irregular in large, ulcers; it is usually small, but sometimes invades most of the surface of the stomach and in one case it measured four by seven and a half inches; it is funnel-shaped in old cases; its apex is eccentric and directed toward the serosa, its sides are terrace-shaped and its floor is clean. In 80 per cent. the ulcer is *single,* though as many as 34 ulcers have been reported. Extension is more common in acute than in chronic cases and more toward the peritoneum than laterally. Microscopically there is no infiltration with round cells. Its *localization* is chiefly near the pylorus on the posterior wall or lesser curvature; its *complications,* as hemorrhage and perforation, and its *sequelæ,* as pyloric stenosis and deformations, will be considered under clinical symptoms.

Symptoms.—The *general clinical picture* varies, cases being typical, doubtful or entirely latent.

In the *typical case* the symptoms develop gradually with a sense of epigastric pressure after eating, which later becomes a more violent, cutting, *localized pain;* this is observed one-half to one and a half hours after eating; it lasts throughout the period of digestion and ceases only with vomiting or passage of the food into the bowel. The appetite is usually good; *hyperacidity* is noted in the test meal or the vomitus; *tenderness* occurs over the epigastrium, and in fully half the cases *bleeding* from the stomach or blood in the stools is observed.

The *typical triad* of cardinal symptoms, taken together or possibly singly, is characteristic: (1) the pain with localized tenderness, (2) hæmatemesis and (3) hyperacidity.

1. Pain.—The most important, characteristic and constant symptom (in 90 per cent.) is pain, which is usually paroxysmal, is localized and occurs during digestion. It is boring or burning, seldom lancinating and is due to irritation of the gastric nerves aroused by peristalsis, nerve erosion or hyperacidity. The *diet* affects the pain greatly; milk, for instance, is well tolerated in certain cases, but solid food usually produces excessive pain and very *hot* or very *cold foods* or beverages almost invariably cause discomfort. This disappears quickly with rectal feeding. The seat of the pain varies in different patients, but is the same in a given case. Generally it is *epigastric* and very often near the ensiform. Ewald describes it as cramp-like in the right mammary line under the liver. Sometimes there is wide irradiation toward the sternum, ribs, shoulder, scapula and infrequently in the brachial plexus and pulmonary branches of the vagus, resembling angina pectoris (Traube) or producing pain in the arms and legs (Müller). The pain varies with change of position, depending on the localization of the ulcer, but there is always pain when the patient lies on the left side.

In relation to eating, sometimes the pain occurs two to ten minutes after eating, the old "wound pain," supposedly due to contact of food with the eroded nerves in the ulcer. *Typically,* pain occurs at the height of digestion, *i. e., one-half to one and one-half hours after ingestion of food,* caused by the hyperacidity. Patients fear to eat because of the inevitable pain. Pain may be worst at the time of the menses. Pain from the scars or adhesions of an ulcer is infrequent and is usually drawing or vague. In old ulcers pain is atypical or absent. The time of development of the pain does not locate the ulcer, though if it comes on at once it may suggest œsophageal or cardiac ulcer; or, if it occurs two and one-half hours or more after eating it is suggestive of ulcer of the duodenum.

Tenderness is most often found under the ensiform. It is usually *circumscribed* and is more often and more advantageously found on the lightest touch rather than on deep palpation. A diagnosis from tenderness alone is somewhat unsafe. Tenderness in the back (Cruveilhier) suggests ulcer on the posterior wall of the stomach and is

found in 25 to 33 per cent. of cases over an area of one or two inches to the left of the tenth to twelfth dorsal or upper lumbar vertebræ. The abdomen, back and chest are often exquisitely hyperæsthetic. Palpation must be guarded, lest perforation ensue.

2. Hæmatemesis.—The second cardinal symptom is hæmatemesis. Its frequency is from 25 to even 80 per cent.; the average figure is probably 50 per cent. Vomiting of blood in ulcer is most frequently from ulcer in the pyloric region (posterior wall and lesser curvature) or, less frequently, from ulceration reaching the liver, pancreas or spleen. Hemorrhage when small may be overlooked (occult hemorrhage) or blood may be found only in the stools; melæna alone is found in 11 per cent. of cases. The hemorrhage must amount to a pint to turn the stools black. It may be large and lethal; no blood may be vomited and the stomach and bowels are found to be filled with one massive clot. The usual signs of internal hemorrhage exist, as collapse, pallor, moderate fever, brain anæmia, hæmic murmurs, amaurosis and rarely optic atrophy or hemiplegia. If profuse it is voided *bright red;* if slow and moderate the acid turns it dark, *"coffee-ground vomitus."* If red blood cells cannot be found in the vomitus because of complete disintegration of the cells, blood can be detected by treating it with acetic acid, extracting with ether and then testing with the guaiac and turpentine reaction, when blood will be shown even if the spectroscope shows nothing (Bial). "Occult hemorrhage" occurs in 25 to 50 per cent. of cases (see Hæmatemesis and Cancer); it is not as constantly present as in gastric cancer. The tests for *occult hemorrhage* are valuable to determine the length of the medical treatment of ulcer and to detect the tendency to bleeding during the course of gastric ulcer. Macroscopic blood is found in the stomach washings less frequently than is to be inferred from textbook descriptions, and apparently normal vomitus may show blood microscopically or chemically. Profuse hemorrhage usually indicates deep ulceration. Hemorrhage complicates acute more than chronic cases. The hemorrhage is usually fairly profuse in ulcer; it may be spontaneous during sleep or rest or may result from excitement, physical exertion or improper diet. In a case of "chronic dyspepsia" with vague symptoms the writer inflated the stomach with the usual amount of carbon dioxide; the patient collapsed in the office and vomited nearly a quart of blood.

3. Hyperacidity.—This is the third cardinal finding. Hemmeter finds it in 90 per cent. and Leube in 50 per cent.:

HCl according to	Normal acidity.	Hypochlorhydria.	Achlorhydria.	Hyperchlorhydria.
Ewald	57%	9%	..	34%
Eichhorst	36%	34%	23%	7%
Author	46%	20%	4%	30%

The variance noted in these figures is due to combining old callous

ulcers with the fresh chlorotic ulcers; in old ulcers hyperchlorhydria is present in only 10 per cent. and they explain most of the cases of decreased acidity; in fresh ulcers hyperacidity occurs in 70 per cent. Hyperchlorhydria is probably due to irritation of the vagus (Pawlow). Complicating cancer, hypersecretion or catarrh may cause subacidity.

Test meals should not be given to determine an excess of acid when there is pain, tenderness and vomiting of blood. The stomach tube, however, is used by Riegel, Boas, Rosenheim and Fleiner not only for diagnosis in atypical cases, but also to relieve the more dangerous vomiting and to introduce silver and bismuth into the stomach. Organic acids, as lactic, are absent, the carbohydrates are poorly digested and the albumins are rapidly converted into albumoses or peptones. The motility of the stomach is normal in one-quarter and slow in three-quarters of the cases. If pyloric obstruction exists the HCl is increased.

4. Other Symptoms.—*Vomiting,* present in 75 per cent., is a less valuable diagnostic than pain. Some cases never vomit or do so only several hours after meals. It occurs at the height of the pain, one, two or three hours after eating, whereas in dilatation or hypersecretion it occurs much later. Vomiting and pain depend directly on the diet, heavy meals increasing and appropriate diet lessening each. Some degree of parallelism exists between the pain and vomiting, the latter usually relieving the former. The vomitus consists of fine particles, is sour, grits the teeth and shows much acid and no fermentation. Dyspepsia occurs from catarrhal gastritis, dilatation or other complications. The appetite is frequently good or exaggerated. Acid eructations (pyrosis) may cause a burning sensation back of the sternum or between the shoulder blades. If there is much œsophageal burning it indicates incontinence of the cardiac orifice. Diarrhœa is rare. Constipation is due to anæmia, diet, rest, vomiting, possibly reflex inhibition of the motility of the bowel or to impaired peristalsis by scars and adhesions. Dysmenorrhœa and amenorrhœa are not infrequent. The nutrition varies and emaciation may be observed (cachectic type); anæmia may become profound. The urinary findings are not characteristic; reduction of the chlorides cannot be used to differentiate from carcinoma; the urine is slightly acid or alkaline when the stomach is dilated; the phosphates are increased; with dilatation, indican and ethereal sulphates are increased. Acetonuria and diaceturia sometimes result from inanition and rectal feeding.

Clinical Forms of Lebert.—(*a*) The *hemorrhagic* form may be either acute or chronic; hemorrhage may be the first or the last and fatal symptom. (*b*) In the *perforative* form there may be entire latency of previous symptoms. (*c*) In the *dyspeptic* type dyspeptic symptoms are most prominent; the evidences of ulcer are absent or there are atypical symptoms, such as pain which is not sharp, vomiting which is infrequent and excess of hydrochloric acid, but no increase

in mucus. (*d*) The *cachectic* form characterizes old ulcers with a cicatrix producing dilatation and with chronic hypersecretion; this form may be confused with carcinoma. The other types are as follows: (*e*) *gastralgic* or neuralgic form; (*f*) *vomitive* form; (*g*) latent ulcer (scars at autopsy); (*h*) a form with *tumor;* (*i*) *recurrent* type; (*j*) *stenotic* type; (*k*) *carcinomatous* type.

Clinical Course.—The course is usually chronic, for ulceration is frequently long latent before diagnosis is possible and probably lasts some time after all symptoms have disappeared. Pain usually indicates incomplete cicatrization or certain other sequences, but cessation of pain is no certain criterion of healing. The course may be acute and rapid, especially under appropriate therapy. The course lasts from three to five years (Lebert) or even twenty to thirty-five years, usually due to complicating pyloric obstruction, dilatation or adhesions. Very large ulcers probably never heal. They frequently *remit* with appropriate therapy, but recur with the resumption of the old diet. Ulcer can be considered cured only when all symptoms have been absent for six months.

Localization.—This can rarely be determined accurately (Riegel and Leube). Tenderness and tumor are indicative of ulcer on the anterior wall, but hemorrhage and dorsal pain suggest posterior involvement (Gerhardt). Pain in a lateral decubitus suggests localization in the fundus or pylorus; dilatation, in the pylorus or duodenum; pain under the ensiform during the act of deglutition suggests cardiac ulcer. Ulcers are palpable in only 0.1 per cent. of cases (Leube). The percentage of involvement of the cardia is 6; of the fundus 4; of the lesser curvature 36; of the posterior wall 30; of the pylorus 12; of the anterior wall 9. "In over 80 per cent. of the cases it occurs in a relatively small area about the pylorus, lesser curvature and posterior wall."

Complications and Sequels.—(*a*) *Perforation* (3 per cent.) may occur into the peritoneum, usually with symptoms of collapse, peritonitis and cessation of vomiting (Traube); (Brunner finds vomiting in 66 per cent. and Gross in 33 per cent. of such cases). Perforation may be prevented by omental, peritoneal, pericardial and pleural adhesions. The danger of perforation in ulcer of the anterior wall is seven times greater than of other locations. Perforation of a latent ulcer may be confused with rupturing appendicitis or salpingitis. Perforation may occur into the heart, colon, gall-bladder, portal vein, pericardium, thoracic or abdominal wall; or it may reach the space between the liver or spleen and the diaphragm, producing pneumothorax subphrenicus, which is suggested by pleurisy, epigastric and hypochondriac pain, stiffness in the back, painful eructations and swallowing, singultus or local œdema. (*b*) *Simple peritonitis* (perigastritis) may cause a friction murmur over the stomach. (*c*) According to Rosenheim 6 per cent., Liebert 9 per cent. or Zenger, the majority of cases of *cancer* develop from ulcer. The author saw with Dr. Harpole a case of carcinoma developing in

an ulcer scar in a girl twenty-six years old. The typical symptoms of ulcer are followed by loss of weight and persistence of symptoms, usually by gradual lessening of hydrochloric acid and other evidences of carcinoma. (*d*) *Adhesions* about the stomach may interfere with its motility, produce deformation, such as the hour-glass stomach or diverticulum, neuralgia which outlasts the ulcer and obstruction of either orifice of the stomach. Dilatation of the stomach usually indicates pyloric stenosis; it may also result from spasm of the pylorus. Scars may, in rare cases, produce intestinal obstruction. Troublesome adhesions may be finally so stretched as to relieve all symptoms.

Diagnosis.—The diagnosis is determined by (*a*) the etiological factors, (*b*) the cardinal symptoms and (*c*) the various clinical types enumerated. In general the diagnosis is easy in the young, but may be difficult in older individuals with chronic ulcer.

Any two cardinal features confirm the diagnosis. *Pain* is often the only symptom; if it is atypical, hyperacidity may indicate ulcer, though the former is often observed without ulcer; Leube proposes for such cases the therapeutic test, although both cases may respond to the treatment. According to Riegel the chief difference is that pain in simple hyperacidity is less regular than in ulcer and that localized tenderness is absent. Seemingly primary hyperchlorhydria may be found to be ulcer. Where pain alone exists one must always remember the gastric crises of tabes. Ewald has called attention to the pain of small hernias in the linea alba. Pain in the transverse colon may cause confusion, but it usually ceases on evacuation of the bowels. Without careful examination movable kidney may be confused with ulcer. The pain in *gall-stones* is paroxysmal, but more irregular and independent of eating. The gall-bladder is tender and may be distended and palpable. The paroxysm in gall-stones is longer and is often attended by fever and a chill. The pain more often radiates to the right side and to the shoulder and the vomiting is not that of ulcer, though hyperacidity and gastric dilatation may follow cholelithiasis. Chronic poisoning, as by arsenic, may be suspected, as in the case of the Duchess of Orleans until Littré diagnosed ulcer.

Hemorrhagic erosions and fissures of the stomach present no constant clinical picture. After eating there is a sense of burning over the entire stomach, which is not altered by change of position. The chemism and tender points of ulcer are lacking, though Cohnheim usually finds hyperchlorhydria; he also believes that solid food relieves the pain. Shreds of tissue are always found, according to Pariser, which cease with cessation of pain. Fatal hemorrhage is recorded.

In *duodenal ulcer,* inflammation, new growths of the liver, perihepatitis, cysts and chronic pancreatitis, hyperacidity is absent, although in some instances of duodenal ulceration hyperacidity is observed. Duodenal ulcer occurs mostly in men, in alcoholics and after burns. Pain is often absent in duodenal ulcer, but when present occurs later than in gastric ulcer and in the parasternal line. Vomiting gives less relief and the vomitus is usually alkaline, con-

tains bile and may digest fibrin (Boas). Icterus is not constant enough for diagnostic reliance. Hemorrhage into the bowels is more common than in gastric ulcer. Its sudden appearance with recurrence and profound anæmia is most suggestive.

Pyloric spasm may also occur in myocarditis, aneurysm, coronary atheroma, tænia, ascaris, the spastic form of constipation or chronic nicotinism (Ageron).

Differentiation from *carcinoma* is not very often necessary, though in middle-aged patients confusion with ulcer may occur. In Kraus' clinic the author saw two cases; one was an old man with cachexia, pyloric tumor and decreased hydrochloric acid, which was diagnosticated cancer; the other was a young girl with vomiting of blood, in whom the diagnosis of ulcer was made. The post-mortem examination showed that the old man had a round ulcer and the girl had a cancer. The author has seen five cases of carcinoma under the age of thirty. The table on page 601 considers differentiation from cancer, gastritis, gastralgia and hyperchlorhydria.

Prognosis.—Though ulcer occurs most frequently between the ages of twenty and forty, it is most fatal between forty and sixty. It is generally stated that the mortality is 6 to 10 per cent., of which about 4 per cent. each is due to hemorrhage and perforation.

	Recovery.	Death.	Improvement.	No Improvement.
Eichhorst	85%	8%	4%	3%
Leube	74%	2.4%	22%	1.6%
Köhler	61%	6%	30%	3%

Very deep, old ulcers rarely heal. The deeper the ulcer the greater the danger of hemorrhage and perforation. Ulcers with hypersecretion give a relatively poor prognosis. Carcinoma must be considered when treatment is not successful. One of the author's cases died from exhaustion, although the autopsy showed perfect healing of the ulcer. In over one thousand cases the deaths from hemorrhage and perforation (each 1 per cent.) were about equally divided (Leube).

Treatment.—1. Prophylaxis.—Prophylaxis concerns care of the stomach in anæmia and chlorosis and care in the diet.

2. Absolute Rest.—Absolute rest in bed for a month or more was first recommended by Cruveilhier, in addition to which Leube advises the frequent application of hot fomentations to the epigastrium, which relieve pain within a few days. Women should remain in bed during menstruation, even after recovery is seemingly complete.

3. Absolute Rest of the Stomach.—Distention of the stomach by even small quantities of food or water excites peristalsis and secretion, distends the ulcer and prevents granulation. Rest permits the stomach to contract and allows the edges of the ulcer to come more completely in apposition. The more absolute the rest of the stomach the more rapidly the ulcer granulates and cicatrizes. The time for

	CANCER.	ULCER.	CHRONIC GASTRITIS.	GASTRALGIA.	HYPERCHLORHYDRIA.
Etiology.	75% over 40 years.	75% under 40 years—2-4 times oftener in women.	Males.	Women.	Males.
Tongue. Appetite.	Heavily coated. Usually anorexia.	Usually clean and red. Good; only fears to eat.	Gray-white fur. Poor.	Normal. Variable. Sometimes perverted.	Clean. Good, often increased.
Pyrosis.	Often pyrosis.	Water brash.	Frequent.	None.	Present.
Belching.	Usually fœtid belching.	Usually absent.	Copious.	Variable.	Usual.
Pain.	The pain is less intense, but more constant; seldom free intermissions, during which no distress is felt in gastric region. Pain not increased at the height of digestion. Tenderness less frequent and intense.	Intense; appears shortly after meals; grows severer on pressure; disappears at the end of digestive period; perfectly free periods more frequent; paroxysmal.	May be present but not pronounced and rarely actual pain but tenderness of diffuse character. Lancinating pains in atrophic gastritis only.	The pain appears without regularity, and not dependent upon meals; may be relieved by pressure; intervals of several days free from pain. Tenderness rare; conforms to outlines of the stomach. Electrical test (Leube) during digestion; if pain decreases, it is gastralgia; if it does not, may be ulcer or gastralgia.	The pain appears about one or two hours after meals, disappears after eating, especially meat, milk or eggs, or after the administration of bicarbonate of sodium. Pain is less regular.
Vomiting.	Usual. Hours after eating or once a day or alternate days, etc. Copious, coarse mucus, fermented.	Frequent—at height of digestion—smaller amount, finely homogeneous, no mucus, acid.	Frequent; mucus.	Rare; no mucus.	Much less frequent, then acid and no mucus.
Hæmatemesis.	Frequent — small dark, like coffee grounds, decomposed, usually late in disease.	Frequent—copious, bright; may be early symptom.	None.	None.	None.
Test Meal.	Early reduction HCl and later, total absence; ferments decreased or absent; lactic acid present; impaired motility and absorption; abundant mucus, Oppler-Boas bacillus—yeast—possibly tumor shreds; coarse undigested particles of food.	Hyperchlorhydria often; ferments normal or increased; no lactic or Oppler-Boas bacilli; food well digested. No mucus.	Decreased HCl (rarely increased, gastritis acida); later may be achylia. Much mucus.	Normal or variable. If HCl is decreased, the ferments are normal.	As in ulcer.
Tumor.	In 80 per cent.	In but one pro mille (from pyloric spasm, hypertrophy, cicatrix, etc.).	None.	None.	None (very rarely from pyloric spasm).
Collateral Symptoms.	Cachexia—œdema.	Absent.	Absent.	Absent. Nervous stigmata often.	Absent.
Course.	Part of a year.	Longer; helped by treatment—aggravated by arsenic.	Chronic. —ditto.	Arsenic helps.	Like ulcer.

complete granulation varies with each case and chronic ulcers show little or no tendency to cicatrize. An ice-bag promotes contraction of the stomach. *Rectal feeding* is imperative and the usual rules should be followed; peptonized milk, grape sugar, baby foods, meat juice and eggs, should be given, in amounts not to exceed eight ounces, at eight-hour intervals; they should be injected slowly, with the patient on his left side and should be given warm, which precautions obviate excessive peristalsis. A fountain syringe with a soft tube is always used. Nutrient rectal enemata can be retained invariably if the technique is carefully carried out. Wine or spirits almost always irritate the bowel. Rectal tenesmus is obviated by administering, a short time before the enema, an opium suppository of one-half grain of the extract. Though rectal feeding gives the patient but one-quarter of the food necessary to maintain metabolism, it is sufficient, because the patient is absolutely quiet. The mouth should be kept clean to avoid parotitis. Thirst should be relieved by giving two daily warm saline enemata; the patient may rinse his mouth out, but he should swallow nothing, either water or melted ice.

4. Pain.—Rest of the stomach relieves the pain in almost every case; the use of narcotics should be limited; small doses of opium or morphine by rectum, or atropine, may be given. There is danger of the opium habit.

5. Superacidity.—The treatment of superacidity should be initiated after two weeks of absolute gastric rest. *Massive doses of sod. bicarbonate* 3ss–3j in starch wafer, followed by *a swallow of water,* give the best results. Then Carlsbad water should be given; it should be lukewarm, given in small quantities and taken in sips. Ziemssen and Leube have proven that alkalies are compatible with digestion. Carlsbad water consists of sodium sulphate 50 parts, sodium bicarbonate 6 parts and sodium chloride 3 parts; it possesses no value other than its alkalinity; it neutralizes the acid, thereby tending to check the ulceration, aids intestinal digestion and cleanses the stomach. Ziemssen administered half an ounce of the solid salts in a pint of water; this was taken in quarters every ten minutes, the last dose being given one-half hour before meals. Vichy water may be used. Roberts prescribed a lozenge composed of magnesium carbonate grains 3½, chalk grains 2½, sodium chloride grain 1, which dissolved slowly in the mouth, increased the salivary flow and neutralized the excess of acid.

6. Diet.—After ten days of rectal feeding, five or six meals daily of predigested milk with lime water should be given in small quantities, that the edge of the ulcer may not be stretched. Cooked milk is more easily digested and curds less. The casein engages and neutralizes the acid. If peptonized milk is distasteful it may be administered as a thin gruel, especially when the stomach is irritable and painful. Potato purée, arrow-root, wheat-meal, baby foods, meat juice and buttermilk may be given; soups with meal and alkalies which precipitate the casein in finer form, beef peptone jelly and

ice cream may also be given; but it should be remembered that the milk diet, recommended by Cruveilhier is most efficacious. After the patient has been on the above-mentioned diet for ten days, soups without fat, rice, sago, soft-boiled eggs, calves' brains and chicken may be administered. Slightly boiled or poached eggs are digested better than raw eggs and may be given with bread-crumbs from the inside of the loaf. Custards may be given. Tea and coffee should be forbidden. Sugar was considered deleterious by Cruveilhier, but Strauss has found that it lessens the amount of acid and he recommends that the patient should have a dram of a 20 per cent. solution. Cohnheim gives olive-oil freely at all times to relieve spasm and inhibit hyperchlorhydria. After fifteen days Leube permits grated beef, and after about another week tender roast beef, chicken and pigeon, to which others would add lamb, macaroni, thymus gland and bread. After the fifth week roasts and soufflés may be given.

7. The Bowels.—Plain enemata may be administered.

8. Drugs.—Much has been written in regard to bismuth and silver. *Bismuth* relieves pain and is taken most advantageously in solution, but it has no curative value and is less efficacious than absolute, prolonged rest of the stomach. Fleiner, Kussmaul and others administer bismuth by means of the stomach tube. Large doses may accumulate in the ulcer, and the drug is expensive. *Silver* has been used and some writers even wash the stomach of the patient before meals with a solution of 1 to 500 or to 1,000 (Reichmann). The silver is readily precipitated by the acid or the albuminoids and may produce nausea, diarrhœa and even argyria. Iodides are indicated in syphilitics, by whom they are well tolerated.

9. Symptomatic Treatment.—*Pain* is best relieved by rectal alimentation, as is vomiting, which sometimes may necessitate the administration of bismuth, minute doses of carbolic acid in mint water or the use of the ice-bag or sinapisms. *Hemorrhage* is treated by absolute rest of the stomach, intestine, mind and body; morphine quiets peristalsis and mental excitement. No styptics or astringents should be given and ergot is contra-indicated. Syncope promotes spontaneous coagulation and stimulation should be avoided as long as possible. In severe hemorrhages the use of saline transfusions should be considered, but more than one pint should seldom be given, for death has occurred from reaction and renewal of hemorrhage. Gelatin injections may be indicated for severe hemorrhages.

10. Surgical Treatment.—Seventy-five to 96 per cent. of cases, according to Leube, heal absolutely or improve greatly under proper medical treatment; this is especially true of fresh ulcers. Leube gives the following indications for surgical interference: (*a*) When the *hemorrhages* are small and repeated and persist in spite of rectal nutrition; in single large hemorrhages operation is contra-indicated. Moynihan holds a similar view and lost but 1 out of 70 cases of chronic hemorrhages, which result he attributes to his treating the ulcer instead of performing simple gastro-enterostomy. Savariand

estimates the operative mortality at 50 and Rodman at 33 per cent. Tuffier (1905) finds a mortality of 36 per cent. (56 cases). (*b*) *Gastro-enterostomy* is indicated for mechanical difficulties, pyloric obstruction, persistent pyloric spasm and pain; obstruction at the cardia, or hour-glass stomach, also requires surgical treatment. Forty-nine cases of jejunal ulcer have followed operation, since Braun's first case; it develops usually after a year or more. Mikulicz in operations for benign gastric disease finds a 7.5 per cent. mortality in his series of 1897–1899. Moynihan in 15 operations for hour-glass stomach had a mortality of 20 per cent. and in 100 cases of gastro-enterostomy a mortality of 2 per cent. (*c*) *Perigastric adhesions* or abscess and collections of pus beneath the diaphragm, necessitate operation. (*d*) In cases of *perforation* if operation is undertaken within 12 hours, 71 per cent. recover. The best figures are those of English and Graffe with 50 per cent. of recoveries and, between 1900 and 1904, of H. A. Hare, with 37 per cent. Donati, of Florence, collected in 1905, 1,041 cases of operation on ulcer cases; the mortality in perforation was 48 per cent.

Surgical interference is necessary in a limited number of cases. Sahli argues that (i) reports only show that many of the patients stand an operation; (ii) gastro-enterostomy is indicated only for anatomical stenosis; too many cases of spasmodic stenosis are called anatomical and at operation too many contracted pylori are called cicatricial. (iii) Gastro-enterostomy is not proven beneficial for hemorrhage, diet being the important indication. Cases operated on often die from hemorrhage after the operation. (iv) No proof exists that ulcers heal better with gastro-enterostomy than with medical treatment. (v) Excision of the ulcer is too radical. The writer's experience justifies the conclusions that (*i*) fresh ulcers always and (*ii*) chronic ulcers in half the instances belong to internal medicine; (*iii*) half the inveterate cases respond to medical treatment; (*iv*) most of the cases with "chronic dyspepsia" and almost none of the cases with hemorrhage call for surgical interference.

CANCER OF THE STOMACH.

Cancer of the stomach is more frequent than cancer of any other organ; it constitutes 33 to 50 per cent. of all cancers and is found in 2 per cent. of autopsies.

Etiology.—This is as obscure as in other carcinomata. (*a*) *Age.*—75 to 80 per cent. of the cases occur in subjects after the fortieth year. According to Lebert 1 per cent. occurs in persons under thirty years; 18 per cent. in those between thirty and forty; 61 per cent. in those between forty and sixty and 20 per cent. in those over sixty years of age. The author has seen five cases in persons under thirty years, which leads him to believe that Débove's collection of 32 cases underestimates their frequency. Rare congenital cases are recorded (Wilkinson, Débove, Collingsworth). Of late years cancer is be-

coming more frequent in relatively young persons. (*b*) *Sex.*—It is slightly more frequent in males. (*c*) *Heredity* is noted as an etiological factor in 6 to 17 per cent. and in some families the number of cases of carcinoma is astonishing. (*d*) Ulcer of the stomach is an antecedent in at least 6 per cent.; C. Graham found ulcer in 60 per cent. of his cases. (*e*) Other causes are less definite. Trauma, external pressure, as in shoemakers, or internal ulceration by caustics, is noted in some cases. Pulmonary tuberculosis and alcoholism are slightly predisposing factors. Cancer is almost unknown in certain tropical and subtropical localities and in temperate zones a local disposition is occasionally apparent.

Pathology.—(*a*) In *origin* it is an abnormal growth of the glandular epithelium, as was first proven by Waldeyer, which unlike mere adenomatous or papillomatous proliferations, extends beyond the mucosa into the other coats of the stomach and very often into other organs and tissues. Cancer may develop in the normal mucosa, in an atrophied mucosa or in open or cicatrized ulcers. (*b*) It is almost *always primary,* is very rarely metastatic or extends to the stomach by contiguity; secondary growths are usually from mammary or pancreatic cancer; Welch collected 37 cases. It is almost invariably single.

Histology and Morphology.—A tumor, in the clinical sense, usually develops, although in some cases the cancer is infiltrative and flat (*carcinoma planum*). There are three main types: (*a*) *Carcinoma fibrosum* (scirrhus), the most frequent type, contains much poorly vascularized stroma and relatively little carcinomatous tissue; it is hard and, leading to much induration, is prone to stenose the pylorus, where it is especially frequent, or to stenose the cardia; if it is diffuse it causes the stomach to shrink (carcinoma atrophicans); its onset is insidious; it grows slowly and ulcerates less than other types. (*b*) *Carcinoma medullare* is next in frequency; the cell elements preponderate over the stroma; there is much soft, vascular tissue and the knife scrapes out much "cancer juice"; it may form cauliflower-like exuberances; early extensive degeneration, ulceration and metastases are common; this type includes the cylindrical celled adenocarcinoma, epithelioma or the very vascular fungus forms. (*c*) *Carcinoma gelatinosum* (colloides) is least frequent; it produces nodular tumors or diffuse infiltrations, which contain colloid material; it inclines rather to extension by contiguity than to metastatic deposits.

The *gastric mucosa* shows focal wasting of the peptic- and acid-secreting epithelium and substitution by cylindrical epithelium, round cells and connective tissue. This *secondary atrophic gastritis* explains the chemism of gastric cancer—*viz.,* diminution in the secretion of acid, rennet and pepsin; in some cases it has regressed after surgical extirpation of the tumor, the secretion of acid and pepsin returning.

Localization.—Cancer occurs (*a*) in the pylorus in about 60 per cent., (*b*) in the lesser curvature in 20 per cent. and (*c*) in the cardia

in 10 per cent. of the cases; in round numbers, therefore, about 90 per cent. of cancers occur in these sites; this is explained by their greater exposure to mechanical insult, their relative fixation and the greater muscular activity of the pylorus and cardia; many tumors, apparently pyloric in origin, really begin in the lesser curvature, whence, in other words, a palpable pyloric tumor often means one of considerable extension; this is an important surgical point.

Complications.—(*a*) *Ulceration,* which is really more a part than a complication of cancer, results from inadequate vascularization; it is usually central and does not depend on the size of the tumor. In some cases the tumor may ulcerate almost completely and Ziegler has observed total disappearance, though metastases declared the nature of the scar. (*b*) *Hemorrhage* or slow seeping of blood is caused by ulceration, as is also (*c*) *perforation* (3 per cent.) into the peritoneum or by adhesions into adjacent abdominal organs or vessels, into the lung, pleura or pericardium or into the abdominal wall, of which latter 25 cases are recorded (Taussig); perforation is not more common because adhesions often prevent perforation. (*d*) *Stenosis* of the cardia or pylorus is common, sometimes causing hypertrophy of the muscle fibers, which on section of the stomach appear as red lines. (*e*) *Dilatation* of the stomach follows pyloric obstruction, atrophy is observed in cardiac localization or in carcinoma atrophicans (diffuse cancer) and hour-glass deformation may result when an annular scirrhus develops in the mid-stomach. (*f*) *Local extension* by contiguity occurs by the submucous lymphatics and there is early involvement of the lymph nodes along the lesser curvature; other lymphatics are soon invaded; the peritoneum is quite often involved and less often there is extension into the portal vein. (*g*) *Metastases* occur in 50 per cent.; they are distributed as follows: the liver is involved in 28 per cent., the peritoneum in 37 per cent., the lymphatics in 23 per cent., the pancreas in 20 per cent. and the lungs in 8 per cent.

Symptoms.—General Clinical Picture.—Anorexia, distaste for meats, mild dyspepsia, gastric oppression and eructations after eating, usually mark the insidious onset. Much less frequently the disease begins acutely or follows closely on symptoms of gastric ulcer. Vomiting occurs and then symptoms of gastric distress after eating solid food and of mechanical or secretory insufficiency develop. There is gradual loss of weight together with loss of strength and endurance. Examination reveals anæmia, cachexia, emaciation, lax skin, flabby muscles and often a tumor near the pylorus. A test meal shows imperfectly digested food and absence of free hydrochloric acid. The symptoms progress steadily or remissions occur, especially early in the disease. The ankles become puffy and the prostration is profound. Ulceration may relieve pyloric or cardiac stenosis, but gradually within a year after the first marked symptoms death results from the exhaustion due to pain or vomiting, from local complications or intercurrent disease.

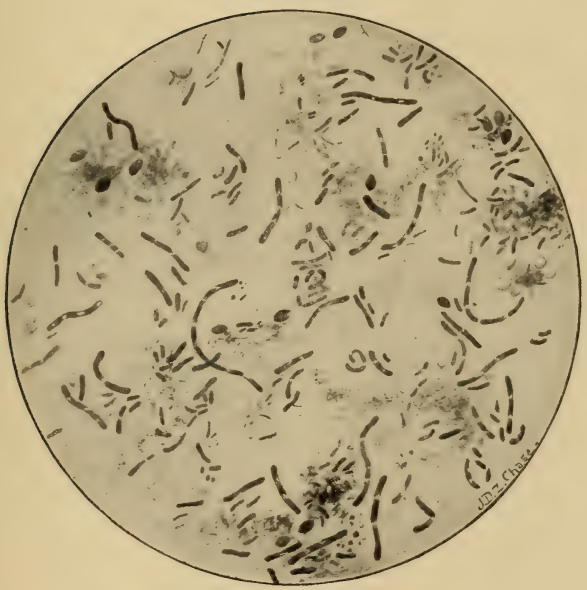
1. General Digestive Symptoms.—(*a*) The *tongue* is almost

always heavily coated, not being cleaned by mastication, and there is a bad taste in the mouth. The appetite is decreased or absent in 85 per cent. and there is a special distaste for meat and fats; the appetite is very rarely excessive; a normal appetite may be retained when the motor power is good and cancer develops on an ulcer. (*b*) *Pain* is present in 90 per cent. It is usually diffuse, dull, oppressive and less severe than in ulcer. It is increased several hours after eating or in the night, but is not wholly relieved by vomiting. Localized pain and tenderness are suggestive of circumscribed peritonitis. Pain may be reflected to the sternum, shoulders and acromion. (*c*) *Vomiting* (in 85 per cent.) is an obstinate and rather late symptom, resulting chiefly from pyloric stenosis, with which it runs more or less parallel. It appears late in the day or in the night, and may occur only on alternate days. There may be no vomiting in cancer of the curvatures, in diffuse cancerous infiltration involving the muscularis or in pyloric cancer when ulceration is marked. The vomitus contains much mucus, poorly digested food, little or no free hydrochloric acid, much lactic acid and often blood, which may be obvious or occult. Fæcal vomiting usually indicates gastrocolic fistula (of which 70 cases are recorded). (*d*) *Hæmatemesis,* present in over 50 per cent. of cases, is due to parenchymatous oozing and is therefore moderate in amount; it is dark in color, as the blood is retained long in the stomach and is converted from oxyhæmoglobin to hæmatin. The author has seen four cases with fatal, profuse, bright hæmatemesis; profuse bleeding occurs in 1 per cent. (*e*) *Constipation* is present in 75 per cent. and diarrhœa in 25 per cent. of the cases; in two cases the writer has seen ichorous stools resulting from necrosis of the lower jejunum and ileum.

2. Special Gastric Symptoms.—(*a*) *Tumor* is felt in 80 per cent. and seen in 40 per cent. of the cases, but is not an early symptom. It is most often missed in persons with wide thoraces and rigid abdominal muscles. In most instances it occupies the *right epigastric area,* as a round, ovoid, irregular mass, best determined when the knees are drawn up and the mouth is open, after the bowels have been flushed out and the stomach distended; in some cases the tumor may be felt lower or even on the left side. Examination should be made with the patient in several postures, as on the side, on the face, genupectoral position, etc. The tumor may be palpable one day and not to be found on the next. *Distention* of the stomach *by gas* (von Frerichs) brings forward tumors of the anterior wall and greater curvature, obliterates tumors of the posterior wall and lesser curvature and pushes downward and to the right tumors of the pylorus; motility on distention excludes adhesions. Distention must be moderate lest the stomach rupture. If the pylorus is incontinent from ulceration or infiltration, the gas escapes rapidly into the gut (Ebstein). The *x*-rays outline the stomach after administration of bismuth. With or without distention, a *visible peristaltic wave* suggests pyloric obstruction. Percussion is wholly subordinate to palpation

and inspection, and auscultation occasionally elicits friction or a stenotic murmur over the abdominal aorta. *Respiratory excursion,* generally stated to be absent if the stomach is not adherent to the liver, is often present, though less in degree than the excursion of the liver or spleen; if adherent to the omentum there is no excursion. The *expiratory fixation* is more important; if nonadherent tumors of the stomach, intestine and omentum are grasped at the height of inspiration their ascent upward during expiration can be prevented (Minkowski's expiratory fixation); on the other hand, tumors of the liver and spleen, or tumors of the stomach, if adherent to the liver, must ascend during expiration; *i. e.,* they cannot be retained where they were grasped and felt during inspiration. (*b*) *Chemism.*—(i) Bird (1842) found the *hydrochloric acid reduced,* but Van der Velden (1879) showed the especial frequency of *achlorhydria* in gastric carcinoma. Absence or decrease of hydrochloric acid is not pathognomonic, as it is also absent, permanently or temporarily, in numerous other conditions, as nervous dyspepsia, fevers, cardiac, renal or hepatic disease, pernicious anæmia, cachectic states, gastric atrophy, amyloid degeneration and toxic gastritis. Benjamin Moore states that the hydrochloric acid is absent in 66 per cent. and decreased in 33 per cent. of carcinoma involving other organs than the stomach; the writer has repeatedly seen cases of rectal cancer in which digestive symptoms and achlorhydria have led to an erroneous diagnosis of gastric cancer. In cancer it is due to the secondary *atrophic gastritis.* After administration of an Ewald test breakfast the food is withdrawn in an hour and tested as described under chronic gastritis. If there is no stagnation the total acidity ranges from 6 to 8; in stagnation it is higher from lactic and other organic acids. The absence of free hydrochloric acid is often observed early, while the total acidity remains normal; later the total acidity is decreased. In exceptional cases free acid may persist, both in those cases which develop from peptic ulcer and in those without this antecedent. (ii) The *pepsin* and *rennet* are decreased in 66 per cent. of cases (*v.* Chronic Gastritis). (iii) *Lactic acid,* which is always pathological in appreciable quantities, is present in 75 per cent. of cases and though present in other conditions, as in atony, gastritis and pyloric obstruction, 85 per cent. of the cases in which it is found are gastric cancer. Boas, who developed this topic, considers it a very early finding; while others, as Riegel, hold that it does not develop until later. The factors involved in its formation are somewhat disputed, but depend on the reduced hydrochloric acid, motor insufficiency, stagnation, pyloric obstruction, slow absorption and probably poor digestion of proteids; in the normal stomach it is absorbed as fast as formed (Sticker). As a test meal Boas employs 1.5 per cent. oatmeal to 100 parts of water, given at night after lavage; the contents are removed and tested the next morning; the Uffelmann method is employed as modified by Strauss, for phosphates, alcohol and sugar also give the reaction; 5 c.c. of the gastric filtrate are added to 25 c.c.

PLATE XIV



Oppler-Boas Bacillus in Vomitus. (Musser.)

of ether and shaken in a burette, which is provided with a cock at its lowest part; the lower 25 c.c. are drained away and 25 c.c. of distilled water are added to what remains in the burette (the ethereal extract). Two drops of a 10 per cent. solution of ferric chloride and 20 c.c. of water are added; the blue solution turns to green if lactic acid is present. (*c*) *Microscopically* the long, thread-like *Oppler-Boas bacilli* are found, which are probably the chief source of lactic acid and are called Gram-positive (Plate XIV). The Oppler-Boas bacilli are also found in the fæces in gastric cancer; the bacilli point very strongly to the existence of gastric cancer, but may occur, in the writer's experience, in gastric ulcer and atrophic gastritis (with hypertrophy of the pyloric musculature). Small particles of tumor tissue are seen in rare cases, showing carcinoma structure and irregular mitoses. Sarcinæ are rarely observed. *Occult hemorrhage* (*v.* HÆMATEMESIS), *i. e.,* blood which can be seen only with the microscope or detected chemically or spectroscopically, has the same significance as in gastric ulcer, but is more frequent (over 90 per cent.); pus in the fasting and washed stomach is very important. (*d*) *Motility.*—If the stomach is washed out seven hours after eating there are evidences of stagnation in the undigested food, particularly in pyloric localization; disturbed motility may appear very early in the disease. The stomach is usually much dilated in patients whose appetites persist and little dilated in those with anorexia. If the stomach is empty after seven hours, good motility alone is proven, not good digestion. Every case of stagnation should arouse suspicion of carcinoma.

3. OTHER SYMPTOMS AND COMPLICATIONS.—(*a*) *Cachexia* is something more than malnutrition; it is probably the result of toxins which break down the proteids. The *body weight* is usually greatly *reduced;* in exceptional cases it may temporarily increase, as in cases of pyloric stenosis; it may increase from hope, as in a case of Keen and Stewart, who gained 70 pounds after operation; or when the intestine digests what the stomach can not. The skin itches and is *anæmic* or clay-colored, somewhat resembling the tint it assumes in pernicious anæmia, though distinguished (i) by its later appearance, (ii) by its association with loss of weight, (iii) by the red cells being seldom lower than two million and the hæmoglobin seldom below 40 per cent., though an apparently good count sometimes results from thickening of the blood which is due to lack of absorption; and (iv) by the leukocytosis (see PERNICIOUS ANÆMIA). Schneyer has described the *absence* of the *digestive leukocytosis;* it occurs in about half the cases. As in nephritis or chlorosis (rubra), the face may not show the real anæmia. The wrinkling and wasting of the hands may be far more significant. (*b*) The *urine* is decreased from poor absorption and vomiting, the physiological change in the acidity-curve after eating is absent, the urea is increased from increased toxæmic catabolism, albuminuria is present in 35 per cent. of cases and indicanuria is common. (*c*) *Toxæmic symptoms. Fever* of a low, irregular or intermittent type results in half the cases, from the cancer products,

from septic absorption through the ulcer or from complications, as pleurisy or peritonitis. A coma closely resembling *coma diabeticum* may result from the cancer toxins or the breaking down of the proteids; acetonuria has been noted. Phlebitis and the less common multiple neuritis, tetany and amaurosis are probably toxæmic. (*d*) *Metastases* or *extension by contiguity* may occur. Enlargement of the supraclavicular glands may result from any abdominal carcinoma, but 50 per cent. are due to cancer of the stomach (Troisier). Liver deposits may appear before any suggestive stomach symptoms. Icterus occurs in 5 per cent. (For other metastases *v.* PATHOLOGY.) (*e*) *Ascites* results from the cachexia and, like the swelling of the ankles, appears late in the malady. Some cases are due to carcinomatous peritonitis, the fluid often showing blood and cancer cells, with mitoses (Dock), and others result from glands or tumor compressing the *porta hepatis.* (*f*) *Perforation* may occur (*v.* PATHOLOGY).

Cancer of the cardia presents symptoms like those of œsophageal cancer (*q. v.*). The symptoms may be few or absent. The stomach is never distended. There may be tenderness under the xiphoid. The tumor when advanced may be palpated, as well as the perigastric lymph nodes.

Diagnosis.—Diagnosis is easy in the typical case outlined in the general clinical picture. No single finding, as achlorhydria or even tumor, is pathognomonic; *only the grouping of symptoms and signs is final.* Single analyses of the stomach contents are often deceptive; they should be repeated frequently and considered in connection with other findings. Vomiting in middle or advanced life is suspicious when accompanied by emaciation, anæmia, pruritus and insomnia. With absence of hydrochloric acid and the ferments, the findings of tumor, blood, lactic acid, Oppler-Boas bacilli, dilated stomach, and perhaps of cancer tissue, are positive. The diagnosis may be doubtful, even in the presence of pyloric tumor, emaciation and gastric dilatation, when hydrochloric acid is present; the tumor may then be peptic ulcer, hypertrophy of the pyloric musculature or pyloric spasm due to hyperchlorhydria; in the last instance the tumor is alternately hard and then soft, and often the chyme may be felt or heard as it intermittently squirts through the pylorus. The diagnosis between beginning cancer, nervous dyspepsia and atrophic gastritis must be reserved for a time. *Atrophic gastritis* is more chronic, often lasting years without emaciation. Youth and adolescence do not exclude cancer; in the young, gastric cancer is more rapid in evolution, and hydrops and peritonitis are more common. Abdominal atheroma may closely simulate cancer. *Ulcer* (*q. v.*).

The so-called latent, dyspeptic, gastralgic, anasarcous, ascitic and anæmic forms of cancer, and those causing early metastases (for example, in the liver) and intestinal obstruction, show in what direction error may occur, without due balancing of the individual and combined symptoms.

DIFFERENTIATION.—(*a*) *Pancreatic* tumors (*q. v.*) are deep, fixed,

immobile and disappear on gaseous inflation of the stomach. They may be accompanied by fatty stools and glycosuria. It must be recalled that the normal pancreas is sometimes palpable in meagre subjects. (*b*) *Cancer of the gall-bladder* has respiratory excursion but neither expiratory fixation nor lateral movement; the digestive symptoms, chemism, ectasia and changes on inflation, of gastric cancer are lacking. (*c*) *Duodenal* carcinoma is hard to separate, though the gastric chemism is usually lacking; passage of blood in the fæces and icterus are suggestive. (*d*) *Cancer of the colon* is usually distinguished by the intestinal signs and absence of stomach symptoms or chemism. (*e*) Enlarged *aortic glands* are distinguished by lack of altered chemism, and abdominal *aneurysm* by presence of expansile pulsation; (*f*) *omental* and peritoneal tumors are more diffuse. (*g*) *Hypertrophy of the pyloric muscle* (*v. s.*), felt as a tumor, is less common than is usually assumed; it is usually associated with the findings of ulcer or of hyperacidity; (*h*) foreign bodies (hair tumors), subserous lipoma, fibroma, myxoma and other benign gastric tumors, and palpable round ulcers, are much rarer sources of error. Of sarcoma 87 cases are reported; a dozen were successfully operated on.

Prognosis.—The prognosis unhappily is determined by the diagnosis, an error in which is the only hope. The average duration is one or two years (three months to three years). Death results from exhaustion by pain or vomiting, hemorrhage, gangrene, perforation, portal pressure, intercurrent pneumonia, nephritis and tuberculosis.

Treatment.—1. Medical or Palliative.—(*a*) Condurango bark (Friedreich) seems for a time to increase the appetite and strength. (*b*) *Vomiting* is treated as in gastritis, dilatation and simple ulcer, but with less success; *lavage* is indicated in pyloric stenosis; boric or salicylic acid or resorcinol may be added for fermentation; narcotics by rectum, rectal feeding, champagne and carbolic acid, as outlined before, are also useful. (*c*) Hemorrhage is rarely profuse and usually subsides on rectal feeding; iron, ergot and lead should be avoided. (*d*) The achylia is seldom helped by hydrochloric acid or pepsin. (*e*) *Pain* necessitates the use of fomentations, lavage, chloroform (3 to 5 drops) with ice, and as a last resort atropine or opium by rectum. (*f*) For *constipation,* cathartics are inferior to warm *colonic flushings* or salts, glycerine or olive oil by rectum. (*g*) *Diarrhœa* is lessened by lavage; when due to pyloric insufficiency, salol, betanaphthol (āā gr. x) and opiates are indicated. (*h*) Stenosis of the cardia may yield to dilatation. (*i*) Motor insufficiency, evidenced by food in the stomach seven hours after a test meal, necessitates lavage, which should be practiced before supper. (*j*) The diet should be soft and small in bulk. Meats and fats are digested with difficulty. Vegetables, cereals, small quantities of milk, koumyss, honey, pastries, tea, coffee, diluted wine, peptones, baby foods, somatose and nutrose may be tolerated, but finally the stomach rebels at all food. (*k*) For inoperable cases Jacobi recommends methylene blue, gr. ij daily and increasing to gr. vi,

2. Surgical Treatment.—Cases having good surgical prospects are few, but operation only can cure. When a tumor is found it usually has a rather wide extension; pyloric tumors, for instance, have often reached the pylorus from the lesser curvature (Israel). For resection of the cancer the cases should reach the surgeon, if possible, before the tumor is palpable, a condition possible only in private practice, for hospital cases are always advanced. Metastases, peritonitis and glandular invasion are contra-indications to operation. *Pylorectomy* results in an immediate mortality of 15.6 per cent. as against the original death rate of 56 per cent. in the time of Billroth, who did the first operation (1878). The ultimate outlook is very doubtful, recurrence having been noted after five years. Leriche reports one case well after 16, and Kocher one well after 19 years. The Mayos, in 1906, reported 100 resections of gastric cancer, with a mortality of 14 per cent.; in one group of 25 cases only one case died; 27.7 per cent. of their cases were living three years after operation, and 22 per cent. were well over three years after resection. The Mayo brothers logically plead for an early diagnosis (by exploratory laparotomy) and for early resection. *Gastrostomy* is indicated in cardiac cancer and *gastro-enterostomy* for pyloric obstruction.

HÆMATEMESIS.

Definition.—Hæmatemesis is not absolutely synonymous with gastric hemorrhage; it is the vomiting of blood, which may come directly from the stomach or may reach it from the gums, nose, larynx, lungs, œsophagus, or even regurgitate backward from the intestine. Gastrorrhagia designates profuse hemorrhage from the stomach. It may be arterial, venous or capillary.

Etiology.—(*a*) Various forms of *ulceration,* as simple ulcer, carcinomatous, tuberculous (of which Letorey, 1895, found only 21 cases), diphtheritic, typhoid, syphilitic, uræmic or phlegmonous ulcers, hemorrhagic erosions, and perforations into the stomach from without, as gall-stones, or aneurysm dissecting down from the arch of the aorta (Minkowski), are etiological factors. Parrot described superficial ulcers in the new-born. Dieulafoy describes a pneumococcic hemorrhagic gastritis. Chiari and Murchison, but especially Dieulafoy, have described very small simple ulcers causing profuse, often repeated and fatal hemorrhage; their diagnosis seems probable when they develop in perfect health and with no previous stomach symptoms; they are easily overlooked at operation or autopsy because they involve only the mucosa or perhaps some of the muscularis; some regard this form as the beginning of round ulcer. (*b*) *Trauma,* such as external violence, or internal trauma, as from foreign bodies, thermal and chemical injury, and violent vomiting, as sea-sickness and hyperemesis, may be causal. (*c*) The cause may be *vascular,* such as amyloid degeneration which sometimes involves the stomach and bowels alone; aneurysm, arterial hyperæmia, embolism, vicarious

congestion from suppression of hemorrhoids or menstruation, passive congestion of cardiac or hepatic origin; and œsophageal varix, though it should be remembered that when due to cirrhosis of the liver, there is in 33 per cent. of cases no other evidence of cirrhosis; Welch and Powell have found varices in the floor of peptic ulcers; malarial deposits in the liver; acute pylethrombosis, causing profuse hemorrhage; and parenchymatous oozing, where no postmortem changes are found (Hale White reports 36 cases of what he terms gastrotaxis). (*d*) *Nervous* causes are injuries to the central nervous system, vasomotor influences and hysteria in which the blood is small in amount and occurs usually in the morning vomit; tabetic crises; and general paralysis. (*e*) *Acute infections* may be etiological factors, as malaria, hemorrhagic exanthemata, yellow fever, sepsis, embolism after operations (von Eiselsberg), appendicitis (Dieulafoy) and genitourinary sepsis (Guyon); Busse (1905) collected 96 cases following operations; they result from direct embolism (of which 35 per cent. were in the mesentery or omentum) or retrograde embolism; the hemorrhage occurred in the stomach or bowels; 55 per cent. died. (*f*) *Blood diseases,* as pernicious anæmia, leukæmia, hæmophilia, purpura, scurvy and splenic anæmia, may be causal. (*g*) *Poisons,* which early erode and later ulcerate, as acids and caustics, those causing fatty degeneration of the vessels as arsenic and phosphorus, and uræmia or cholæmia, may cause hæmatemesis.

Most cases occur in *women* between *fifteen and forty* years of age. Of all causes, round ulcer and liver cirrhosis are the first suggested. At autopsy it may be impossible to find the point of issue of blood.

Symptoms.—The symptoms are (*a*) those of the *antecedent disease.* (*b*) In some cases no blood may be vomited up, and the incident remains undiscovered, unless the stools are examined. In *vomitus cruentus* gastric uneasiness is common. The amount vomited varies from microscopic quantities to those sufficiently large to cause death. The occurrence of "*occult hemorrhage*" has been particularly emphasized by Boas; in microscopic examination the diet for days previously should contain no meat, blood, iron or chlorophyll, but should consist solely of eggs, fruit, flour or fat. In profuse gastrorrhagia the blood is bright red, if it remains in the stomach only a short while, as it does in most cases of peptic ulcer. In smaller hemorrhages it is black or rust-colored, as in cancer, uræmia, cholæmia and various poisonings; rust-colored blood may become bright on the edges or surface when it has stood for some time. Blood may issue in the fluid form, or as clots, in which case it is suggestive of valvular disease, vascular diseases or the dangerous hæmatemesis of cirrhosis. In a fatal typhoid hemorrhage, in a medical student, enormous clots were vomited; the author has seen large clots vomited by patients having carcinoma of the stomach. (*c*) *Melæna* may render the stools black, tarry and offensive. (*d*) *Systemic signs.* A gradual or sudden intense *anæmia* may be noted, with all the usual signs of anæmia (*q. v.*), with vertigo, syncope, rapid heart action and some dyspnœa.

Later there may be amaurosis, slight febrile movements, dicrotic pulse, hæmic murmurs, moderate œdema about the ankles, albuminuria and sometimes hemiplegia or convulsions.

Diagnosis.—Differentiation from hæmoptysis (*q. v.*), from the swallowing of blood from the nipples in nurslings or from the vagina in the new-born, fracture of the skull, œsophageal or throat disease and from simulation of blood in the stools from ingestion of bismuth, coffee, tea, claret, iron or blueberries, does not need particular elaboration as the microscopic, chemical and spectroscopic examination determines doubtful cases. *Treatment* is identical with that of the hæmatemesis which occurs in round ulcer.

NEUROSES OF THE STOMACH.

The neuroses of the stomach occur in nervous individuals, either as a part of a neurosis or as a rather isolated nervous symptom. Their relations will also be considered under neurasthenia and hysteria. They sometimes occur reflexly, as in gall or renal stones or in uterine disease. Though their varieties will be considered as (I) secretory, (II) motor and (III) sensory, combinations of symptoms are more frequent than single symptoms.

(I). Secretory Neuroses.

These have been described especially by Reichmann, Rossbach, Sahli and Riegel.

1. Hyperchlorhydria (Hyperaciditas Hydrochlorica).—This is a qualitative anomaly of gastric secretion in which the free hydrochloric acid is increased; it is not solely a neurosis, though conveniently classified as such; it is attended by no known gastric lesion; it is an excessive secretion of hydrochloric acid during the height of digestion.

Etiology.—(*a*) *General* etiological factors are as follows: (i) It is most frequent in young and middle-aged persons; (ii) in the better situated classes; (iii) in Polish Jews and (iv) in brain-workers; (v) it is somewhat more common in women than in men; (vi) a neurotic or psychopathic tendency is a predisposing factor, as is also (vii) chlorosis, in which hyperchlorhydria is found in 85 to 95 per cent. of cases. (*b*) *Local* causes are (i) rapid eating and cold drinks; (ii) condiments; (iii) smoking; (iv) certain foods and coffee; (v) gallstones or possibly renal stones; and (vi) habitual constipation.

Symptoms.—The clinical picture varies much and no single symptom is pathognomonic. (*a*) *Pain,* the chief symptom, varies from a sense of pressure or discomfort in mild cases to extreme gastralgia in severe cases. It develops at the height of gastric digestion—*i. e.,* one to two hours after eating—and is due to the excess of free hydrochloric acid; it depends on the kind and amount of food ingested; the more food, especially meat, ingested the later is the pain, for the

food engages the free acid longer. Carbohydrates cause increased pain as they do not engage the acid and amylolysis is decreased, perhaps suspended; in exceptional cases the patient tolerates small and frequent meals better than larger ones. The patient is often aware of the increased peristalsis and feels a cramping or *spasm of the pylorus.* Pain lasts one-half to several hours. It is *relieved by vomiting* and unless it has become quite severe is also relieved by ingestion of more food or of alkalies. (*b*) *Belching and pyrosis* are frequent and the eructated hyperacid fluid causes a burning in the œsophagus. (*c*) *Vomiting* of a burning sour fluid is not frequent, save in severe cases. (*d*) The appetite is usually good, and most patients desire food frequently, though in small amounts; occasionally it is ravenous. (*e*) *Tenderness* over the stomach is *usually diffuse,* moderate in degree and coincident with the pain; it is sometimes pyloric and is associated with some epigastric distention. *Gastric hyperæsthesia* is as important as the hyperchlorhydria. (*f*) *Stomach tests* show that the total acidity is increased to 100, even to 150 or 160; the digestive power is shown to be increased by the egg-disk test, the proteids are well subdivided, the amylolysis delayed, motility and absorption normal or increased in uncomplicated cases and no lactic acid, no fermentation and no gas are present. (*g*) *Constipation* is more frequent than diarrhœa; *headache* is common and migraine or neurasthenic symptoms may develop; the gums may retract (Herter); in the *urine* the *chlorides are decreased,* the acidity lessened during digestion and frequently an alkaline reaction and phosphaturia are present.

Diagnosis.—The diagnosis is determined by the presence of hyperchlorhydria in the vomitus and test meal, by the relief of the chemical pain by vomiting or ingestion of more food or alkalies and by negative physical findings.

Differentiation.—(*a*) *Ulcer* (*q. v.*) is marked by well-localized tenderness, hæmatemesis or occult bleeding and more symptomatic regularity than in hyperchlorhydria. (*b*) *Gastritis* is characterized by mucus formation and decreased secretion. (*c*) *Gastrosuccorrhœa* or *continuous secretion* (*v. i.*). (*d*) *Gastrectasia* and *gastroptosis* are excluded by percussion and by inflation. (*e*) *Gall-stones,* in which pain has no relation to eating, occurs at longer, irregular intervals and is associated with tenderness of the gall-bladder.

Course and Prognosis.—The disease begins slowly and may disappear, to recur after a few days to months, from irregular living, nervous strain, excesses, certain articles of diet or without obvious cause. The nutrition is fairly well maintained. The outlook is good if early treatment is instituted; complications, as continuous secretion, gall-stones or gastric atony, modify the prognosis.

Treatment.—1. Prophylaxis.—Nervous factors, smoking, haste in eating, chlorosis, etc., should receive appropriate treatment.

2. Diet.—(*a*) A *proteid* diet is most beneficial, for it takes up the largest amount of hydrochloric acid, which is not essentially increased

by proteids, as has been maintained; veal, beefsteak, mutton and ham absorb twice as much hydrochloric acid as equal weights of other proteids (Fleischer); fish fat is particularly to be avoided; milk and some kinds of cheese may be given. (*b*) *Carbohydrates* should be restricted, because the early amylolytic digestion by the ptyalin is delayed or arrested; a slice or two of stale bread, biscuits, toast and zwieback or purées of aleuronat meal, which contains much nitrogenous matter, may be given; Strauss allows six to nine ounces of 2 per cent. grape sugar solution, unless there is a complicating motor insufficiency. Potatoes, salads and most uncooked vegetables should be interdicted. (*c*) *Fats* may be given in limited quantities, including small amounts of cream or unsalted butter (one ounce); one or two drams of olive oil an hour after meals inhibit the secretion of acid. (*d*) *Condiments,* mustard, pepper, vinegar, spices, lemon, horseradish, coffee, alcohol, cold foods and drinks should be forbidden. Weak tea, cocoa or milk may be given. (*e*) *Thorough mastication* must be insisted upon. (*f*) *Small meals,* given at frequent intervals, are generally most efficacious, though Bouveret's three meals may sometimes be given if the patient's appetite is good.

3. Medication.—(*a*) *Alkalies,* as sodium and magnesium carbonates and bismuth, āā gr. xv to xxx, should be given at the height of digestion to neutralize the hyperchlorhydria and relieve its symptoms; Pawlow maintains that alkalies not only act chemically but inhibit secretion by action on the glandular cells. The charged alkaline waters dilute the gastric juice, neutralize the acidity and are sedative. (*b*) Extract of *belladonna,* ¼ to ½ grain, given at meal-time, lessens secretion and mitigates pain; opiates should be avoided. (*c*) *Lavage with silver* nitrate (1 to 1,000) solution is useful in obstinate types.

2. Gastrosuccorrhœa (Hypersecretion, Continuous Secretion).—This is a quantitative perversion of gastric secretion, in which a fairly abundant secretion is found in the fasting stomach; *i. e.,* it occurs without the stimulus of food and when the stomach should be empty. In the lightest forms it only occurs when food is ingested (alimentary form); in the severest forms it occurs without ingestion of food. It is not denied that some gastric juice may be found in the normal stomach, due to secretion initiated by swallowed saliva or mucus, but if this exceeds one ounce it is rarely physiological. Two forms are distinguished, the continuous (Reichmann, 1882) and the intermittent form (Sahli, 1885). (*a*) The *intermittent form* occurs in attacks which last a day or two, is often called acute or nervous dyspepsia and is found in about the same class of patients in whom hyperchlorhydria develops; it has been observed during tabetic crises. *Colic-like pain* develops, *usually at night,* followed by sour *eructations* and *repeated vomiting,* first of food, then of three ounces or more of yellow-green fluid which contains hydrochloric acid and ferments. The first vomitus may contain considerable hydrochloric acid and no more mucus than the small amount which the normal stomach contains; there is seldom blood. During the attack the pulse is small and

rapid, the skin clammy, the urine scanty and alkaline and headache is frequent. Thirst is great and drinking may give temporary relief, but is followed by increased vomiting. Between attacks the acidity is normal and the appetite and general health are usually good; occasionally there is a constant sense of gastric discomfort. (*b*) The second form is *continuous gastrosuccorrhœa.* Its etiology is similar to that of hyperchlorhydria (with which it may co-exist), but rather less clearly established.

Symptoms.—(*a*) *Pain is more marked* than in hyperchlorhydria, develops later after eating and very often at night (11 P. M. to 2 A. M.); it may occur before meals, for the gastric secretion is constant, or in severe cases it may be present both during the day and night; it is relieved by eating and vomiting. There may be local or diffuse tenderness. (*b*) *Vomiting* closely follows the pain and may occur at its height or during the night; the quantity of the vomitus varies; it is large when there is dilatation (a frequent complication and a common cause of this type). It contains hydrochloric acid, ferments and a small quantity of mucus; vomiting may be absent in light cases. (*c*) *Other digestive symptoms.* The condition of the *tongue* varies, being often *red and clean,* sometimes coated. The *teeth* are often *carious.* The appetite is usually good, sometimes ravenous or in advanced cases may be poor. The *thirst is marked* and is sometimes suggestive of diabetes. The *bowels are sluggish.* (*d*) The results of the *test meal and lavage* show that the total acidity ranges up to 100 and free hydrochloric acid to 60. After a test dinner the fluid, when removed, separates into three layers,—the upper foamy, the middle consisting of yellowish fluid and the lower one of sediment of undigested carbohydrates; the proteids are found converted into peptones. When the stomach is washed out thoroughly at night to remove all fluid and food, examination on the next morning of the fasting stomach reveals up to ten ounces of hydrochloric acid and ferments—*i. e., secretion without the stimulus of food;* if the fasting stomach is washed out two or three hours later, gastric juice is again found—*i. e.,* there is continuous secretion when the stomach should be empty. (*e*) The urine is the same as in hyperchlorhydria. Old cases show marked *emaciation,* even cachexia, because of the lax muscles and dry, inelastic skin.

Course and Prognosis.—The onset is slow and the course protracted; it may last for years. The nutrition suffers and death from exhaustion may result in untreated cases. *Complications* are frequent, especially (*a*) dilatation, for the stomach is never empty; the hydrochloric acid does not inhibit the growth of sarcinæ or yeast and gases may generate. The dilatation may be primary but is most often secondary; it is differentiated by thoroughly washing out the stomach at night and when the tube is introduced the next morning it should show nothing in simple dilatation. Dilatation may also result from (*b*) *pyloric spasm.* Pawlow has shown that in health the pylorus relaxes intermittently and allows the acid chyme to reach the duo-

denum; the pylorus is reflexly contracted by the acid fluid in the duodenum until this is neutralized, when it again relaxes. When the secretion is continuous, the passage of acid into the duodenum constantly contracts the pylorus, which may be felt as a tumor. In this case cancer may be suggested, especially when there is emaciation or blood in the stomach, until the constant flow of gastric ferments and hydrochloric acid is found. (*c*) There is some tendency toward gastric ulcer. (*d*) Tetany is a rare complication of pyloric obstruction.

Diagnosis.—The diagnostic features are (*a*) the pain at night; (*b*) vomiting and (*c*) thirst, both of which are more common than in hyperchlorhydria; (*d*) continuous secretion after washing out the stomach and during fasting.

Treatment.—Treatment is in general (*a*) that of hyperchlorhydria as to prophylaxis, diet, mastication and stimulants. (*b*) *Rectal feeding* is a valuable accessory; water is given by rectum to quench the raging thirst. (*c*) *Oral feeding* is allowed at long intervals; overloading the stomach with fluids is carefully avoided. (*d*) Lavage relieves pain and hypersecretion. Reichmann employs irrigation with silver nitrate (1 to 500 or 1,000). (*e*) *Olive oil* lessens hyperchlorhydria (Riegel). (*f*) *Alkalies* and belladonna are indicated as in hyperchlorhydria. (*g*) Treatment of *coincident dilatation;* gastroenterostomy may be indicated by pyloric obstruction, never by hypersecretion *per se.*

3. Hyposecretion.—Hypacidity (hypochlorhydria or subacidity) and anacidity (achlorhydria) refer respectively to reduction or absence of hydrochloric acid, which may occur from nervous as well as from organic causes (Achylia). When the ferments and hydrochloric acid are absent, achylia gastrica nervosa is a more appropriate term; the condition may be transient or enduring and Einhorn reported a case in which secretion returned after an absence of five years. Suppressed secretion may occur in hysteria or tabetic crises.

(II.) Motor Neuroses of the Stomach.

These may be irritative (increased motility) or depressive (decreased motility).

1. Irritative Types.—(*a*) Hyperkinesis or *hypermotility* hurries the food into the intestine, so that it disappears in three to five hours or even less. It exists by itself as a neurosis, but may occur in the hasty digestion of hyperchlorhydria and in gastric achylia, in order that the intestines may earlier digest the unaltered food. (*b*) *Peristaltic unrest* (Kussmaul) or *tormina ventriculi nervosa* occurs particularly in neurasthenia and in organic pyloric stenosis. In some cases the unrest may be wholly subjective, the patient experiencing a rolling or cramping sensation. In others, both neurotic and organic, the peristaltic movements from left to right may be seen, as well as antiperistalsis in the opposite direction. The treatment of hyper-

kinesis and peristaltic unrest is that of the underlying neurosis. (*c*) *Pyloric spasm* occurs very seldom as an independent neurosis; it occurs usually in connection with ulcer, hyperacidity, hypersecretion, erosions, chronic stenosing gastritis, sometimes with cancer of the stomach and poor mastication of food and drinking of cold fluids. Its relation to dilatation has been discussed. (*d*) *Spasm of the cardia* occurs in organic disease of the œsophagus and stomach (ulcer, cancer, erosions, hyperchlorhydria) or in neuroses; it is the cause of idiopathic dilatation of the œsophagus (*q. v.*), and the spasm in tetanus and hydrophobia. It makes vomiting difficult and is differentiated from organic stricture by the fact that in cardiospasm a large sound passes more easily than a small one, and that spasm is often intermittent. The treatment of pyloric spasm is that of the cause (almost invariably gastric); the treatment of cardiospasm is discussed under idiopathic œsophageal dilatation. (*e*) *Nervous eructations* (Bouveret, Mathieu, Follet, Stern) occur in various neuroses and psychoses. They occur especially in sexual neuroses. The odorless *ructus* does not result from fermentation, but from swallowing or air (aërophagia), which constitutes a most intractable habit. The mechanism by which air is swallowed is disputed; Bouveret correctly thinks it is forced down by pharyngeal contractions. It is swallowed unconsciously or again it enters during what Matthieu terms false eructations, in which the patient, in attempting to raise gas, swallows air. The ructus must be caused by aërophagia, as such large quantities could never generate in the stomach; Bardet estimated that one patient raised twenty quarts in an afternoon. Coincident cardiac and pyloric spasm causes distention (pneumatosis). Treatment lies in the use of the sound, suggestive therapy and in the difficult task of breaking the habit; asafœtida is valuable as the ructus becomes offensive. (*f*) *Nervous vomiting* occurs (i) only in the neuroses, as hysteria, neurasthenia, migraine and exophthalmic goitre; but under this caption we may also bring for differential purposes (ii) cerebrospinal conditions, as compression of the brain, meningitis, tumor, tabes, general paralysis, disseminated sclerosis and myelitis; toxic conditions, as uræmia or cholæmia, may cause cerebral vomiting. (iii) Most cases are *reflex* in origin; they originate in the nose, as nausea from disagreeable odors; in the respiratory tract; in pelvic or abdominal conditions, as pregnancy, menstruation, gall-stones, renal calculi and peritonitis. The symptoms may present many incompatible elements; thus the patient may vomit without nausea, almost without effort, so that the vomiting is rather an eructation; it often occurs without pain or without reference to the fulness or emptiness of the stomach; even though it is frequent, the nutrition may be remarkably good, the tongue clean and the appetite good. Periodical or cyclical vomiting, described by Leyden, has some resemblance to tabetic crises; Edsall considers it an acidosis, for which ʒss to ij of sodium bicarbonate is indicated. Robert's conclusions are as follows:

1. Vomiting of a chronic type, following a gradual epigastralgia, from one-half to three hours after eating, is attributable to gastric or duodenal ulcer.

2. Chronic vomiting, from ten to fifteen minutes after food ingestion is attributable to stenosis of the cardia, nervous abnormality, cerebral lesion or acute gastritis.

3. Copious vomiting ten or more hours after eating indicates muscular insufficiency; frequent repetition indicates a permanent mechanical lesion interfering with the exit of food.

4. Vomiting in the night occurs in cholelithiasis, periodic hypersecretion, muscular insufficiency and nervous abnormality.

5. Vomiting attempts when the stomach is empty indicate a cause other than a gastric lesion, either a reflex cause, a toxæmia, a cerebral lesion or a nervous abnormality.

6. Morning nausea and retching indicate pregnancy, alcoholism, pharyngitis, nephritis or a nervous abnormality.

7. Periodic vomiting of clear gastric juice of normal or supernormal acidity in considerable amounts indicates a secretory neurosis or an ulcerative lesion.

8. Vomiting as a sequel of headache, accompanied by severe nausea, but no gastric or abdominal symptoms, characterizes attacks of migraine; in a majority of cases eyestrain is the underlying cause.

9. Attacks of sudden vomiting with tinnitus, deafness and vertigo are attributable to disturbances of pressure in the internal or middle ear.

10. Periodic sudden vomiting, with gastric pain and nausea, retraction of the abdomen, obstinate constipation during but not preceding the attack and freedom from abdominal tenderness are suggestive of the spinal crises, idiopathic nervous vomiting and lead-poisoning.

11. Periodic attacks of vomiting with colic, constipation and tympany suggest chronic intestinal stenosis.

2. Depressive Motor Neuroses (Lessened Motility).—There are three types: (*a*) *Atony,* which has been considered under dilatation; (*b*) *pyloric insufficiency* (Séré and Ebstein), which is seldom a neurotic condition, as compared with its frequency in cancer, ulcer and dilatation of the pylorus from duodenal obstruction. Its presence is determined by inflating the stomach with carbon dioxide, which rapidly passes into the intestine with a gurgling gush. (*c*) *Insufficiency of the cardia* causes *regurgitation* of food and fluid and rumination (*merycismus*), which is a regurgitation in which the food is again chewed and reswallowed. These are generally involuntary processes, but some subjects can regurgitate at will. The patients are nearly always neurotic males, psychopaths or idiots. The affection is either hereditary or imitative when it occurs in several members of a family. Its mechanism is disputed; congenital weakness of the cardia, stimulation of the vagus and aspiration into the œsophagus by the inspiratory act are considered causal. It should be treated by giving small

meals at frequent intervals; these should be well masticated; intragastric electrical applications and suggestive measures are indicated.

(III.) Sensory Neuroses of the Stomach.

1. Hyperæsthesia.—Hyperæsthesia may give the sensation of burning in the stomach or a sense of weight; this usually occurs when the organ is full, but at times also when it is empty. Carbohydrates, fats or spices may induce the condition. Silver nitrate, gr. ¼ to ½, phenol, gr. ¼ to j, or fluidextr. gelsemii ♏ij given on the empty stomach, often affords relief, besides which the basic neurasthenia should receive treatment.

2. Gastralgia.—Gastralgia (gastrodynia or cardialgia, which is a poor but widely used term) does not strictly include the gastric pain due to ulcer, hyperacidity or hypersecretion. Rosenthal's etiological classification is as follows: (i) Cerebrospinal causes, including various brain, cord (tabes), peripheral and functional nervous affections. (ii) Constitutional causes, including chlorosis and debility. (iii) Toxic causes, resulting from gout, rheumatism, malaria, nicotine and constipation. (iv) Reflex causes, from the genito-urinary and alimentary tracts; floating kidney, intermittent hydronephrosis, splanchnoptosis, uterine or ovarian disease or masturbation. Fliess holds that many cases of dysmenorrhœa and gastralgia arise from disease of the lower nasal turbinates. Most cases occur in women between fifteen and forty-five years of age.

Symptoms.—As the term implies, the affection occurs with severe gastric pain which begins suddenly, often at night, lasts for a few minutes to an hour or so and subsides usually to recur after a variable interval. The pain is epigastric and may radiate widely. It bears no relation to eating, though food sometimes relieves it. Diffuse superficial tenderness or hyperæsthesia over the stomach is not infrequent, but deeper pressure usually affords some relief. Vomiting is rather uncommon; hiccough, hunger, headache, depression, polyuria, clammy sweats, small rapid pulse or even collapse may attend the seizure.

Diagnosis.—A diagnosis is made only by exclusion and usually after the seizure is over. Rheumatic myalgia is more constant. A tabetic crisis is at once recognized by the Argyll-Robertson pupil and the absent knee-jerks. Intercostal neuralgia gives Valleix's three tender points which, however, are quite frequent in neurotic subjects. In ulcer (*q. v.*) or circumscribed peritonitis the pain is associated with localized tenderness. Hyperacidity and hypersecretion should be excluded. Biliary colic (*q. v.*) is more difficult to differentiate, but a sharp thrust over the gall-bladder during deep inspiration nearly always elicits well-localized tenderness.

Treatment.—Treatment is that of (*a*) the etiology, especially the neurasthenic condition, anæmia and constipation. Fowler's solution ♏ij before meals is excellent. (*b*) The attack. Morphine hypodermatically affords the only certain immediate relief, but in these

very cases may establish the habit. If given at all, it should be given by mouth without the patient's knowledge. Heat and sinapisms may relieve or aggravate the attack. Silver, phenol or gelsemium employed in hyperæsthesia often gives relief, as may extract of belladonna ½ grain, charged waters, chloroform and Hoffmann's anodyne āā ♏xx to xxx and galvanization with the anode to the epigastrium and cathode to the spine, though the attack is often over before application is possible.

3. Disturbances in the Sense of Hunger and Appetite.—Loss of appetite (*anorexia*) may be purely nervous, though more commonly due to gastric affections, fevers, tuberculosis or toxins. As described under Hysteria, anorexia nervosa may be absolute and cause profound emaciation, as in the extreme cases of Lasègue, Wallet and Brissaud. *Parorexia* is perversion of the appetite, in which the patient craves for unusual kinds of food. In *polyphagia* the appetite is enormous, but is capable of satiation; in boulimia (*hyperorexia*) the intense craving for large quantities of food comes on paroxysmally, sometimes with vertigo, palpitation and other neurotic manifestations; polyphagia and boulimia are most frequent in hyperchlorhydria, hypersecretion and hypermotility, but also occur in diabetes, focal brain disease or tumors, intestinal parasites or from quinine, orexin or even opium. *Acoria* is loss of the sense of satiation. These anomalies are central and probably result from organic or functional changes in the bulbar hunger centre. The treatment of the neurotic forms is that of the causal neurosis.

(IV.) Mixed Neuroses of the Stomach.

Mixed neuroses, dyspepsia nervosa (Leube), neurasthenia gastrica (Ewald) or pseudodyspepsia, is marked by no stereotyped symptoms. It may occur alone as a species of local neurosis; it is sometimes reflex from other organs, but is much oftener a part of the neurasthenic symptom-complex. Leube's original contention that digestion itself is normal must be modified, as the hydrochloric acid may be increased, normal or decreased, but perhaps in the majority of cases the gastric functions are nearly or quite normal. Two main groups of symptoms prevail; (*a*) the *centric,* neurasthenic or hysteric apathy, irritability and vertigo; (*b*) the *gastric* eructations, variations in appetite, oppression and diffuse tenderness, which are probably also cerebral in origin. The symptoms vary greatly in degree, often change their form with considerable rapidity and the general nutrition is generally good. Organic disease must be carefully excluded by repeated examinations of the secretions and motility of the stomach, for nervous dyspepsia is too often "a collective term to cover incorrect diagnoses." The treatment is that of the neurotic basis; rest, regulation of worry and strain, self-control and suggestion are important.

DISEASES OF THE INTESTINES.

ACUTE ENTERITIS.

It is difficult to draw a close distinction between acute catarrhal enteritis, intestinal dyspepsia and simple diarrhœa. Catarrhal enteritis is the most common intestinal disease.

Etiology.—1. Primary Forms.—(*a*) Errors in *diet* and the ingestion of spoiled food containing ptomaines, unripe fruit and impure water are common causes. (*b*) Disturbance in intestinal secretion may possibly occasion acute enteritis, though little is known of the succus entericus. (*c*) Cold, trauma, foreign bodies, fæcal retention and toxins eliminated from the blood may initiate inflammation. (*d*) Some cases are *infectious,* though our knowledge of them is still imperfect. Bacteria are undoubtedly an important factor; it is stated that one-third of the weight of the normal stool is caused by bacteria, of which 48 species are normally present; acute inflammation may arise from pathogenic organisms or from virulence attained by some inhabitant of the intestine; the Bac. coli is the most important and Gärtner's Bacillus enteritidis is probably one of its manifold forms. In a milk diet the Bacterium lactis aërogenes and in a meat diet the Proteus vulgaris may be possible factors.

2. Symptomatic Forms.—(*a*) Enteritis is constant in some infections, from the action of microörganisms or their toxins, as in typhoid; it is frequent in others, as in sepsis or influenza; and rare in others, as in measles, scarlatina, rheumatism or malaria. (*b*) General diseases, as nephritis; (*c*) burns; (*d*) local intestinal diseases, as intestinal parasites, ulcers or tumors; and (*e*) metallic or other poisons are etiological factors; (*f*) stasis is usually listed as a factor, but is rarely by itself a potent cause.

Pathology.—The pathology is that of other inflammations of the mucous membranes; *viz.,* redness, swelling, increased mucus secretion, parenchymatous degeneration and some interstitial infiltration with leukocytes. In the few cases coming to autopsy these changes may not be at all conspicuous, as they regress soon after death. In some cases follicular swelling or even ulceration may be observed, as well as punctate ecchymoses, catarrhal erosions or slight swelling of the mesenteric glands.

Symptoms.—The chief symptom is *diarrhœa,* due to increased peristalsis, increased secretion or actual inflammatory exudation. The *stools* number two to fifteen daily and contain more or less mucus; they may be brown-yellow with moderately frequent, or colorless and odorless with very frequent evacuations; their reaction is commonly alkaline or neutral; they contain triple phosphates, calcium phosphate and oxalate crystals, sometimes cholesterin and Charcot-Leyden crystals, but rarely leukocytes in any considerable number. There may be concomitant acute gastritis. Thirst, dryness of the mouth, colic pains, borborygmus, visible peristaltic movements, tym-

pany and slopping or gurgling in the intestine are frequent. Fever is unusual, though febrile forms with acute splenic tumor are recorded, which may at first resemble typhoid. Albuminuria, cylindruria or even acute nephritis may develop in exceptional cases; the urine is scanty in proportion to the amount of fluid lost by the bowel; indican, ethereal sulphates and acetone may be present. In some few cases herpes or muscle and joint pains occur. Collapse is rare in adults, though cases occur which bear a close resemblance to cholera. In the vast majority of cases convalescence is established in a few days.

Diagnosis.—(*a*) *Duodenitis* can seldom be diagnosticated; pain and tenderness over the duodenum are of most uncertain, but catarrhal icterus is of somewhat presumptive diagnostic value. (*b*) *Jejuno-ileitis* seldom exists alone; diarrhœa is absent if the colon is not affected; the stools contain intimately mixed mucus, which often appears as small hyaline globules under the microscope (Nothnagel). (*c*) In *enterocolitis* the bile is decomposed below the small intestine; when it is present in the stools it signifies increased peristalsis which moves the bile along before it can be disintegrated and indicates acute enterocolitis; bile may be found only in the small globules of mucus and not in the fluid evacuation. Many *undigested food particles* (lientery) are found, and this is rather significant if fever, biliary or pancreatic obstruction and gastric atrophy are excluded; they consist of undigested meat, starch granules (which are more significant than muscle fibers) and fat particles. (*d*) *Colitis* is characterized by the absence of bile and lientery, by thin stools and by more or less mucus, which is obtained especially on washing out the bowel immediately after a movement (Boas). (*e*) *Proctitis* is marked by much tenesmus, dysuria, evacuation of mucus alone and the passage of bloody mucus covering but not mixed with the solid stools; it is also diagnosticated by the local examination with the finger or speculum.

Treatment.—(*a*) In the early stage *evacuation* of the causal irritant is indicated, to which end fractional doses of calomel should be given, followed by castor oil; the stronger evacuants, which irritate or inflame the intestine, should be avoided. Colonic flushings with pure water or injections of olive oil may be added. (*b*) *Absolute rest* in bed is always indicated. (*c*) The *diet* should be restricted to barley water, or no food should be given for a day or two. (*d*) *Heat* should be applied to the abdomen. (*e*) A dram each of *paregoric* and *bismuth* should be given after each bowel movement until the number is controlled. A quarter of a grain of extract of belladonna relieves colic. The following preparation is excellent; the amount for a child being given in the first and the amount for an adult in the second column:

℞	Phenylis salicylatis (salol)	gr. xx.	ʒj.
	Sodii bicarbonatis	ʒss.	ʒj.
	Cretæ preparatæ	ʒss.	ʒiss.
	Bismuthi subnitratis	ʒj.	ʒj.

M. et divide in pulveres X.

S.—One powder in milk or thin arrowroot solution every three hours.

Lack of tone is relieved by gambir (catechu), colic by belladonna and flatulency by ammonia and ginger:

℞ Tincturæ gambir compositæ ..... ℥j.
Fluidextracti belladonnæ ..... gtt. xij.
Spiritus ammoniæ aromatici ..... ℥ij.
Tincturæ kino ..... ℥j.
Syrupi zingiberis ..... ℥ss.
Misturæ cretæ .....q.s. ad ℥iv.
M. et S.—A tablespoonful every two or three hours.

Other astringents and intestinal antiseptics, as betanaphthol gr. x p. c. in capsules, or 10 to 15 grains of tannigen (a compound of acetic acid and tannin) every two or three hours are seldom indicated. (*f*) No laxatives should be given for the constipation resulting from this treatment. (*g*) Opium suppositories (see DYSENTERY) relieve *tenesmus*. (*h*) The diet may be increased in a few days to bouillon with raw egg, hashed beef or chicken, toast, zwieback, roast beef and milk with equal parts of carbonated water; fruits and vegetables even when cooked should be given only with extreme caution, because recurrence readily predisposes to chronic enteritis. (*i*) Cramps, collapse and other choleraic symptoms are treated as in cholera (*q. v.*).

CHRONIC ENTERITIS.

Etiology and Pathology.—The etiology is essentially that of acute enteritis, from repeated attacks of which, or of "acute indigestion," the chronic type may develop.

Chronic enteritis always suggests a causative chronic gastritis, though both affections may result from overeating and drinking. Chronic enteritis may follow chronic gastritis, achylia gastrica and other gastric conditions; or intestinal catarrh may cause anorexia, eructations, abdominal distention and kindred symptoms. Hyperchlorhydria may disturb the intestines by the escape into them of more acid than they can neutralize; on the other hand hypo- or achlorhydria readily initiates bowel trouble because hydrochloric acid bears an important relation to the intestinal and pancreatic secretions.

The pylorus remains closed until the food in the stomach is ready for the intestinal juices and then it relaxes; the hydrochloric acid, reaching the duodenum, provokes a flow of bile (which arrests the action of the pepsin), and the acid also provokes the pancreatic secretion (which neutralizes the hydrochloric acid) by acting upon the duodenal mucosa, which gives off a substance known as "secretin," into the blood; secretin acting upon the pancreas causes it to secrete (Bayliss and Starling). When these ends are accomplished the pylorus again relaxes. When the pancreatic juice is poured out it contains no active proteolytic ferment, but in the succus entericus there is an "activating" ferment (enterokinase) which converts trypsinogen into trypsin; *i. e.,* the intestinal juice "activates" the trypsinogen (Pawlow).

The bile doubtless increases the activity of the pancreatic enzymes; it activates the fat-splitting pancreatic ferment.

The pathology of chronic enteritis resembles that of the acute type. The mucous membrane is brown-red or slate-gray. The mucosa and submucosa are often thickened and the muscularis is sometimes hypertrophied. Polypoid hyperplasia and cystic degeneration of the intestinal glands as well as atrophic changes may result. Catarrhal erosions and follicular ulcers may occur as described under the last type of dysentery (*q. v.*).

Symptoms.—Generally speaking, *constipation* is more frequent than diarrhœa; in comparatively few cases a single soft morning evacuation is noted, or perhaps several; in others constipation is observed most of the time, broken occasionally by short attacks of colic and diarrhœa; continuous diarrhœa indicates inflammation of both the large and small gut. Constipation is due to alteration of the nerve fibers in the gut or to inflammation in the muscular coat. A sense of abdominal tension or colic may be present and is distinguished from gastric discomfort and pyloric spasm in that it occurs independently of ingestion of food. Diarrhœa results from decomposition and fæcal irritation. There may be pain, flatulency, borborygmus or occasionally tenderness over the bowels, which may occur together or at intervals; but the sole diagnostic criterion is the presence of *mucus* in the passages; an intimate mixture with the stool indicates inflammation in the small gut; a mere coating of the passages with mucus indicates colitis, and evacuations of mucopus alone, proctitis. Small sago-like globules may be mucus or vegetable matter. Mucus globules when bile-stained indicate inflammation of the small gut, as do shreds of bile-stained mucus. Small homogeneous denucleated epithelial cells may be found in the mucus adhering to scybala. Pus cells are rare except in proctitis. The presence of *mucus is always indicative of enteritis* and never occurs from ulceration or carcinoma alone (a slight coating of mucus over hard fæces is not significant). *Blood always indicates a complication,* such as piles, ulceration or cancer. The stools may contain much *undigested meat, starch* or *fat.*

Schmidt and Strasburger have proposed the *functional examination* of the fæces; while their method does not give us as exact information as does the examination of the stomach contents, valuable facts may be determined. Schmidt's test diet includes 3 pints of milk, 3 ounces of zwieback or toast, two eggs, 1.6 ounces of butter, one-quarter pound of rare tender steak,6 ounces of boiled potatoes, 2 ounces of oat-meal and two-thirds of an ounce of sugar; this total is variously distributed among the three meals. With the first meal a capsule of five grains of charcoal or carmine is given and the first examination is not made until the stools become colored black or red. In this way (*i*) the "period of passage" is established, the normal time being ten to twenty hours. (*ii*) Mucus is detected, microscopically, by mixing a piece of fæces as large as a walnut with a small amount of water; it appears as translucent, sago-like and often bile-stained flakes, which

must not be confused with starch particles. (*iii*) Much connective tissue, recognized by its yellow-white color and its toughness, indicates reduction of the hydrochloric acid. (*iv*) Muscle fibers, appearing as reddish threads or lumps, may indicate achylia gastrica, lack of trypsin, lack of the activating enterokinase or very active peristalsis. (*v*) Free starch granules always indicate intestinal catarrh; normally only starch enveloped in cellulose is present. (*vi*) The normal stool shows hydrobilirubin, detected by mixing a small amount of the fluid fæces with an equal amount of a 5 per cent. solution of bichloride of mercury, which mixture turns yellow-red after twenty-four hours; bilirubin, recognized by a green color in the above test, always indicates catarrh of the small bowel (except in young children). (*vii*) The fermentation test is made by mixing a dram of the fæces with sterile water in a bottle which is connected above with a tube containing water; almost no gas results in normal cases; if the reaction becomes acid with a pathological fermentation it is due to carbohydrate fermentation; if it becomes alkaline and foul in odor it is due to proteid fermentation. (*viii*) With a little experience with the amount of fat normally found it can be readily determined whether the fat is pathologically increased.

The appetite is usually good. The general nutrition is good in some cases, though in others pallor, emaciation, slow pulse and cold extremities may be noted. Attacks of diarrhœa and undigested food in the stools suggest achylia gastrica (Einhorn, Oppler).

Prognosis.—The prognosis, save in either extreme of age, is good as to life itself, but is always doubtful at any age as to complete recovery. Improvement at the best is tardy and often transient. Intestinal atrophy may develop, though this is less frequent than in puerile forms.

Treatment.—(*a*) Moderate exercise and an *hygienic* conduct of life are most essential. A flannel band should be worn over the abdomen. (*b*) The *diet* must be carefully supervised. Though it is impossible to give here a complete dietary, salads, beer, wine, fruits and vegetables of nearly every description, either raw or cooked, sweets, pickles and acids are among the forbidden foods; eggs, fresh butter, lean meats, stale bread, rusks, thoroughly cooked rice or sago and potato purée are permissible; *i. e., simple foods,* in *small amounts* and at *frequent intervals,* controlled by frequent examination of the fæces. (*c*) Regulation of the *bowels* is necessary. For constipation, purgatives should be avoided and the bowels moved by *enemata of olive oil.* Constipation is often due to the careful diet, which does not provoke peristalsis. For diarrhœa, bismuth in dram doses and tannigen (5 to 15 grains) are most efficacious (see ACUTE ENTERITIS). Calcium carbonate and salicylate are recommended by Boas and Javorski (āā 3ss to a quart of charged water; one-half glass q. i. d.). (*d*) *Flatulency*—Some flatus is not pathological. As it results from stagnation plus catarrh (chiefly in the ileum, cæcum and ascending colon), the first indication is that of the catarrhal enteritis;

the second is the restriction of foods which ferment (*v. s.*); and the third is the administration of aromatic remedies (see THERAPY OF ACUTE ENTERITIS).

℞ Mentholis ..... 3ss.
Phenylis salicylatis ..... 3iij.
Extracti belladonnæ ..... gr. iv.
M. et ft. capsulæ XX.
S.—One after meals.

(*e*) *Colitis* is treated by rectal flushings, 1 to 2,000 silver nitrate, borax, salicylic acid or ichthyol. (See TREATMENT OF DYSENTERY.)

ENTERITIS (COLITIS) MUCOSA OR MEMBRANACEA.

This disease was first described by Mason Good (1825), though the first classical description was Da Costa's in 1871. The name implies that the malady is an inflammation, but in reality most cases are neither enteritis nor colitis. Nothnagel proposed the name *colica mucosa* for the non-inflammatory variety, which is a secretory neurosis.

Etiology.—(*a*) Eighty to 90 per cent. occur in *nervous* or *hysterical women* between twenty and forty years old, 40 per cent. of whom have uterine troubles; males who are affected are usually nervous or hypochondriacal; a few cases have been seen in nervous children. (*b*) Nearly all cases suffer from years of previous *constipation.* Its pathology is unknown; in some autopsies no anatomical alteration is noted, in others that of enteritis.

Symptoms.—(*a*) *Mucus casts* of the bowels, shreds or lumps are voided periodically; the mucus may resemble a huge diphtheritic cast, is usually evacuated in large amounts, which often constitutes the entire bowel movement and contains very few leukocytes or other signs of inflammation; it is rarely simulated by curds of milk. (*b*) There is usually, but not always, *colic* or pain and tenderness with the attack, which frequently occurs over the splenic flexure of the colon, is sometimes diffuse and occasionally radiates, as into the leg. If the mucus is firmly adherent to the bowel wall the pain is particularly severe and obstruction of the bowels may be simulated very closely. *Tenesmus* in voiding the mucus is usual, as are formation of gas and *increase* of the original *nervous symptoms,* as palpitation, tremor or vertigo. There may also be coincident diarrhœa, achylia gastrica and a periodic sand formation (Dieulafoy). (*c*) *Constipation,* abdominal distention or pressure and anorexia exist between the paroxysms, which last a day or even a week and are separated by intervals of weeks or months. It is said that death may occur during an attack.

Treatment.—Treatment concerns (*a*) the *fundamental neurosis.* As a rule the less the neurotic symptoms are in evidence the better are the results of therapy; education, the rest cure and other measures outlined in the treatment of hysteria and neurasthenia (*q. v.*)

are indicated. (*b*) *Constipation* (*v. i.*) is treated in the usual way and an appropriate diet is essential. Von Noorden advises the use of graham bread, leguminous vegetables, including the husks, vegetables with much cellulose—which seems to be the most important element—fruits with small seeds and thick skins, as gooseberries, currants or grapes, large amounts of cream, butter and bacon, and olive oil. Von Noorden's results with this diet are: 79 per cent. cured (15.8 per cent. partially cured, 50 per cent. permanently cured, 13 per cent. relapsed), 15.8 per cent. unknown result and 5.2 per cent. failure. In White's series 50 per cent. recovered, 13 per cent. improved and 37 per cent. were not improved. (*c*) Local measures include *colonic flushings* with normal salt solution, or half a pint of olive oil left in the rectum over night (Kussmaul). (*d*) *Narcotics, purgatives,* irrigation with alum, tannin and silver solutions, and operation are distinctly *contra-indicated.* The morphine habit may be readily contracted. Belladonna in a suppository is very efficient in types characterized by pain. The patient in an attack is kept in bed and heat is applied to the abdomen.

DIPHTHERITIC, CROUPOUS AND PHLEGMONOUS ENTERITIS.

True diphtheria, due to the Klebs-Loeffler bacillus, is very rarely encountered, and practically all membranous enteritis is *diphtheroid;* it develops in various acute infections, in uræmia and in metallic poisoning and has been described under the third form of dysentery.

Phlegmonous or *purulent enteritis* is likewise extremely rare and chiefly of anatomical interest. Most cases develop in the duodenum. The affection may be primary, but more often is secondary to intestinal ulceration, carcinoma or intussusception. Maragliano described a form probably due to the colon bacillus, in which multiple purulent foci are found in the intestinal wall, which frequently cause perforation.

INTESTINAL ULCERATION.

I. Duodenal Peptic Ulcer.—This is almost identical in etiology, pathology and treatment with the peptic gastric ulcer. It was first described by Abercrombie (1824).

Etiology.—(*a*) *Gastric ulcer* is 2 to 10 times as frequent as duodenal ulcer, whose frequency Kinnicutt estimates at $\frac{4}{10}$ per cent. in 30,000 autopsies. In 121 duodenal ulcers collected by Oppenheimer, gastric ulcer was present in about 6 per cent., and in Lebert's series of 237 stomach ulcers, duodenal ulcer was present in about 10 per cent. Moynihan and Mayo found gastric ulcer in about 50 per cent. Forty-nine cases of jejunal ulcer are reported after gastro-enterostomy for round ulcer of the stomach (*q. v.*); ulceration may be obviated by alkalies after operation. (*b*) Seventy-nine per cent. occur in *males* as a result of alcohol. (*c*) In Collin's series of 279 cases, two-thirds occurred between twenty and sixty years of age, being

about equally frequent in each decade. (*d*) Other etiological factors are: (i) in extensive *burns* (Curling, 1842) acute ulcers develop in one to two weeks, rarely earlier or later; Holmes found them in 13 per cent. of 125 fatal burns; their cause is probably vascular obstruction, though the precise factors are not yet established (fibrin-ferment, toxins absorbed from the skin, aggregation of blood plaques). (ii) *Nephritis,* which occurs in 17 per cent. of the cases, is suggested as a cause by Perry and Shaw. (iii) It may result from *embolism* and thrombosis. Endocarditis and sepsis occasionally cause duodenal ulcer. Thrombosis of the *umbilical* and other veins produces the cases seen in the new-born (Landau). It has also followed trauma, multiple neuritis, periarteritis nodosa and foreign bodies such as gall-stones. *Pathologically* the resemblance to gastric ulcer is marked in all details. It is *single* in 84 per cent., is located in the *upper horizontal part* of the duodenum in 87 per cent. of cases and is almost always above the papilla.

Symptoms.—*Symptoms are absent* in 30 to 60 per cent., at least until hemorrhage or perforation occurs. (*a*) It is generally stated that *gastric symptoms* are absent in the great majority of cases. C. Graham, in an analysis of 163 cases, found stomach symptoms, especially vomiting and gas formation, in 70 per cent. The appetite is usually good. Graham found superacidity in half the cases. It must be remembered that associated gastric ulcer is quite frequent. Moynihan describes the symptoms of duodenal ulcer as follows: "The patient tells you that he has certain definite attacks, and if you take the history given in detail, letting the man tell his own story, he will give you the impression of having read something which has been written about duodenal ulcer, which he is recounting to the best of his recollection to please you. He says that his trouble comes on in attacks which are nearly always worse in winter than in summer and are very apt to be precipitated by a chill. Let us follow the patient through the day. He takes a meal at eight in the morning and from two to two and one-half hours after it he is fairly comfortable; it is his best time. At the end of that time he has a feeling of discomfort in the epigastrium; he feels full and heavy and may get some relief from the belching of gas. Some of these patients develop a habit of belching. They may bring up a very sour fluid, which tastes very bitter and acid and makes the mouth dry and the teeth chalky. This pain gradually increases until the next meal time comes. To this I some years ago applied the name of 'hungry pain.' At the next meal the patient almost instantly gets relief and that relief persists for two or three hours again. He probably eats a heavy dinner and will nearly always tell you he has something before he gets into bed, a glass of milk or a cup of cocoa and a biscuit. He sleeps comfortably, until he wakes about 2 A. M. He gets relief from nibbling a biscuit, which he keeps at the bedside. The pain is found to be most relievable by something stodgy and indigestible. Taking an alkali relieves the pain; so will emptying the stomach by washing

it out. If these symptoms which I have described are recurrent you can diagnose duodenal ulcer." In one of the writer's cases of gastric ulcer in which there was anacidity, Murphy found at operation a gastric ulcer and also a greatly dilated duodenum; in the jejunum there was marked stenosis due to a healed round ulcer, as shown by microscopic examination. This case is, as far as we know, quite unique; Tobieson reports a jejunal ulcer following burns. (*b*) *Pain* is present in 80 per cent., occurs 2 to 5 hours after eating, is located over the duodenum and is sometimes accompanied by *tenderness.* Both pain and tenderness may be peritonitic. (*c*) *Hemorrhage* from the bowels occurs in 33 per cent. of cases. The stools are usually tarry, but sometimes bright red from copious bleeding. "Occult" hemorrhage (small or microscopic hemorrhage) is even more common and is as important as in gastric ulcer or cancer. Intestinal hemorrhage is often fatal from erosion of the pancreatico- or gastro-duodenal arteries, less often of the aorta or cava. Hæmatemesis is infrequent, though it has been said to occur in a quarter of the cases where there is intestinal hemorrhage and usually indicates duodenal stenosis, in which the blood passes more readily upward than downward. Constipation is frequent.

Complications and Issue.—(i) Spontaneous *healing* is infrequent and cicatrization occurs in 11 per cent.; the clinical course is often long (five to thirty years). (ii) *Perforation,* total with peritonitis, or partial (chronic) with gall-bladder or pancreas symptoms, is very frequent (50 per cent., Perry and Shaw). (iii) *Gastric dilatation* occurs in 7 per cent. and duodenal dilatation in nearly 2 per cent.; both result from duodenal stenosis. (iv) *Icterus* occurs in less than 3 per cent.; (v) duodenal cancer is an infrequent sequel.

Diagnosis.—Leube holds that a certain differentiation from *gastric ulcer* is never possible and Von Wyl holds that differentiation from gastric ulcer is possible in but 10 per cent. of cases. The other chief cause of error is *disease of the gall-bladder.* As Graham states, "There will always remain a certain proportion of cases that will mislead the careful physician. Those cases of gall-stones in which the stomach symptoms of gas, distress, sour belching and dilatation predominate, and pain is only of a dull character, will usually be diagnosticated as ulcer; but the duodenal case, of which the chief symptom is the sudden, sharp, intense pain of perforative peritonitis, and in which, with no obstruction or hyperacidity, the other stomach symptoms are in abeyance, will be diagnosticated cholelithiasis. Both conditions are purely surgical and the differentiation in many cases must be made on the operating table." Acute pancreatitis (*q. v.*), intestinal obstruction and appendicitis must be considered in the diagnosis.

Treatment.—The treatment is that of peptic ulcer of the stomach (*q. v.*), including its surgical indications. In 7 perforations, 71 per cent. recovered; in 22 chronic ulcers, 95.5 per cent. recovered;

in 22 ulcers associated with gastric ulcer, 95.5 per cent. recovered (Moynihan, 52 operations).

II. Other Intestinal Ulcers.—Ulceration in acute infections, as typhoid, dysentery, anthrax and diphtheria, has been described. In exceptional cases ulceration may occur in erysipelas, variola and sepsis. *Chronic infections* may cause ulcers, as syphilitic, tuberculous and actinomycotic lesions, which are considered under general infections. Very rare cases of ulceration have been observed from *favus* and *mucor corymbifer*. *Inflammatory ulcers,* as the catarrhal and follicular, usually occur in the colon; they may be simple or so-called dysenteric (*q. v.*) and may develop from the epithelial layer; they are seen in enteritis, infantile diarrhœas, etc. *Stercoral* (*decubital*) *ulcers* develop from the pressure of retained hard fæces or foreign bodies, in the dilated pouches of the colon or above an intestinal stenosis. *Constitutional affections* are occasionally causes, as leukæmia, scurvy, gout and amyloidosis. *Toxic* ulcers may develop in uræmia (*q. v.*) and in mercurial and arsenical poisoning.

Symptoms.—The symptoms are rarely distinctive without suggestive etiological factors; most ulcerations occur without symptoms or with ambiguous symptoms. (*a*) *Diarrhœa* is frequent, particularly in ulceration of the large gut; it is often absent with lesions in the small gut or in the upper colon. Diarrhœa results from decreased absorption and from increased peristalsis, due to exposure of the intestinal nerves. (*b*) *Blood* in the stools is mixed with them in lesions of the small gut, sometimes covers them in lesions of the large gut and is voided independently of the fæces in rectal lesions. It is more often dark and tarry, especially in tuberculosis, than bright, except in low-situated lesions and in some profuse typhoid, dysenteric or duodenal hemorrhages. Blood is common in other diseases than ulceration, as in cancer, piles, stasis and blood diseases. (*c*) *Pus* is seen more frequently in colonic than in ileac ulceration; it is common in cancer, proctitis and croupous enteritis. Pure pus in considerable amounts is rather indicative of rupture of extra-intestinal pockets into the intestine, as appendicitic and salpingitic abscesses. (*d*) The presence of *mucus* has no special significance, as it indicates enteritis only, which of course may complicate ulceration. (*e*) *Shreds of tissue* are very rare, save in dysentery. (*f*) *Local symptoms* are likewise ambiguous, as tenderness, pain and tympanites. (*g*) *General symptoms* depend on the basic disease; fever is most frequent in cancerous, dysenteric or tuberculous ulcers. (*h*) *Rupture* causes peritonitic adhesions, diffuse peritonitis, retroperitoneal cellulitis or subphrenic pyopneumothorax.

Treatment.—Treatment is that of enteritis or colitis. Of chronic ulcerative colitis Mehrkorn has published 38 cases in which colostomy resulted favorably.

INTESTINAL DISORDERS IN INFANTS.

Etiology.—Classification of the etiological factors is extremely difficult because of the numerous species of bacteria in the intestine and the impossibility of drawing any absolute line between chemical and bacterial factors and between functional and organic changes. Most cases occur in children between *six and eighteen months old,* in the *summer months,* July especially, and in the *poorer classes.* It occurs largely in infants who are *not fed at the breast,* and for two main reasons; (*a*) cow's milk is prone to disturb digestion (see TREATMENT), and (*b*) there is a greater chance for bacterial infection. Bacteria operate in several ways. Vaughan and Flügge have demonstrated that toxins may form in milk. This factor is especially potent when milk is long in transit, particularly in hot weather. Bacteria may cause inflammation of the intestines; the *Bacterium lactis aërogenes* causes inflammation in the small and the *Bacterium coli* in the large intestine; the proteus, pyocyaneus, pyogenic cocci and, since 1897, Shiga's Bacillus dysenteriæ are also factors, but their relative importance is not yet clearly defined. Escherich, Booker and Favel describe a streptococcus enteritis, which, however, can hardly be considered as a distinct type; Baginsky considers the saprophytes of the intestine and of cow's milk causative, and Czerny holds that toxins may develop in the liver. The distinction between intoxication and infection is maintained with difficulty; *intoxication* causes such symptoms as erythema, hemorrhagic diathesis, anæmia, nervous disturbances and dyspnœa; and *infection* produces certain complications, as lobular pneumonia, nephritis, encephalitis, phlegmon, arthritis, purulent pleurisy and necroses in the liver. In institutional epidemics infection may be carried from one individual to another by fæcal contaminations. Insects may also convey infection.

Symptoms.—(1) The symptoms of *the acute dyspeptic or fermental form* cannot be sharply distinguished from those of the other severer forms, though possibly caused by the same microörganisms. The pathological findings are often, as in adult forms, remarkably insignificant. The *temperature* usually rises suddenly to 103° or 104°, remains high for a few days and falls rather suddenly. The tongue is dry, the face red, the pulse rapid and the breathing sometimes dyspnœic. There is considerable restlessness—sometimes initial convulsions—anorexia and vomiting. The *stools* in infants are normally yellow from bilirubin, are thick and slightly acid in reaction; they become greenish from bacterial action or reduction of the bilirubin into biliverdin, react alkaline from albuminous decomposition or acid from acid fermentation and become thin and offensive; the evacuations become frequent (6 to 10 daily). The microscopic findings are those of acute enteritis in adults, as mucus, leukocytes, triple phosphates in the alkaline stools or cholesterin in the acid stools; blood in any quantity is uncommon. Involvement of the small intestine is attended by considerable gas, some cramping, large move-

ments and slight tenderness and tenesmus. In severe cases the nervous symptoms may become conspicuous and considerable emaciation may result. Albuminuria and cylindruria occur in about 20 per cent. The author has seen two cases of extensive œdema follow dyspeptic diarrhœa without nephritis. Recovery is usual and prompt after thorough intestinal evacuation, though a few cases run a subchronic course.

2. *Cholera infantum* occurs in severe infections, usually in children already suffering from some intestinal disorder. According to Holt, it constitutes but 2 to 3 per cent. of summer diarrhœas. The *bowel movements are copious,* alkaline and contain flocculi; though at first they contain food and are perhaps offensive, they become watery and odorless. Both the stools and the general clinical aspect resemble the picture of *cholera Asiatica* and *cholera nostras* (*q. v.*). Though there is *fever,* the *extremities* are *algid and cyanotic,* the *pulse small,* the heart tones weak and all the symptoms of *collapse* are present; the eyes are sunken and there are the pinched pallid *facies cholerica,* the *thirst* and the incoercible *vomiting.* The skin is rigid, as though the fatty tissue had hardened (*sclerœdema* or *sclerema adiposum*), and nephritis is almost invariable. These symptoms result not only from loss of great quantities of fluid by purging and vomiting, but also from intoxication which produces the convulsions, irregular and Cheyne-Stokes' breathing, retraction of the neck, stupor and other symptoms, termed by Marshall Hall the "hydrencephalic state," and explained as œdema of the brain (*q. v.*), though better explained by intoxication. Oestreich thinks that hypostasis, resulting from compression of the lungs by the tympany, causes more deaths than does the toxæmia.

3. *Acute enterocolitis* (ileocolitis) may follow the dyspeptic form or the various specific infections in children, or may begin as an independent form. Pathologically it includes types which cannot be separated clinically, as (*a*) simple enterocolitis, (*b*) follicular ulceration and (*c*) diphtheroid enteritis. The growing importance of Shiga's Bacillus dysenteriæ in infantile enterocolitis is established by the recent work in this country of Vedder, Duval, Flexner, Bassett, Knox, Park, Zahorsky, Dunham, Wollstein and Rotch. In some epidemics the bacillus produces acid in mannit media, which Shiga's bacillus does not produce. In Rotch's cases of infantile diarrhœa the Bacillus dysenteriæ was found in 16 per cent.; it was sometimes found in the dyspeptic or fermental type, but most often in the ileocolitic type (78 per cent.). In one collection of 412 cases Shiga's bacillus was found in 63 per cent.; Geo. Weaver never found it, though the Flexner-Harris type occurred in 25 per cent. (*v.* Bacillary Dysentery).

The *symptoms* closely resemble those of dysentery in the adult. The stools are small, yellow, green or brown, are very frequent, are attended by tenesmus and void mucus, blood, pus and sometimes shreds of tissue or membrane. Blood may be present from congestion

or acute ulceration. Rotch tabulates the differential features as follows:

Acute Fermental Diarrhœa.	Acute Ileocolitis.
(*a*) Small intestine, mostly.	Large intestine, mostly.
(*b*) 10 to 12 discharges; often large.	10, 15 to 50 discharges, usually small.
(*c*) No or little blood.	Blood and sometimes shreds of tissue or membrane.
(*d*) Mucus.	Mucus.
(*e*) No tenesmus.	Tenesmus.
(*f*) Not much abdominal tenderness and pain.	Abdominal tenderness and pain.
(*g*) No or slight lesions.	Lesions marked.
(*h*) Temperature high—104° F. for one or two days, and then falling sharply by crisis.	Temperature may be high at first (103° F. to 104° F.) but usually soon moderates (99° to 101° F.) and falls gradually by lysis to normal after some weeks.

The severity of the symptoms varies greatly; the catarrhal form may cause mild symptoms or result in death, with or without gastric symptoms. The ulcerative and diphtheroid types are less often attended by vomiting, but run a fatal course of days or weeks.

4. *Chronic dyspeptic disturbances* often occur with gastric dilatation. The onset is gradual or it may directly follow the acute forms. The stools are green or gray, contain mucus, gas and sometimes pus and tissue shreds. Food and fluid are not absorbed, or absorbed in part only; undigested food particles and fatty stools are observed; mucus is not common in marked cases of intestinal atrophy. Vomiting is rather frequent. There is usually but little general disturbance, no fever and little or no colic. The scales show marked loss in weight and in some cases the emaciation may become extreme (pædiatrophy, athrepsia), the muscles becoming weak, the voice faint, the eyes lustreless, the face of the "Voltaire type," and the abdomen distended. Death results from marasmus or intercurrent infections.

Preventive Treatment.—Children should, if possible, be taken to the country in the hot months. Excessive swaddling is to be avoided.

Diet.—(*a*) *Human milk* is superior to all others, and if the mother cannot nurse her child, a wet-nurse should be procured if possible, as few cases of stomach and bowel disease occur in breast-fed infants. The wet-nurse must be carefully examined for syphilis and tuberculosis. The milk from the breast should spurt freely in several streams. A weak child may become dyspeptic even at the breast; calomel irritates but magnesium carbonate gives the most rapid relief. Other children thrive, even when the stools are green, for which Heubner blames the digestion of the infant. Sometimes colic and dyspepsia in the child are due to menstruation, beginning pregnancy, excitement, lack of exercise or alcoholic indulgence in the nurse or mother. If a wet-nurse cannot be obtained, (*b*) *cow's milk* or some *prepared food* must be used, but most of the latter are objectionable because their carbohydrates are not dextrinized, as shown by the iodine test. Cow's milk differs from

human milk in many details; its contamination is far more likely, whence the importance of pure milk and sterilized nipples and bottles. Human milk contains about 1 per cent. of proteids, 4 per cent. fats and 7 per cent. of milk sugar, but in cow's milk the percentages are respectively about 3.5, 3.5 and 5 per cent.; in cow's milk the casein constitutes 80 per cent. of the proteids, which are physically and chemically different in mother's milk, *e. g.,* they contain more whey proteid and more of certain relatively uninvestigated substances, as nucleon. In cow's milk the ferments and fats are different and the mineral salts, especially phosphates, are increased which, as Blauberg has shown, impede digestion. The first objection is the high percentage of proteid (casein), which must be predigested or the milk diluted. Simple dilution, however, necessitates giving too much water or reducing the fat and milk-sugar, fat being essential and milk-sugar being important because the Bacillus lactis aërogenes, operating upon it, produces lactic acid, which is inimical to pathogenic bacteria in the bowel. In modification of milk or in *substitute feeding,* then, the *proteid of cow's milk must be reduced, fat added in the form of cream,* and *milk-sugar added* to attain the normal percentage. The milk may be diluted with thin oatmeal water, as a diastase ferment is present earlier and in larger amounts than previously admitted, though nature doubtless has her designs in withholding starches from mother's milk. The fats of Holstein, Durham, Devon and common breeds can be better emulsified than those of Jerseys and Guernseys, which contain more volatile glycerides.

THEORETICAL BASIS FOR FEEDING A HEALTHY INFANT.

(According to Rotch.)

Age.	Fat.	Sugar.	Proteids.	Proteids if split.		Amount at each feeding, in—		Intervals between feeding, in hours.	No. of feedings, in 24 hours.
				Whey proteids.	Caseinogen.	Ounces.	C. c.		
Premature	1.00	4.00	0.25	0.25	0.25	$\frac{1}{8}$–$\frac{3}{4}$	4–25	1–1$\frac{1}{2}$	24–18
Premature	1.50	4.50	0.25	0.50	0.25	$\frac{1}{8}$–$\frac{3}{4}$	4–25	1–1$\frac{1}{2}$	24–18
At term	2.00	5.00	0.50	0.50	0.25	1	30	2	10
End of second week	2.50	5.50	0.50	0.50	0.25	1$\frac{1}{2}$	45	2	10
End of third week	3.00	6.00	0.75	0.75	0.25	2	60	2	9
End of fourth week	3.50	6.50	1.00	0.75	0.50	2$\frac{1}{2}$	75	2	8
End of sixth week	4.00	7.00	1.00	0.90	0.60	3	90	2$\frac{1}{2}$	8
End of eighth week	4.00	7.00	1.25	0.90	0.75	3$\frac{1}{2}$	105	2$\frac{1}{2}$	7
End of twelfth week	4.00	7.00	1.50	0.90	1.00	4	120	2$\frac{1}{2}$	7
End of four months	4.00	7.00	1.50	0.75	1.25	4$\frac{1}{2}$	135	2$\frac{1}{2}$	6
End of five months	4.00	7.00	1.75			5$\frac{1}{2}$	165	3	6
End of six months	4.00	7.00	2.00			6	180	3	6
End of eight months	4.00	7.00	2.50			7	210	3	6
End of nine months	4.00	7.00	3.00			8	240	3	6
End of ten months	4.00	6.00	3.00			8	240	3	6
End of eleven months	4.00	5.00	3.00			10	300	3	5
End of twelve months	4.00	4.75	3.50			10	300	3	5

"The data are simply a guide for the feeding of the average healthy baby; they are only approximate and not intended to be followed without due consideration to the individual requirements."

Quite low dilutions should be given at first and they should be gradually increased. Attention should be given not only to digestion but to *nutrition,* as determined by regular weighing. Sterilization of milk destroys ferments and protective substances (Ehrlich).

Treatment of Acute Dyspepsia.—(*a*) The *stomach* should be *washed out,* if toxic symptoms are at all marked (Heubner); plain water or a 1 to 3,000 resorcinol solution should be used. (*b*) *Calomel* gr. ¼ and magnesium carbonate gr. j every three hours for about three doses, or one dose of castor oil (3ss to j) are usually administered, but cathartics congest or even erode the mucosa. (*c*) The *bowel should be flushed* with warm water; one pint is sufficient in children of one year, or somewhat more in children two years old. (*d*) *Food should be withheld* (Czerny) for two days, and plenty of boiled water should be given. Albumin water, which consists of the whites of two eggs beaten in a pint of water, may then be given, and thin arrow-root water, beef juice and peptonized milk, may gradually be administered until the green color of the stools changes to a normal yellow-brown. (*e*) *Other medication* is usually superfluous. Lesage recommends that lactic acid be given for alkaline green stools, 3j of a two per cent. solution (p. c.). Resorcinol ⅓ grain and dilute hydrochloric acid ♏v may be given after meals.

Treatment of (Dominating) Enteritis.—If the case is seen early the *stomach* is *washed* out; in all cases *feeding must be suspended* as outlined above. The child should have plenty of boiled water and cool applications to the skin are indicated unless the extremities are blue and cool. *Tannigen* may be given in relatively large doses, 10 to 15 grains, several times daily, even in the very young, possibly combined with minute doses of calomel (gr. $\frac{1}{30}$). As in enteritis of adults, bismuth subnitrate is given in large doses, 3ss. Bismuth salicylate, in doses of ⅓ to 1 grain, is also given. If these are ineffective, opium may be administered. (See page 91 for dosage.)

Treatment of Cholera Infantum.—Food should be withheld. Lavage of the stomach and bowels is ineffective, unless it is done in the earliest stages. There are *three main indications:* (*a*) *fluid must be restored* to the tissues; this is possible only by subcutaneous saline transfusions which support the heart, fill the vessels, stimulate the kidneys and eliminate the toxins; (*b*) *cardiac stimulation,* by hypodermics of strychnine and camphor and local heat; champagne quiets vomiting and supports the heart; brandy and whiskey are given in doses like those employed in bronchopneumonia (*q. v.*), though Heubner believes that alcohol further injures the intestinal epithelium; and (*c*) *secretion must be checked* by administration of paregoric every four hours, of small enemas of starch and laudanum or, which is more effectual, of morphine. (See page 91.) In the fortunate cases which rally, nourishment can be resumed only with the greatest caution. The serum treatment is of uncertain value.

The Treatment of (Dominating) Colitis.—This is much like that of dysentery and includes the measures above indicated. Flushings with normal salt solution, 1 per cent. tannin or 1 to 1,000 salicylic acid may be employed. Tannigen and bismuth should be given freely.

The Treatment of Chronic Dyspepsia.—Treatment in institutional cases begins with nursing; one wet-nurse can nurse several children. Lavage of the stomach with two to three ounces of plain warm water, normal salt solution or 1 per cent. Carlsbad salts solution, increases the functional activity of the stomach. The extract of nux vomica stimulates secretion. The food must be given in the smallest amounts compatible with maintenance of weight and energy.

APPENDICITIS.

Definition.—Appendicitis, the chief abdominal malady, is an inflammation of the vermiform appendix.

History.—Fitz in 1886 drew attention to the subject and coined the name. Appendicitis was previously confused with typhlitis, inflammation of the cæcum; typhlitis is rare and the term "perityphlitis" should be discarded. Our present knowledge of appendicitis is largely due to the work of American surgeons, Willard Parker, who first (1867) advocated operation, Sands, Bull, McBurney, Keen, Senn, Murphy, Weir, Morton, White, Deaver and others.

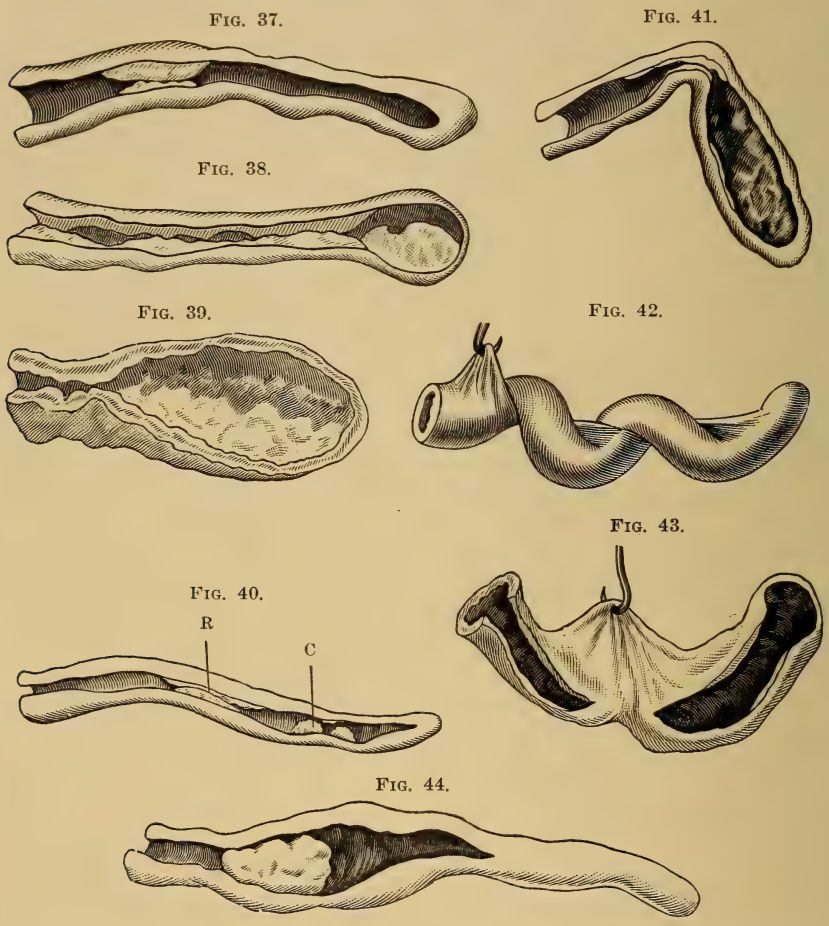
Etiology.—1. Predisposing Factors.—(*a*) The appendix, being a functionless vestige of the cæcum, is inherently *less resistant.* (*b*) Its *lumen* is *smallest at its cæcal end,* which favors retention of foreign bodies, promotes microbic growth and decreases or suspends drainage into the bowel. (*c*) The *meso-appendix,* which conveys the blood to the appendix, is *shorter than the appendix,* which therefore curls; the blood supply is then easily altered by appendiceal œdema and infiltration. (*d*) *Foreign bodies* and fæcal concretions may produce erosions which apparently promote microbic access and therefore inflammation. In J. B. Murphy's series of 2,140 cases, foreign bodies were found in about 20 per cent. and fæcal concretions in 38 per cent. J. F. Mitchell found foreign bodies in 7 per cent. of 1,400 cases of appendicitis and Louis J. Mitchell in 1,600 autopsies found them in about 1 per cent. Foreign bodies include seeds, capsules, gall-stones, pins, nails, shot, fish-bones, etc. (*e*) *Age.*—Over 50 per cent. occur under twenty years of age and 30 per cent. between twenty and thirty. It is rare in infancy and with advancing years obliteration and atrophy lessen its frequency. (*f*) *Sex.*—Males constitute about 80 per cent. of cases; in women it is thought that an extra blood supply from the ovary lessens the results of infection, though Murphy's large series shows but a slight preponderance in males. (*g*) Other rare or disputable factors are trauma, severe exertion, exposure and enteritis.

2. Bacteriology.—The colon bacillus is the most frequent cause and is found in 80 per cent. of cases, though probably in some of them

it has overgrown some other causal organism. Next is the staphylococcus, the virulent streptococcus, then the pneumococcus, proteus and pyocyaneus. Less frequently the typhoid, tubercle, influenza or actinomycosis organism is found; of the last 150 cases exist; Fitz found 5 typhoid perforations of the appendix and Fenwick in a collection of 2,000 cases of phthisis recorded 56 per cent. of tuberculous ulcerations in the bowel, of which but 0.8 per cent. was in the appendix.

Symptomatology and Pathology.—1. The Early Clinical Course.—The *onset is precipitate,* without prodromes in most cases, and is characterized by *four main symptoms* upon the sequence of which Murphy has placed especial stress. (*a*) *Sudden severe abdominal pain,* the intensity of which ranges between colic and agony, is due to acute retention within the appendix caused by the occlusion of the cæcal end. It is felt over the *right lower abdominal quadrant* or over the pit of the stomach, and may even radiate along the genitocrural nerve to the testis, which is sometimes retracted. Reaching its greatest severity in a few hours, the pain persists until the pressure of the pus is relieved by drainage into the bowel, perforation or gangrene. Sudden relief from pain is ominous, as it often indicates gangrene. (*b*) *Nausea and vomiting* result reflexly from occlusion and follow within a few hours after the pain. (*c*) *Abdominal tenderness* soon develops, is rather *general at first,* and is often associated with a cutaneous hyperæsthesia (Sherrin), which covers like half a girdle the right lower abdomen from the spine to the mid-line. The right rectus muscle resists palpation. A little *later* the general tenderness disappears, leaving *well-localized tenderness* over the appendix; *McBurney's point* is in the right lower quadrant of the abdomen (about two inches from the anterior superior iliac spine in a line drawn between it and the navel) or a little below it in adults or above it in children; less frequently the tender point may be found in the loin, in the pelvis (5 per cent.), near the bladder, rectum or uterus on rectal or vaginal examination, near the gall-bladder or very exceptionally on the left side of the abdomen or in hernial sacs, all of which correspond to possible variations in appendicular location. Pressure with the hand on the left half of the abdomen causes pain in the right side when the pressure is relieved. Palpation must be made gently, lest rupture occur. In the early stage of appendicitis it sometimes seems as though a tumor could be felt, which is usually only the contracting rectus. (*d*) *Fever* is *almost invariably* present in the early stage of acute forms, and develops in a few hours to a day, according to the severity of the infection. An initial chill is rare. The temperature rises to 101°–102° or higher in some virulent types or in children. It signifies septic absorption and may cease suddenly in gangrene or gradually in rupture and may slowly recur from extension. It does not signify cessation or absence of pus formation, as abscess or peritonitis may co-exist with low temperature. The *pulse* usually follows the temperature curve, though the

author has seen it remain normal in cases of early gangrene. The leukocyte count runs parallel with the fever, and often—though not always—measures the sepsis; *leukocytosis* may amount to 12,000, 25,000 or even 50,000, but no confident reliance can be placed on its



Figs. 37 and 38.—Obliterating appendicitis. Fig. 39.—Obliterating appendicitis, with distal enlargement. Fig. 40.—Obliterating appendicitis (at R) with calculi (C). Fig. 41.—Appendicitis with bending, obliteration and distal distention. Fig. 42.—Appendicitis with curling around its mesentery. Fig. 43.—Strangulation by a band. Fig. 44.—Calculous obstruction. (Dieulafoy.)

absence as an assuring sign nor on its presence as an indication for operation. Constipation is usual. The urine is often febrile, scanty and usually contains indican and sometimes albumin; dysuria may result from appendicitis in the pelvis. Profuse bowel hemorrhage has been recorded. In Dieulafoy's toxæmic type toxæmia may obscure the local symptoms.

2. The Later Clinical Course.—(*a*) The *inflammation subsides* gradually by drainage into the cæcum. Though the inflammation

may be very circumscribed, the appendix rarely returns to a completely normal condition; it suffers necrosis and ulceration from microbes, fæcal concretions or both, which leave the appendix infiltrated, thickened, rigid and more or less constricted at one or more points. The mucous membrane may be wholly substituted by granulation tissue. The lumen is obliterated in appendicitis obliterans. Occlusion at its intestinal end causes a cyst-like swelling as large as the thumb or even as a sausage. Adhesions to the abdominal wall and gut are common. (*b*) After a few days a localized peritonitic *abscess* frequently forms, usually over the psoas muscle at the angle between the ileum and cæcum, but occasionally in some unusual localization of the appendix (*v. s.* under PAIN). The appendix perforates, generally near its mesenteric attachment, from necrosis or gangrene, and a localized abscess of yellow, thick, offensive pus forms; when the abscess is well demarked, the pain, fever and pulse rate decrease. A *well-defined tumor* may develop, over which no peristalsis occurs; dulness or tympany is elicited according to the depth of the tumor or its fluid or gaseous contents; fluctuation is present only in very large abscesses; the tumor in infections due to the colon bacillus consists more largely of infiltration than of pus. In the incised abscess the green or black and totally gangrenous appendix may be seen; it may be difficult to find. The abscess may form without any symptoms other than those of the onset. The *result of the abscess* may vary greatly: (i) it may rupture into the intestine which forms its wall (2 per cent.), and possibly carry the appendix with it; (ii) it may rupture into the urinary tract, of which cases nearly one-half are fatal; (iii) it may cause thrombophlebitis purulenta, and any or all of the manifestations of septicopyæmia may follow, as secondary fever, suppurative pylephlebitis, liver abscess, subphrenic abscess and vomiting of blood; (iv) it may rupture into the retroperitoneal tissue, which is followed by high temperature; and all possible extensions may occur, as into the groin, scrotum, pelvis, pararenal tissue, pleura (84 per cent. are right-sided), lung, gluteal region, back, hip joint, etc.; and (v) it may rupture into the free peritoneal cavity. (*c*) *The third cardinal issue* is diffuse septic *peritonitis,* which is recognized by all its unfortunate and unmistakable symptoms; these are a sudden fall of temperature followed by a secondary fever, diffuse replacing localized tenderness and rigidity, leukocytosis, persistent projectile vomiting, rapid pulse, Hippocratic facies and collapse from profound intoxication. It most often follows the localized abscess, but may occur directly after a gangrene which is so sudden as to allow of no protective adhesions, or after perforation from a foreign body in the appendix without infection; the latter occurs in 1 per cent. of cases (Murphy). This writer distinguishes variations due to the type of infecting organism: (i) The staphylococcic peritonitis which generally produces but little pus, or, if any, a seropurulent fluid; it forms a plastic lymph which prevents absorption for a short time, but, as the peritoneum becomes abraded, leads to

intoxication; this is evidenced by collapse, an anxious expression and diarrhœa. (ii) The streptococcic form, which produces no pus, but causes a granular, blistered, dry peritonitis, which is quickly followed by rapid pulse, talkative delirium and tympany. (iii) Colon infection, which produces an abundant, creamy, thick, offensive pus and slowly causes death after great depression, low, muttering delirium, somnolence and low arterial pressure.

3. The More Remote Sequences.—Intestinal strangulation by adhesions, deformity and traction from chronic changes in the appendix and intractable intestinal, neurasthenic and nutritional disturbances may result from chronic appendicitis.

In *children* the onset is usually very abrupt; the vomiting is repeated, the pain is greater, the appendix is more often palpable, diarrhœa is more common and the percentage of diffuse peritonitis is greater than in adults, because the omentum is less able to envelop the appendix. In *aged persons* gangrene is more frequent than at other ages.

Diagnosis.—The diagnosis depends on the four cardinal symptoms of onset, in the sequence named; *viz.,* pain, nausea or vomiting, abdominal tenderness and temperature (Murphy), followed by leukocytosis and the other secondary manifestations and by the developments of the later course, as circumscribed abscess or diffuse peritonitis. If all these symptoms are considered separately an analysis is necessary of every abdominal lesion causing pain alone, vomiting alone, temperature, sensitiveness, abscess, tumor, disease in the right lower abdominal quadrant, perforation or peritonitis. Acute typhlitis is very rare and should seldom receive consideration.

Pain occurs in gall-stones, renal calculi, tabetic crises, internal incarcerated hernias, epigastric hernia, intussusception, gastric or duodenal ulcer, tuberculous peritonitis, stone or torsion of the ureter, Dietl's crisis in floating kidney, uræmia, colica mucosa, acute pancreatitis, the crises of angioneurotic œdema, pelvic peritonitis, rupture of extra-uterine pregnancy, twisted pedicles (ovarian tumors), pyosalpinx, dysmenorrhœa and the reflected pain of vertebral disease, pleurisy and pneumonia. *Fever* in typhoid precedes the local symptoms. These affections, as well as gravitating abscesses, infected urachus, lesions of the abdominal wall, cancer, hysteria and hypochondriacal fear of appendicitis, are almost invariably excluded by regarding the sequence of symptoms and by a careful general examination. In *appendicitis larvata* (Ewald), pain is slight or absent, and there may be diarrhœa; the thickened sensitive appendix is often palpable; *appendicitis, chronic ab initio,* causes gastric dyspepsia but, more often, flatulency, constipation alternating with diarrhœa and a dragging discomfort in the right lower abdomen.

Prognosis.—The prognosis is most uncertain, because (*a*) there is no safe criterion for early diagnosis of the type; (*b*) the symptoms and signs are not proportionate to the actual lesions—*e. g.,* light cases clinically may prove to be the most severe anatomically; (*c*) apparent

improvement to the experienced observer is often only the onset of alarming complications, which are in reality less complications than essential parts of the disease; Dieulafoy well calls them "treacherous lulls" (*accalmies traitresses*); (*d*) though appendicitis often subsides clinically, recovery is seldom complete anatomically, whence recurrence (chronic, relapsing or residual appendicitis) often occurs in a more severe form in 24 per cent. (Hawkins) to 44 per cent. of cases (Fitz).

Treatment.—Cases of appendicitis may recover, temporarily (70 to 80 per cent. or perhaps more), but it is impossible to predict which ones will do so. Personal prejudice was formerly against early, frequent or universal operative treatment as advocated by many, as Deaver, Murphy, Holmes and others. After testing and seeing tested the so-called medical treatment, consisting of rest, starvation, laxatives and other placebos, and seeing gangrene, peritonitis with low temperature and even death occur in "mild" cases, the conviction was inevitable that *appendicitis is always surgical, regardless of type, time or tendencies,* and a surgeon should at once be associated with the practitioner in every case. Medical treatment is out of date and recovery under it is due to good fortune alone. The so-called indications for operation are too often signs of irreparable damage. Of course the results of operation depend upon the experience of the operator. Though this is not the place for discussion of technique or of the *indication* for simple drainage or appendicectomy, the following points may be given: (*a*) Early operation *within one to two days* of onset is attended by the least danger. (*b*) Many operators dislike to operate between the *second* and *eighth days* or other arbitrary limits, but this position implies that the clinician can diagnosticate the anatomical changes which are present; gangrene, thrombophlebitis, imminent rupture, etc., cannot be excluded; in this period simple drainage is indicated—possibly, too, appendicectomy, if the appendix obtrudes in the field of incision; lavage, exploration and protracted operation are dangerous. (*c*) *In later cases,* when there is apparent regression, treatment may be more conservative; complete demarkation may be waited for, but even in such cases thrombophlebitis or rupture into the general peritoneum may intervene. (*d*) In cases of *diffuse peritonitis* incision and simple drainage are indicated unless the patient is moribund. The patient is placed in the semi-sitting posture (Fowler) and given frequent or constant normal salt enemas (Murphy). Many cases recover (see PERITONITIS). (*e*) *After the attack*—Murphy's reasons for operation are: "First, after an attack of appendicitis the patient is prone to recurrence; second, upward of 60 per cent. of the cases operated on by me in the intermediate stage had had more than one attack; third, in the large proportion of the cases in which there was only incision and drainage of the abscess (which was a procedure frequently recorded by me in my first three hundred cases) the attacks recurred; fourth, the adhesions, flexions and stenoses, following a primary attack, favor a recurrence;

fifth, even if there were no danger of recurrence of the inflammatory conditions, the adhesions, fixations, stenoses, flexions and retentions in the appendix produce sufficient disturbance of the digestive tract to necessitate operation; sixth, recurrences are as dangerous as primary attacks; seventh, the appendix can be removed without danger." Dennis states that under medical treatment the mortality is 16 per cent., relapses occur in 30 per cent. and diffuse peritonitis causes 100 per cent. mortality; under surgical treatment 4 per cent. die, there are no relapses, and 30 per cent. of cases of peritonitis recover.

INTESTINAL OBSTRUCTION.

Etiology.—Obstruction of the bowels may be due: (I) to intussusception, the most frequent cause, which is due to invagination of one part of the bowel into another; (II) to strangulation, which ranks next; (III) to twists (volvulus) and knots; (IV) to strictures; (V) to tumors; (VI) to foreign bodies; (VII) to dynamic (paralytic and spastic) obstruction. The figures quoted below are from Fitz's series of 295 cases, Leichtenstern's 1,134 cases and Treves' series.

I. Intussusception (Invagination).—This is the most frequent form of acute obstruction (40 per cent.). The external part (the intussuscipiens or vagina) receives into it the intestine just above (the intussusceptum or invaginatum). The actual *cause* is unknown; it develops in perfect health, in various acute and chronic affections, in tumors and strictures of the gut, in diarrhœa and other functional and intestinal disorders and in contusion. *Two theories* are advanced: (*a*) that the gut is spastically contracted (Nothnagel) and therefore enters the gut below, and (*b*) that the gut below is paretic (Leichtenstern). The author saw, with Dr. Walls, a marked case in lead-colic, which recovered under operation. In acute intussusception 50 per cent. occur under ten years of age (Leichtenstern and Fitz); Wiggin in 103 cases found 50 per cent. in the fourth, fifth and sixth months of life. In chronic intussusception 50 per cent. occur in persons between twenty and forty years of age; 66 per cent. occur in males. *Pathologically* only the vital form is of clinical importance, in which the mesentery and its vessels are also invaginated; the "agonal" form occurs during the death agony, chiefly in children, in the small gut, is often multiple and does not involve the mesentery. The vital form is usually single, descending (in 99 per cent.) and complete; incomplete forms may result from polyps or other tumors, usually benign, which drag down the gut. There are three main forms: (*a*) the enteric, the small gut invaginating into the small gut, 30 per cent.; (*b*) the ileocæcal, the small gut entering the large gut, 52 per cent. Leichtenstern (75 per cent. Fitz and 89 per cent. Wiggin); and (*c*) the colonic, the large gut invaginating into the large gut (18 per cent.), which includes the colon invaginating into the rectum; in rare cases a Meckel's diverticulum may invaginate into the small gut.

Symptoms.—(*a*) There is sudden, severe, *reflex pain,* which is usually umbilical in children but sometimes occurs elsewhere in adults, is continuous or intermittent, or paroxysmal in chronic forms of intussusception. (*b*) Early *reflex vomiting* may occur in children, though in adults it is less frequent than in other forms of obstruction; the later vomiting is seldom fæcal (13 per cent., Fitz); vomiting is more marked in high than in low invagination and depends considerably on the degree of mesenteric invagination and its necessary vascular disturbance. (*c*) The *stools are characteristic,* showing blood and mucus in 80 per cent. of acute and in 50 per cent. of chronic cases; in 108 cases collected by Hare and Martin blood was present in the stools in 104; the movements may not stop at once but only after several evacuations. Their evacuation is attended by severe tenesmus (50 per cent.), especially in the young. (*d*) A *sausage- or egg-like movable firm tumor* (50 per cent. Leichtenstern, 61 per cent. Fitz) may be palpated, most often over the ascending or transverse colon, sometimes by the rectum or vagina, and is occasionally visible at the anus, where it has been mistaken for prolapse or polyp and extirpated. It is most common in children and in the ileocæcal form. According to Treves it is nearly always to be felt during paroxysms of pain. (*e*) Meteorism and a depression in the right flank or ileocæcal region are infrequent.

Clubbe's description is excellent: "The very sudden onset in a previously healthy baby is a peculiarity of intussusception. The child is seized suddenly, screams, turns pale, vomits and in a short time seems to recover itself, only to cry again at intervals as if in pain. Soon after the first scream it may pass a normal motion. In from two to ten hours, in the majority of cases (97 per cent.), blood will be passed by rectum. The mass, which varies much in size and position, can nearly always be made out by careful palpation when the child is under an anæsthetic. The child may not look ill, its pulse rate may be hardly raised and its temperature may be normal."

Issues.—Peritonitis results in 50 per cent. of acute and nearly 100 per cent. of chronic cases; perforation occurs in chronic (15 per cent.) more often than in acute cases; sloughing off of the gangrenous bowel occurs in 42 per cent. of adult cases, but seldom in children; in Cruveilhier's case three yards of dead gut were discharged. Spontaneous reposition and fistula are very uncommon.

II. Strangulation.—This causes 35 per cent. of intestinal obstructions. Its *mechanisms* are innumerable; it is generally due to *bands* or isolated peritonitic adhesions; Meckel's diverticulum, the remains of the omphalomesenteric duct, which lies within a yard of the ileocæcal valve and is sometimes continuous as a cord to the navel, has caused strangulation in 183 cases (Hilgenreiner, 1902); adhesions from previous peritonitis, appendicitis or salpingitis; omental strands forming bands under which the gut is caught; holding of the gut in abnormal positions; and binding of coils to each other. Incarceration may occur when there are congenital or acquired *orifices and clefts*

in the omentum or mesentery or under the suspensory ligament of the liver, ligamentum teres or ligamentum latum uteri. *Hernias* include strangulations in the femoral, inguinal, umbilical locations or in the less frequent types, as the obturator, sciatic, lumbar, intersigmoid, retrocæcal (Rieux), foramen of Winslow, duodenojejunal (Treitz's retroperitoneal form) or diaphragmatic. Seventy per cent. of cases occur in males, 40 per cent. between fifteen and thirty years of age, 90 per cent. in the small intestine, 67 per cent. in the right iliac fossa and 83 per cent. in the lower abdomen (Fitz).

Symptoms.—The symptoms are usually acute and urgent. (*a*) Early reflex *vomiting,* from shock, voiding in succession gastric contents, bile and fæcal matter which usually consists of yellow, brown or black fluid or very rarely solid fæces, is strongly indicative of organic strangulation; vomiting usually lasts from the beginning to the end of the disease. (*b*) There is early severe reflex *pain,* which is greatest at the navel and lasts until collapse, peritonitis or paralysis of the gut develops. (*c*) *Collapse,* with Hippocratic expression, weak pulse and hurried breathing, results reflexly and may occur early from the disturbed local circulation in the gut or later from peritonitis. (*d*) *Complete sudden stoppage of the bowels* may result, in which no fæces or flatus can pass; some fæces from the lower gut may pass after enemata, but no gas. Meteorism of two kinds occurs—(i) the static variety in which gas accumulates in loops above the obstruction and (ii) the local variety, in which the strangulated loop becomes distended, because its circulation is impaired; it may be seen or felt as an *immovable tense mass,* showing no peristalsis and corresponding to the thick, œdematous, hemorrhagically infiltrated and finally gangrenous gut. (*e*) The *urine* is decreased, because of the vomiting, lack of food and nervous collapse; it contains indican if the obstruction lies in the small gut. Sometimes casts, albumin or even hemorrhagic nephritis are observed. (*f*) Strangulation in some cases may be found in the usual or accessible hernia locations.

Diaphragmatic hernia was first diagnosticated by Lichtenstern (1874); it is left-sided in 88 per cent., is congenital or acquired (from trauma especially) and is recognized in but 3 per cent. of the reported cases; it closely resembles pneumothorax (*q. v.*).

In strangulation and other acute obstructions (*v. i.*) the mucosa in the occluded area may become eroded, more easily penetrated by bacteria, ulcerated and hemorrhagically infarcted, leading to necrosis, perforation and peritonitis.

III. Twists (Volvulus) and Knots.—Volvulus is a twisting of the intestine around its mesenteric axis; twisting of the gut on itself is rare; volvulus occurs in 14 per cent. of obstructions (Fitz); 68 per cent. of the cases are males, usually between forty and sixty years old; it occurs in the sigmoid flexure in 66 per cent., less frequently in the cæcum (75 cases collected in 1905 by Corner and Sargent), colon ascendens and small gut, and then especially when the large

and small intestines have a small mesentery; only nine cases of volvulus of the stomach are recorded. The predisposing causes of volvulus are a vegetable diet (said to explain its frequency in Russian peasants), persistent constipation, a long sigmoid flexure with a long narrow mesentery, and mesenteritis. The twist may occur through 180 to 360 degrees or more.

Symptoms.—The symptoms closely resemble those of the forms described, especially when knots form between two coils of the small intestine. In volvulus of the sigmoid there is pain and vomiting, which is less marked than in strangulation or invagination and is seldom fæcal; the *local meteorism* is *more pronounced* than in any other type of obstruction, for the huge coil of the obstructed gut passes upward, possibly to the right, in extreme cases pushes up the diaphragm to the third or fourth ribs and seldom shows any peristalsis, which its disturbed circulation prevents. But little water can be injected into the rectum. Hemorrhagic peritoneal exudate is common.

IV. Strictures (Enterostenosis).—Strictures more often cause chronic than acute obstruction. (*a*) *Congenital* stricture is even rarer than the rare congenital atresia, of which Helmholz (1907) collected 199 cases in the small bowel. (*b*) *Cicatricial* stricture results from healing of tuberculous, syphilitic, dysenteric, duodenal and very rarely typhoid ulcers. In 1904 Arbuson collated 129 cases of tuberculous stricture. (*c*) Annular *cancer,* (*d*) compression by various organs or *tumors* (80 per cent. of which are pelvic) and (*e*) *traction* are other causes. (*f*) Inflammatory hypertrophy of the mucosa and muscularis is exceedingly uncommon.

Symptoms.—The symptoms are those of chronic obstruction and vary with the location of the stenosis. (1) *Stenosis of the small intestine* may occur (*a*) above the duodenal papilla, as a result of gallstones or duodenal ulcer and resembles pyloric stenosis, from which differentiation is practically impossible; it may occur (*b*) in the duodenum below the papilla, as a result of gall-bladder adhesions, duodenal ulcer, tumors or pancreatic disease (pancreatitis, tumor, cyst); icterus is frequent, as well as vomiting of bile and pancreatic juice which digests starch and fibrin in an alkaline medium; other symptoms are dilatation of the stomach, pale stools, very slight abdominal meteorism, except over the epigastrium, and the absence of indicanuria. (*c*) Jejuno-ileac stricture may result from healed ulcers, inflammation in replaced hernias and adhesions to the internal genitalia; the ulcer stricture may be multiple, as many as twelve being recorded in one case; it is remarkable that extreme stenosis may cause no symptoms. There is constipation, which sometimes alternates with diarrhœa, colic, meteorism and visible palpable peristalsis, causing "intestinal stiffness" (Nothnagel) until the obstruction is finally overcome by the hypertrophied muscle above the stricture, when a gurgling sound is sometimes heard. The colon is collapsed. (2) *Stricture of the large gut* is marked by *constipation,* which is significant when it develops rapidly in a person not pre-

viously constipated or when cathartics have no effect in constipated individuals; by paroxysmal or *colicky pain,* even more than in stricture of the small gut, because the stools are harder; eventually by nausea and vomiting, which are successively gastric, bilious and stercoraceous; by *meteorism* along the colon and in the lumbar region behind, which is usually dull; by increased, even tetanic, *peristalsis* in the intestinal coils above the stricture, which possesses a distinct localizing value; and by the evacuations which may be *ribbon-like* or *sheep-stools;* these are overestimated diagnostically, for they may occur in tumors, starvation, colica mucosa and spastic constipation.

V. Tumors.—(See page 651.)

VI. Foreign Bodies.—Foreign bodies caused obstruction in 12 per cent. of Fitz's series. (*a*) *Gall-stones*—of which 250 obstruction cases are on record—lodge chiefly in the upper duodenum or lower ileum, ulcerate through the gall-bladder into the duodenum or less often through the colon, or compress the duodenum through a dilated pouch of the cystic duct. In a surprisingly small per cent. is there a history of colic (19 per cent.) or of icterus. Collapse is usually not marked, a tumor is seldom felt and the constipation is often but relative; indeed, diarrhœa may occur. (*b*) *Fæcal masses* are far more common than statistics show and form the chief obstruction of the large gut. The writer saw absolute obstruction develop after a Thanksgiving dinner; there was fæcal vomiting and severe collapse; a mass of not particularly hard food, measuring two inches in diameter, was found; recovery occurred, though the bowel was partly necrotic. They are found especially in the sigmoid (see CONSTIPATION). (*c*) *Enteroliths* are not common. They form slowly of phosphates, usually with an organic nucleus; sometimes they are constituted of bismuth, chalk, iron or magnesia. They measure an inch to nine inches in diameter and in one instance an enterolith weighed four pounds. Though most common in the large intestine, they may develop in diverticula of the small intestine. (*d*) Hair tumors, twisted masses of ascarides, fruit seeds, oatmeal husks (especially in Scotland), stones and foreign bodies (in children or insane subjects), are possible causes.

VII. Dynamic Ileus.—The above causes are mechanical. Another form of acute obstruction is distinguished, namely, dynamic ileus (paralytic or spastic obstruction). Under this topic may be included obstruction following mesenteric embolism, trauma, abdominal operations or inflammation in an undescended testicle; it is largely associated with acute peritonitis. Spasmodic obstruction may develop in rare cases without cause, or in neurotics, in gall-stone occlusion, ulceration and intestinal crises of tabes. It rarely produces fæcal vomiting; Bregmann (1901) found in the literature 20 cases of hysterical ileus.

Diagnosis of Intestinal Obstruction.—1. OF THE FACT OF OBSTRUCTION.—The symptoms are generally unmistakable and consist of constipation, cessation of flatus, distention, particularly the local form,

compensatory peristalsis and frequently general reaction (collapse). The hernial sites, the rectum and the vagina must always be carefully examined.

2. Of the Location.—The uncertainties of diagnosis of location are manifold, but the following table emphasizes the leading criteria:

Obstruction of the Small.———	*vs.*———Of the Large Intestine.
General Condition—Earlier and more profoundly impressed (reflexly) by toxæmia and peritonitis.	Later and less impressed.
Symptoms more marked, more stormy onset, more vomiting (both reflex and fæcal), more pain.	*Signs more marked,* as meteorism along colon (later becoming general), less water can be injected, more peristaltic effort and palpable rigid coils, more results by rectal or vaginal examination, more tenesmus.
Indicanuria—Develops early, and to marked degree.	Develops later or not at all (also present in cancer and peritonitis).
Urine—Much decreased.	Little decreased.

3. Of its Nature.—Only the broadest probabilities may be considered. (*a*) It is practically impossible to recognize obstruction by *bands, adhesions,* Meckel's diverticulum, clefts and internal hernias (except diaphragmatic). (*b*) *Invagination* is not only the most frequent but also the most characteristic form, because of its sudden onset (in children), severe intermittent pain, tumor, bloody stool (very rare in hernias replaced by operation or taxis, in strangulation, volvulus or gall-stones), tenesmus, results of rectal palpation and the exfoliation of dead intestine. (*c*) *Strangulation* is next in frequency and is differentiated from coprostasis, stone occlusion, compression, stenosis and tumors by its more marked and earlier collapse, more severe initial pain and vomiting, greater local meteorism and more frequent hemorrhagic peritoneal effusion. (*d*) *Volvulus* is suggested by the advanced age of the patient, constipation, slower onset and course, infrequent fæcal vomiting and the meteorism, which is greater than in any other form and is accompanied by little peristalsis. The small amount of fluid which can be injected, the hemorrhagic peritoneal fluid, tenesmus and slightness, lateness or absence of indicanuria are also suggestive. (*e*) *Gall-stones* often give no positive symptoms; in some instances the lack of shock, of increased pulse and of meteorism and the paradoxical intermittent release of flatus and fæces may be suggestive. In no other type is fæcal vomiting, combined with the passage of flatus and fæces, so common.

4. Differentiation.—Confusion with flatulent colic, gall- or kidney-stones and incarceration of the kidney, uterus or testis is avoided because in intestinal obstruction there is rapid stoppage of the bowels; the author saw one case of lead-colic with intussusception. The possibility of acute pancreatitis must be considered. Peritonitis

is characterized by early fever, early diffuse tenderness, meteorism, cessation of peristalsis, board-like abdominal rigidity, often by fluid in the peritoneum, late collapse, late fæcal vomiting, later and less complete obstruction and by frequent singultus.

Treatment.—In *stenosis,* which is typified by ulcer cicatrices, a concentrated fluid or semi-fluid *diet* should be given and thorough mastication of solid food and avoidance of coarse food or food with chaff, skins, stone or gristle should be insisted on. *Cathartics* are to be avoided and the bowels should be moved with enemata of oil or water.

Thiosinamin has been recommended to dissolve cicatricial stenosis:

℞ Thiosinamin ..... 3j.
Glycerini ..... 3iij.
Alcoholis diluti ..... 3vj.
M. et S.—Inject 8 to 15 drops subcutaneously every 2 to 3 days.

Operative measures may be instituted for foreign bodies or for malignancy, in which case the outlook is best in lesions of the colon and ileum.

1. Medical Treatment of Obstruction.—In every case the physician should at once divide the responsibility with a surgeon. In acute cases, especially in the small intestines, food causes aggravation. Early gastric lavage affords some transitory relief. The possibility of chronic *fæcal obstruction* should be first eliminated by colonic flushings, given with the patient's head down or body inverted; hard desiccated fæces must be dug out with the finger, with frequent enemata to bring away fragments; in colonic injections a soft tube cannot be passed easily above the sigmoid and hard tubes may cause perforation. *High enemata* and *colonic inflation* with air occasionally reduce volvulus and intussusception. Laxatives, massage and puncture of the gut with an exploring needle are always contraindicated. *Opium* is useful in the early stage to relieve shock; in moderate dosage it does not produce intestinal paresis. *Stimulants* hypodermatically are frequently indicated. The use of *atropine* (Theden, 1788) has been recently revived, especially by Batsch (1900); some cases are relieved by it and physostigmine.

2. Operative Treatment.—Medical treatment of strangulation, intussusception or volvulus is of little value. A most convincing statistical argument for *early operation* is Naunyn's series of 288 cases, in which 75 per cent. of recoveries were obtained by operation in the first two days, while each successive day lowered the patient's chances to 40 per cent., 30 per cent., etc. Obalinski reports 35 per cent. recoveries, Treves 54 per cent. and Kocher in his earlier experience 38 per cent. and later 70 per cent. Much depends on the physiological resistance of the patient. In intussusception Gibson's figures give 53 per cent. as the average mortality from operation, being 39 per cent. for operations in the first two days and increasing with each day of postponement; it was 82 per cent. in children under three months of age; Rydgier's mortality in chronic forms was but 24 per

cent. Forty per cent. of adults and 80 per cent. of children die under medical treatment (Leichtenstern). In 100 cases of intussusception in children 63 per cent. of operated recoveries are reported by Clubbe (1905). Fifty-two per cent. of obstruction by gall-stones recover under expectant and 33 per cent. under surgical treatment. For the indications and technique of resection, anastomosis, enterostomy, colostomy, etc., reference should be made to surgical treatises.

INTESTINAL TUMORS.

Carcinoma is the only neoplasm of clinical importance; it constitutes 22 per cent. of all cancers. Its *etiology* is obscure. Trauma is seemingly an occasional factor and previous ulceration is predisposing. It is most frequent in males over forty years of age; according to Maydl, 17 per cent. occur in persons between thirty and forty and 14 per cent. in those under thirty years of age.

Pathology.—According to Leube, 80 per cent. occur in the rectum, 15 per cent. in the cæcum and colon and 5 per cent. in the small intestine. Hausmann, in 268 cancers of the bowel (excluding the rectum), found 91 per cent. in the large and 9 per cent. in the small bowel; if figured more in detail, 37 per cent. occur in the sigmoid, 18 per cent. in the cæcum, 11 in the descending colon, 9 in the transverse and 8 per cent. in the ascending colon, 8 per cent. in the ileum, 4 in the splenic and 3 in the hepatic flexure and 2 per cent. in the jejunum. Kanzler (1906) found in the literature only twenty-five cases of cancer in the jejunum and ileum (*i. e.,* in the small intestine exclusive of the duodenal and ileocæcal regions). The literature records but 53 duodenal carcinomata. The sigmoid, splenic and hepatic flexures are especially predisposed, probably because these sites are more fixed and exposed to irritation. It is usually primary, sometimes secondary by contiguity, but very rarely metastatic. Originating in the intestinal glands, it is usually annular in form and causes stenosis and ultimate occlusion of the bowel; it is less often nodular and is very rarely diffuse and flat. Histologically it may be an adenocarcinoma (especially when it occurs in the duodenum), scirrhus (in the sigmoid), medullary, cylindrical-celled (in the cæcum and sigmoid), colloid or flat-celled (in the rectum). Beginning in the mucosa, early involvement of the other coats and lymph vessels usually occurs, followed by later involvement of the peritoneum and metastasis, especially in the liver. McWilliams finds in the literature 105 appendiceal cancers.

Symptoms.—1. Disturbance of the Intestinal Function.—(*a*) There is usually *constipation,* resulting from narrowing of the intestinal lumen; it is most marked in tumors of the large intestine. Ulceration frequently causes diarrhœa. (*b*) The stools are irregular and sometimes assume peculiar forms, as band-like or *sheep-like stools,* to which, however, undue importance has been attached, as they occur in inanition, constipation, etc. (*c*) *Blood, mucus* and *pus* are com-

mon in the fæces, though equally frequent in other ulcerations and in enteritis. The evacuations may be stinking. (*d*) Fragments of tissue are uncommon; they also occur in dysentery and are diagnostic only when showing carcinomatous arrangement of the cells. (*e*) *Pain,* due to enterostenosis, is usual; it may be sacral when due to sigmoid cancer. (*f*) Sudden complete *occlusion* of the bowel may follow previous gradual narrowing of the lumen. (*g*) The lower the tumor in the intestinal tract the greater is the *tympany* and *fæcal retention* and the purer the pus and blood (*i. e.,* the less mixed with the fæces). Above the stricture the bowel is dilated and its muscle is hypertrophied (*visible peristalsis*). (*h*) Indicanuria has the same significance as in intestinal obstruction.

2. Cachexia and Marasmus.—These, with the above symptoms, are most suggestive.

3. Tumor.—Until a tumor is detected the symptoms described are not diagnostic. It is best felt if the bowel is empty; it may be confused with a normal contracted colon, the edge of the ileopsoas or with fæcal masses; it is irregularly oval in clear cases, though in annular forms it escapes detection, for it merely constricts the bowel. It is usually tender and is movable or immovable accordingly as the bowel involved is naturally free or fixed (incomplete peritoneal investment), or is adherent to other structures by adhesions or extension by contiguity. Whether free or fixed, the tumor is generally below the navel. Gas and fluid are sometimes felt or heard to escape through the stenotic point and distention of the large bowel by introduction *per rectum* of air or fluid may aid in localization of the tumor. If the tumor grows from movable gut it may disappear and reappear from time to time. When no tumor is detected by abdominal palpation and inspection a rectal and vaginal examination should always be made.

4. Complications.—Complications may first declare the malignancy of primarily ambiguous symptoms, as (*a*) icterus, especially in duodenal cancer; (*b*) hemorrhage from the bowels; (*c*) rupture into the bladder or vagina or into the stomach, with fæcal vomiting and diarrhœa from partially digested food entering the colon.

Diagnosis.—The diagnosis is often difficult, except in rectal localization, for latency is common and many symptoms merely indicate stenosis without declaring its nature. According to Leube, differentiation is required from other movable and immovable tumors:

1. Movable Tumors.—(*a*) These may be pyloric, requiring differentiation from duodenal cancers; the symptoms may be identical, but icterus, the presence of free HCl, bile and pancreatic juice in the stomach are aids in diagnosis. (*b*) The corset liver, movable spleen and kidney will be considered under those topics. (*c*) Omental and mesenteric tumors may be confusing, but they do not produce the same enterostenosis. (*d*) Scybala may co-exist with malignancy; the repeated use of enemata and cathartics determines the diagnosis.

2. Immovable Tumors.—(*a*) Renal and retroperitoneal tumors

lie beyond the colon, on its inflation, and rarely cause the same degree of intestinal obstruction. (*b*) Encapsulated peritoneal exudate may be difficult of diagnosis; the author has seen two chronic appendicitic abscesses in individuals over sixty years of age which were diagnosticated as tumor, were hard on palpation, and which recovered by discharging pus into the bowel. (*c*) Ovarian or uterine tumors are less obstructive and are located more in the pelvis. (*d*) Localized inflammatory infiltration of the intestinal wall is distinguished by its regressive evolution. Bittorf, particularly, has called attention to circumscribed inflammation of the sigmoid flexure and colon.

Rectal cancer should receive special consideration in diagnosis. It is usually annular and when developed is rigid, frequently resembles an inverted funnel, becoming narrow upward and may even resemble a hard cervix uteri. The colloid form extends upward and downward, is often tubular and causes much regional invasion. A systematic rectal examination—which is unaccountably neglected more often than any other simple method of physical examination—saves many lamentable errors. The early stricture may yield when ulceration develops, and this usually occurs with discharge of blood, often ichorous pus and sometimes malignant tissue. Pain is usually but not always present, tenesmus is the rule and hemorrhoids are very suggestive. Cachexia develops with great rapidity, though it is no necessary symptom; the author has seen death result from acute obstruction with perfect preservation of color, nutrition and energy. Intoxication symptoms, as stupor, are frequent.

Syphilitic strictures are slower and subject to greater variations; tuberculous ulcers are flatter, softer and more undermined.

Cancer in any part of the bowel cannot be clinically differentiated from sarcoma, of which Lecène (1904) collected 75 cases involving the small bowel; the course is more rapid and sarcomata seldom stenose the lumen. Hellström found 45 lipomata and Steiner 34 myomata recorded.

Prognosis.—The prognosis of all forms and locations is bad. Marasmus, coma carcinomatosum, anasarca, thrombosis, embolism, secondary infections, stenosis or perforation may be the cause of death.

Treatment.—Treatment is that of enterostenosis, in regard to diet. Opium must inevitably be used toward the end. Operation, either palliative enterostomy, colostomy or curative enterectomy, may be indicated. In 1896 Wolfler's series of resection gave a mortality of 54 per cent., as against a death rate of 39 per cent. in resection for causes other than malignancy. Hochenegg (1902) in 194 radical operations, reports a mortality of but 12 per cent. and estimates his recoveries at 16 per cent. Czerny (228 operated cases, 1902) reports 14 per cent. of permanent recoveries.

ENTEROPTOSIS.

Enteroptosis is not a disease *per se,* but only a condition in which the abdominal viscera "fall" or prolapse in the abdomen. The stomach (gastroptosis), intestine (enteroptosis), colon (coloptosis) or solid viscera (splanchnoptosis), as the liver, kidney or spleen, may be implicated. The condition, first described by Morgagni, is known as Glénard's disease; Glénard referred it to laxness of the hepatocolic ligament. Ewald and others combat this view and explain the transverse rigidity felt in the abdomen not as the colon, but the pancreas, exposed by sinking of the intestines. Its exact explanation is still disputed, though whatever causes relaxation of the abdominal wall or of the intra-abdominal suspensory structures may induce prolapse, as trauma, coughing, loss of weight, pregnancy, tumors of the liver, spleen or kidney, spinal deformity and corsets. Seventy-five per cent. of the cases occur in women. Stiller maintains that splanchnoptosis is a congenital asthenic condition whose most frequent stigma is a floating tenth rib (*costa fluctuans decima*); this is found in 80 per cent. of cases. He also speaks of a *habitus enteroptoticus,* which somewhat resembles the *habitus phthisicus;* in the normal chest the vertical line drawn from the ensiform to the navel is short, compared with the distance from the median line, horizontally, to the tips of the floating ribs; in the *habitus enteroptoticus* the vertical line is much longer and the horizontal line is shorter than normal and shorter compared with the vertical line; this *habitus* is considered a congenital predisposing cause and enteroptosis is the resulting acquired disease. The tendency develops the disease when the patient's nutrition suffers.

Symptoms.—1. Stomach Findings.—Gastroptosis is characterized by falling of the organ to a lower point in the abdomen, whereby both its upper and lower limits are abnormally low, the epigastrium is flattened and the hypogastrium is distended. Eichhorst finds it in 35 per cent. of women and in 5 per cent. of men. Dilatation of the stomach is excluded by finding the lesser curvature at a lower point than normal, which is determined by inflation with carbon dioxide. In marked gastroptosis the lesser curvature lies near the umbilicus; dilatation and gastroptosis may co-exist. The sharp angle formed at the pylorus or duodenum, too, may cause difficulty in gastric evacuation. Gastric symptoms may be noted, as anorexia, a sense of pressure, eructations, nausea and gastric or intestinal hyperæsthesia, or as complications, vomiting, hyperacidity or motor insufficiency. In many cases gastric and other symptoms are entirely lacking.

2. Intestinal Findings.—Enteroptosis or coloptosis is frequently found with gastroptosis. The transverse colon sinks in its central segment so as to assume the form of the letter U or V. Constipation, sometimes alternating with diarrhœa, is frequent.

3. Visceral Findings.—Floating kidney, liver and spleen will be described separately; in this connection the only necessary comment

is that floating kidney is neither cause nor effect of gastroptosis or enteroptosis, but merely coördinate to some general causal factor.

4. Nervous Symptoms.—These occur in 50 per cent. of cases. Many subjects are nervous, delicate, slight and irritable women. The most common symptoms are depression, cephalic pressure, backache and other *neurasthenic* manifestations. The abdominal aorta frequently throbs with violence. The patient complains of a "falling out" sensation. Emaciation, constipation and anæmia are common.

Treatment.—(*a*) For the *neurasthenic symptoms* the treatment outlined under neurasthenia (*q. v.*) is indicated. The diagnosis should not be disclosed to the patient, for this initiates, by suggestion, a long train of additional symptoms. (*b*) *Increase in weight* should be sought by ample feeding and rest in bed, which indication is met by the rest cure. (*c*) *Supports* to restore and maintain the viscera in their normal location often give considerable and immediate relief. The direction of the pressure should be upward and inward. Strips of adhesive plaster are useful but uncomfortable. (*d*) *No drugs* can restore tone to the lax ligaments. Strychnine may be beneficial as a general and digestive tonic. (*e*) *Operation* is indicated only in cases with neurasthenic symptoms. Shortening the mesentery, resecting parts of the recti muscles, suture of the liver, stomach and other organs to fixed tissues have been performed seemingly with success, but these procedures can as yet receive no general commendation.

DILATATION OF THE COLON.

Colectasia may be classified as follows: (*a*) *Gaseous distention,* which may occur in acute toxæmias. Fenwick recorded a case so marked that death resulted from heart paralysis. The treatment is that of tympanites (*v.* Typhoid and Enteritis). (*b*) Cases which are due to *obstruction* by fæces, foreign bodies, volvulus. (*c*) The so-called *idiopathic colectasia,* which occurs in children (in 80 per cent. of cases) as a congenital lesion or in male adults over fifty years. Up to 1907, 150 cases were published. It is also known as Hirschsprung's disease, being first described by Hirschsprung, of Copenhagen. Treves holds that it is always due to obstruction, though cases without obstruction are noted, as Formad's case, in which there was great hypertrophy of the muscularis without organic stricture; the circumference of the colon was 30 inches and its weight with the contained fæces was 47 pounds. The leading symptoms are obstinate constipation and meteorism. Of the reported cases only about 25 per cent. recovered either under medical treatment or after resection of the sigmoid or colostomy. Death results from marasmus, auto-intoxication, intestinal obstruction, perforation or peritonitis.

INTESTINAL HEMORRHAGE.

Etiology.—There are five groups of causes. (*a*) *Abnormal intestinal contents,* as hard fæces, foreign bodies, gall-stones, caustic poisons or parasites. (*b*) *Intestinal lesions,* as the numerous ulcerations, inflammation, invagination, volvulus and trauma. (*c*) *General affections;* cardiac or portal stasis, amyloid disease, infarction of the mesenteric vessels, aneurysmal rupture, scurvy, uræmia and cholæmia. (*d*) *Infections,* including typhoid, dysentery, etc., and hemorrhagic exanthemata. Sepsis is a prominent factor (see HEMORRHAGIC DISEASES OF THE NEW-BORN). (*e*) Blood *from the stomach.*

Symptoms.—The symptoms are: (*a*) *Blood in the fæces.* It may be *obvious* or *"occult"*—*i. e.,* found only microscopically, chemically or spectroscopically. Blood is generally present microscopically a number of hours before it is seen by the naked eye. It is usually mixed with fæces and dark, tarry and offensive when it comes from the small gut; it may coat the movements and be bright in color when it comes from the lower intestine. (*b*) *Acute anæmia* may follow profuse hemorrhage, evidenced by pallor, syncope, rapid heart and slight febrile movement. (*c*) There are finally the symptoms of the *causal disease,* as typhoid, cancer, etc.

Diagnosis.—The diagnosis of enterorrhagia concerns (*a*) the detection of *blood,* which may be simulated by bismuth or blueberries, but its presence is easily established by the red disks, the guaiac reaction and the spectroscope; (*b*) the question whether the blood comes from the bowel or from the mouth or stomach; (*c*) whether it comes from the large or small gut (*v. s.*); and (*d*) its causes, which are most often hemorrhoids, typhoid, dysentery and cancer of the lower bowel. It goes without saying that no extended examination of the abdomen is permissible at the time of the hemorrhage.

Prognosis and Treatment.—The prognosis and treatment are involved in the basic lesion (*v.* page 51).

PILES.

Etiology.—Hemorrhoids result from (*a*) *stasis;* portal stasis operates through the superior hemorrhoidal and inferior mesenteric veins and is more potent than cardiac stasis, which congests the middle hemorrhoidal and internal iliac veins; (*b*) from *constipation,* pregnancy and other obstructive lesions, as cancer of the rectum. According to Duret, vigorous contraction of the abdominal muscles is a more potent cause than constipation. Piles are most common in the fifth decade of life and in men. Hemorrhoids are usually spoken of as varices, but, as Reinbach has shown, they are often cavernous angiomata.

Symptoms.—*External piles* appear as tender, painful, dusky-red or purple swellings outside the sphincter ani; they occur singly or may grow in a circle around the anus; they may be soft or hard. The pain produced is throbbing and may be very severe. Itching is very

common. They may bleed, waste, ulcerate or suppurate. Piles which bleed are called "open" and those which do not are called "blind." *Internal piles* occur above the sphincter, below which they may prolapse and strangulate. Tenesmus, mucus secretion and dull pain sometimes referred to the back and the sacro-iliac articulation, may be noted.

Diagnosis.—Mere inspection is sufficient to exclude prolapse, condylomata, papillomata, etc. The possibility of carcinoma in their etiology should be borne in mind.

Treatment.—(*a*) *General* treatment should deal with the basic factors. Exercise is indicated for those of sedentary habits, and constipation (*q. v.*) should be relieved. (*b*) *Local* treatment: a small *injection of cold water* before each movement softens the irritating hard passage and constricts the piles, thereby favoring their reduction. *Cleanliness* is of prime importance in the relief of itching, for which also a 1 per cent. carbolic salve is of great value. In acutely painful cases a cocaine salve is beneficial. The following formulæ are astringent and anodyne:

℞ Extracti opii ..... gr. ij.
Extracti belladonnæ ..... gr. ij.
Acidi tannici ..... ʒss.
Olei theobromatis .....q.s.
M. et ft. in suppositoria No. X.
S.—One at bedtime.

℞ Cocainæ (alkaloid) ..... gr. iij.
Iodoformi ..... gr. x.
Orthoformi ..... gr. v.
Olei theobromatis .....q.s.
M. et ft. in suppositoria No. XII.
S.—One at bedtime.

They must be given with reserve, lest the opium or cocaine habit be acquired. *Surgical* treatment is indicated when local measures fail.

DIARRHŒA.

Definition.—Increased frequency of the stools with decreased consistence. Diarrhœa is not synonymous with intestinal catarrh, though a sharp differentiation is often difficult. In some individuals two or three movements a day are physiological.

Etiology.—(*a*) *Irritation* may cause diarrhœa; some cathartics, as the aromatics, increase peristalsis and others, as salts, cause increased transudation; diarrhœa dyspeptica is caused by intestinal or gastric dyspepsia, fruits and vegetables; diarrhœa stercoralis is caused by irritation from the fæcal masses; diarrhœa entozoica is caused by intestinal parasites. In general there are *two mechanisms:* (i) the stools are hurried through the bowel by increased peristalsis (irritation of the ganglia in the gut), in which case bile is found in them; and (ii) there is increased serum transudation into the intestine when

little or no bile is found. (*b*) *Nervous diarrhœa* causes from two to fifteen thin movements, containing no increased mucus; it is attended by rumbling in the bowels, peristaltic unrest and is chiefly observed after mental excitement or in neurotic individuals; some cases are reflex, as from uterine or dental irritation. (*c*) *Toxic substances* in the blood may cause irritation of the ganglia or act upon the brain, *e. g.,* in uræmia, typhoid or sepsis.

Treatment.—Opium is the most valuable remedy in diarrhœa, except in stercoral and dyspeptic types; it acts less by checking secretion than by suspending peristalsis and mitigating pain and spasm; the crude drug is superior to its alkaloids, as its absorption from the bowel is slower and its effect is therefore more marked; ¾ of a grain of the extract or a dram of the camphorated tincture should be given with bismuth in massive doses as in acute or chronic enteritis (*q. v.*). Important adjuncts are *local heat,* which quiets irritation and peristalsis, *rest* and a *spare diet* (boiled milk, eggs, toast and later lean meat, as in enteritis). In nervous diarrhœa two drops of Fowler's solution before meals is often advantageous.

CONSTIPATION.

Constipation (insufficient amount of fæces) is a relative term, for some persons normally have movements every second or third day and constipation may exist even with daily movements.

Etiology.—(1) *"Physiological"* constipation results from irregularity in going to stool, as in travel, from obesity, from insufficient exercise or water, from loss of fluid by sweating, from a dietary rich in proteids, as milk, and from various kinds of over-medication. (2) Constipation may be *symptomatic of other affections,* as (*a*) *gastric* ulcer, cancer, dilatation or hyperacidity; (*b*) *intestinal* obstruction, cancer, adhesions, catarrh, passive congestion or Glénard's enteroptosis; (*c*) *pelvic* conditions, as pregnancy, ovarian or uterine disease or enlarged prostate; (*d*) *infections* (which stimulate the inhibitory nerves); (*e*) *nervous* disease, meningitis, tumors, myelitis, the neuroses; (*f*) diabetes and old age. (3) *Habitual constipation,* constipation *per se,* may be familial. It may result from insufficient nervous energy (acquired or congenital) in the large intestine. Atony of the intestinal muscle may result from general debility or marasmus; it is very frequently a sign of neurasthenia, hysteria or hypochondriasis and results but seldom from actual disease of the musculature itself—that is, from atrophy or degeneration. Nervous constipation may be due to a spastic condition of the bowels.

Symptoms.—Symptoms other than constipation are often entirely absent, and when present are less often general than *local* (anorexia, coated tongue, bad taste in the mouth or abdominal uneasiness).

Two or three movements in one day or a movement every two or three days come within physiological limits. The stools normally accumulate in the sigmoid and pass every twenty-four hours into the

lower rectum, causing a desire to defecate, usually at the same hour each day. The constipated stool is dry and accumulates in the haustra of the colon or ampullæ of the rectum. Constipation may occur even with daily movements, a small amount being retained each time. It is difficult to state the quantity of the normal passage, which certainly does not depend wholly on the amount of food ingested; fæces consist largely of mucus and bacteria and form even during starvation. The stools may resemble sheep's dung, probably due to intestinal muscular spasm. Retained fæces are usually voided spontaneously in a few days with a serous diarrhœa or as scybala covered with mucus which results from irritation. Considerable obstruction may cause *fæcal colic,* due to contraction of the gut above the obstruction; there may be *stercoral fever* and *meteorism.* The retained *fæces may be palpable.* Still more marked obstruction may rarely simulate organic enterostenosis, being marked by collapse, vomiting and great vesical or rectal *tenesmus,* which should always excite suspicion. Extreme neglected cases may occasionally result fatally, especially in the old, debilitated or insane or in cases in which defecation causes great pain, as from fistula or piles.

Fæcal concrements (coproliths) almost always occur in the large intestine. When large they may simulate tumors of the intestine, omentum, kidney or spleen, from which differentiation may be possible only after thorough purging and flushing. They may be soft and easily indented with the finger, hard, nodular and tender from the bowel ulceration they incite or, far less often, tender from localized peritonitis. The history is not conclusive, because fæcal tumors may develop with daily though inadequate movements. Gersuny alleges that on pressure the bowel may at first seem to adhere to the fæcal masses and then become free (Gersuny's sign).

General symptoms are uncommon. Nervous symptoms are causal rather than sequential, as in the neurasthenic who believes he would be well if his bowels would move and who has most unpleasant sensations when they do. Vertigo, pressure in the head and mild dyspnœa are always neurotic and never coprostatic. Copropsychiatry is grossly exaggerated. *Intoxication* is rare, but has been considered causal of neuralgia, chlorosis and numerous other merely concomitant conditions. The aromatic sulphates of the urine may be increased, due to formation in the gut of indol, skatol and pyrocatechin.

Treatment.—Treatment of the causal factors (*q. v.*) is most important, as of irregularity of habit, insufficient exercise or neurasthenia.

1. Diet.—(*a*) *Coarse foods,* as whole wheat, graham, rye, corn meal and bran breads, act less mechanically than chemically by the production of lactic or oleic acid. (*b*) *Fruits* relax the bowels, because of the malic, tartaric and citric acids they contain, as plums, apples, peaches, pears, prunes, strawberries and grapes; grapes sometimes and bananas and persimmons usually cause constipation. (*c*) *Vegetables* act by the acids and gases they form; melons, sprouts, cabbage, cauliflower, cucumbers, turnips, carrots, spinach, tomatoes,

asparagus, onions, cress, celery and squash should be given freely; excessive amounts may cause fatigue of the bowel and constipation. (*d*) *Sweets,* in so far as they do not cause indigestion, are laxative; if used with discretion, sugars, syrup, candy, sauces, molasses, jellies, jams, marmalades and honey are valuable laxatives. (*e*) *Fats.*—Moderate amounts of butter and olive oil are helpful; vegetable are superior to animal fats, which tend to derange digestion. (*f*) *Cold water,* taken on rising and through the day, excites peristalsis. Charged waters operate similarly. Hard water, containing an excess of lime, constipates, an important point in drinking and cooking. (*g*) The following are *to be avoided:* red wines (containing tannic acid), excess of meat, eggs or carbohydrates (rice, buckwheat, macaroni and others containing little water), tea, milk (sometimes laxative when not taken in sips but swallowed in larger quantities) and beef tea. The diet recommended affords material for bacterial action, which follows the idea advanced by Strassburger and Lohrisch, that constipation is due to impaired motility, resulting from too complete utilization of the average diet by the intestinal bacteria.

2. Punctuality at Stool.—The necessity of being punctual at stool was insisted upon by Trousseau. The best time for evacuation is immediately after breakfast, except in cases of piles or prolapse which may come down during the day after the morning stool; in these cases the bowels should be moved at bedtime.

3. Posture at Stool.—Squatting is the natural posture; this may be approximated by leaning well forward or by placing the feet on a foot-stool; the thighs thus support and compress the abdomen, a matter of particular importance when it is relaxed. Working the thumb of the left hand downward over the abdomen from the navel to the left iliac fossa is also recommended.

4. Massage.—Gentle massage for a quarter of an hour, preferably before breakfast, may give relief if continued for months. Its direction is along the course of the colon. Sahli rolled over the abdomen a 7-pound cannon-ball covered with chamois. Massage is indicated in atonic but contra-indicated in spastic constipation.

5. Mild Faradization.—This arouses short contractions in the colon and is given with a moist sponge. Strong applications are to be avoided. Faradization and galvanization are inferior to massage.

6. Hydrotherapy.—The cold compress to the abdomen is a good adjunct.

7. Exercise.—Exercise is indicated to develop and contract the abdominal muscles, as in tennis or rowing or by lifting the legs while lying on the back or lifting the rigid body from the lying to the erect posture solely by the abdominal muscles. Walking, bicycle and horse-back riding do not exercise these muscles equally well. In women with ovarian and uterine diseases exercise increases constipation, due to reflex spastic inhibition of peristalsis, as shown by the laxative effect of narcotics (Littlejohn and Brunton). It is claimed that constipation is *spastic* (not atonic) in 25 per cent. of female

cases, as evidenced by colic, palpable or visible knots in the intestine and sometimes by ribbon-like passages; in these cases an irritating, coarse diet, massage, cold and electricity are to be avoided. Hot Sitz baths or full hot baths, hot compresses to the abdomen for spasm or colic are indicated; an enema of eight ounces of warm olive oil is given each evening and retained until morning; one-third of a grain of extract of belladonna is given as a suppository one to three times a day to relieve spasm and the frequent rectal tenesmus; the diet should consist of fine carbohydrates, butter and cooked fruit.

8. Enemata.—Enemata excite peristalsis, lessen intestinal hyperæmia and apparently are beneficial to the hepatic secretion and circulation. They do not derange digestion as do cathartics, but after a time they may lose their effect by dilating the colon, whence the indication for their intermittent use. Water and olive or linseed oil may be used; oil enemata (five to eight ounces) are especially indicated in spastic constipation and colica mucosa, in which the oil may be left in the bowel over night. Glycerine suppositories are valuable, if used alternately with other methods. In fæcal impaction with marked obstruction enemata are invaluable and should be combined with cathartics and digital evacuation of the hardened masses.

9. Cathartics.—The use of cathartics is governed by the following general rules: (*a*) they should be administered in chronic cases only after failure of the measures enumerated above; (*b*) their most successful employment is intermittent and alternating; (*c*) drastics, hydragogues and cathartics producing secondary constipation, should be avoided, save in extreme cases.

Varieties.—Aloes, found in nearly all proprietary and official pills, should be avoided in piles and uterine hemorrhage, because it congests the pelvic vessels; it is given in combination, *e. g.*—

℞ Extr. aloes ..... gr. ss.	Acting in fifteen to twenty hours, on the large bowel chiefly.
Extr. rhei ..... gr. ij. to v.	Actively purgative in four to eight hours; stomachic and tonic.
Extr. nucis vomicæ.... gr. ¼.	Acting on peristalsis and combating atony.
Resinæ podophylli .... gr. $\frac{1}{10}$.	Acting in ten hours, producing "bilious stools."
Extr. belladonnæ .... gr. ¼. (or extr. hyoscyami)	Relieving spasm (griping) and operating on the intestinal nerves.
Extr. taraxaci .....gr. j.	
M. et ft. pil. I.	

Rhubarb has the disadvantage of griping and of producing secondary constipation, which is caused by its tannic acid ("a patient sells his soul to rhubarb"), whence it should never be given alone:

℞ Pulveris rhei compositi	ʒiv.
Sodii sulphatis	ʒiv.
Sodii bicarbonatis	ʒj.
M. et S.—One teaspoonful at bedtime.	

Cascara sagrada is an excellent laxative, improves digestion and produces very little constipation afterward; the fluidextract, fluidextractum rhamni purshianæ, ♏xv, is very bitter; the solid extract, extractum rhamni purshianæ, is given in doses of from two to eight grains. *Licorice* is given as pulvis glycyrrhizæ compositus, ʒss to ʒj. *Castor oil* is a soothing laxative, relieving irritation and spasm (in nervous or lead constipation, in which latter it may be combined with opium gr. ¼); its offensive taste may be partly overcome by combining with glycerine and gaultheria:

℞ Olei ricini ..... ʒij.
Olei gaultheriæ ..... gtt. xl.
Glycerini ..... ʒij.
M. et S.—One teaspoonful to one tablespoonful.

In anæmic patients the following formula is recommended:

℞ Ferri sulphatis ..... gr. x.
Extracti aloes ..... gr. v.
Extracti rhamni purshianæ ..... gr. xx.
Extracti belladonnæ ..... gr. iij.
Extracti nucis vomicæ ..... gr. iij.
M. et ft. pilulæ No. X.
S.—One after meals.

Calomel should be given in the evening in fractional doses and may be advantageously combined with podophyllin, which acts in the same time (eight hours); they should be followed the next morning by half an ounce of magnesium sulphate. *Senna* easily deranges digestion; a small amount may be cooked with prunes; *confectio sennæ* is given in dram doses. *Tobacco smoking and coffee* in the morning are beneficial, but tea promotes flatulency and constipation. Consideration of the salines and drastics is intentionally omitted, as their use is contra-indicated except for emergencies.

FUNCTIONAL (NERVOUS) AFFECTIONS OF THE BOWEL.

1. Neuroses of Motility.—(*a*) *Nervous diarrhœa* has been partly considered. It may occur in the neuroses, in reflex irritation during dentition or in the crises of tabes; it is usually transient and is marked by absence of inflammatory symptoms, as mucus, pus or blood. There is constant danger that nervous diarrhœa may be confused with organic disease. In some cases ingestion of food may be immediately followed by diarrhœa and is treated by giving ♏ij of Fowler's solution before meals. (*b*) *Enterospasm* is caused by simultaneous contraction of the circular and longitudinal muscles, which normally contract alternately; the abdomen is temporarily retracted and in very exceptional cases obstruction (*ileus spasticus*) and fæcal vomiting occur. (See Dynamic Ileus, Spastic Constipation and Colica Mucosa.) (*c*) *Sphincter spasm,* aside from local rectal or anal lesions, results from sensory hyperirritability or decreased cere-

bral inhibition; it is most common in hysteria and tabes. (*d*) *Peristaltic unrest* may be associated with diarrhœa if the colon is involved, but more commonly affects the small bowel only, when constipation or normally frequent movements are usual. (*e*) *Nervous constipation* (atony) may result from the neuroses, intoxication from carbon dioxide and organic cerebrospinal diseases, in which conditions (*f*) *sphincter paralysis* may also result.

2. Neuroses of Sensation.—Among the neuroses of sensation, neuralgia mesenterica (*enteralgia,* enterodynia) is most important; it is inflammatory, nervous or colicky in origin; strictly speaking, it is always nervous, *i. e.,* not due to organic causes; practically, other forms of abdominal pain are frequently included under enteralgia. The chief symptom is the abdominal pain, which is usually umbilical, and sometimes relieved by pressure and sometimes not; the abdominal wall itself may be hyperæsthetic. Reflex disturbances in other organs may accompany enterodynia and aid in establishing its functional character; they are hiccough, vomiting, dyspnœa, palpitation, rectal or vesical tenesmus, testicular retraction, muscular twitchings, etc. The *diagnosis* necessitates exclusion of innumerable affections, chiefly abdominal but also general: (*a*) rheumatism of the abdominal muscles which is superficial; (*b*) lumbo-abdominal neuralgia, in which Valleix's three tender points are found (see NEURALGIA); (*c*) hysteria, which may simulate many affections; its stigmata should be looked for; (*d*) tabetic crises; (*e*) intestinal affections, as appendicitis, peritonitis, coprostasis, parasites, flatulent colic (from beer, milk or spoiled food), excessive catharsis, lead colic or colica mucosa; (*f*) *gout,* arthritis or cold; (*g*) malaria and typhoid; (*h*) renal and biliary calculi.

Its *treatment* and that of the motor disturbances is of the fundamental nervous state. Symptomatically, hypodermics of narcotics produce the quickest but most dangerous relief; atropine is much safer.

℞ Spiritus ammoniæ aromatici ..... ʒiij.
Tincturæ cardamomi compositæ ..... ʒiij.
Spiritus chloroformi ..... ʒij.
Tincturæ opii camphoratæ ..... ʒiv.
Tincturæ asafœtidæ ..... ʒiv.

M. et S.—One tablespoonful in hot water every fifteen minutes for three or four doses.

3. Neuroses of Secretion.—The secretion neuroses are important. The chief types are serous nervous diarrhœa and colica mucosa, which have already been considered.

AFFECTIONS OF THE MESENTERY.

I. Inflammation.—Mesenteritis is chiefly important in its relation to ascites, tuberculous peritonitis and other peritonitides (*q. v.*).

II. Hemorrhage.—Primary hemorrhage is most rare. It is generally secondary to acute hemorrhagic pancreatitis, retroperitoneal hæmatoma, aneurysmal extravasation or the hemorrhagic fevers.

III. Diseases of the Mesenteric Vessels.—(*a*) *Infarction* by embolism or thrombosis was first described by Virchow and involves chiefly the arteria mesenterica superior, which is essentially a terminal vessel. Most cases develop in men past middle life. Jackson, Porter and Quimby have collected 225 cases. (See SYMPTOMS OF VALVULAR HEART DISEASE.) Its onset is sudden, with intense abdominal pain, collapse, nausea and vomiting (usually bloody and perhaps fæcal, due to intestinal obstruction), diarrhœa (40 per cent.) and bloody stools (40 per cent.). Unless there is an apparent cause for embolism, as valvular disease, the diagnosis of infarction is rarely made, for peritonitis or obstruction is usually suspected. The usual outcome is early death from collapse, but if the patient lives long enough acute peritonitis sets in, with distended abdomen and diffuse tenderness. In very rare cases collateral circulation allows of recovery. In the Porter, Jackson and Quimby series, 94 per cent. were fatal. The treatment is purely symptomatic and supportive, but successful operation is recorded by J. W. Elliott, Leclerc and Bütter. (*b*) *Periarteritis nodosa* (*v.* page 467). (*c*) *Dilatation* of the mesenteric veins and phlebosclerosis occur, especially in cases of liver cirrhosis. (*d*) *Suppuration* (thrombophlebitis suppurativa) may mark umbilical pyæmia of the new-born or may carry infection from the appendix or rectum. (See HÆMATEMESIS.) (*e*) Simple thrombosis of the mesenteric veins. Kraft in 1897 collected 16 cases; in over half of the cases syphilis or liver cirrhosis was the cause.

IV. Affections of the Chyle Vessels.—These offer more pathological than clinical interest. These vessels may become varicose or even hyperplastic (chylangioma). Cysts containing chyle may cause tumors and, by rupture, may result in chylous extravasation in the mesentery or chylous ascites (*q. v.*). Twenty-four chylous cysts are described by Broca.

V. Mesenteric Tumors.—These may be dermoid, hydatid, serous, sanguineous, chylous or malignant. In some cases they attract pathological attention only or they may fill the entire abdomen. Their symptoms are those of an abdominal tumor, which is centrally located, movable and covered with a zone of resonant intestine. Confusion is possible with hydronephrosis or ovarian cysts.

DISEASES OF THE LIVER.

ACUTE YELLOW ATROPHY.

Definition.—An acute fatal degeneration of the liver cells, accompanied by shrinking of the liver, icterus and nervous toxæmia. Ballonius (1600) reported the first case. It is a rare affection; Best collected 450 cases.

Etiology.—(*a*) Sixty-six per cent. occur in *women* and 50 per cent. between the years of *twenty and thirty;* but 22 cases occurred under 10 years of age. (*b*) Thirty-three to 50 per cent. of women with the disease (or 27 per cent. of all reported cases) were in the *last half of pregnancy;* the first three months of gestation almost render the individual immune (Eichhorst); it is infrequent in the puerperium. (*c*) Ten soldiers were affected in Arnould's *epidemic* series. (*d*) *Infections,* as osteomyelitis, diphtheria, sepsis, erysipelas, typhoid, recurrent fever and secondary syphilis (10 per cent., Lebert); or (*e*) *poisoning,* as from phosphorus, alcohol and chloroform narcosis, may antedate the disease.

It may occur primarily in a sound or secondarily in a diseased liver. Worry is a predisposing factor in pregnancy or syphilis.

Pathology.—The liver is small, the minimum recorded weight being 400 grams; in marked cases it is so lax that it can be rolled up; the capsule is wrinkled, the color usually yellow (*atrophia hepatis fusca*) and the lobular markings obscure. The liver cells are granular, fatty, indistinct in contour or tinged with bile; some are necrotic and others show evidences of regeneration. Foci of red tissue are usually considered as an advanced stage in which the fatty necrotic cells have been absorbed; here the liver is homogeneous and composed of detritus; the left lobe is often red, showing the most advanced process, and the right lobe is yellow, showing more recent changes. The cells of the bile channels are degenerated. Crystals of tyrosin are found. The apparent increase of the connective tissue is relative only. Sometimes infiltration with red or white cells occurs. Icterus staining, small hemorrhages and fatty degeneration are found in the heart, kidneys, muscles, epithelial cells and bronchi, lungs and digestive tract.

In *nature* the disease is sometimes considered inflammatory (acute parenchymatous hepatitis), but is usually classified as a necrotic process. It is not yet determined whether the disease is an *infection,* to which it has a certain resemblance, or an *intoxication,* which initiates autolysis of the liver. The changes in other organs are held to result from the liver change or from a common unknown cause. Bacteria are found inconstantly.

General Clinical Picture.—Acute yellow atrophy begins with a *prodromal stage* in which gastric disturbance predominates; icterus develops in a few days when the *second stage* suddenly develops cerebral symptoms, unrest, delirium, oppression, vomiting and convulsions; shrinking of the liver, hepatic tenderness, enlarged spleen, cutaneous and mucous membrane hemorrhages, abortion and hemorrhage if pregnancy exist; urinary symptoms, as bile, albumin, leucin, tyrosin and other unusual products of metabolism in the urine; and subnormal or normal temperature. The issue is almost invariably fatal.

Individual Symptoms.—The *prodromal stage* may last from a few days to three and a half weeks or more and is not characteristic. In the latter half of gestation, icterus and hepatic tenderness or enlarge-

ment are suspicious symptoms. The gastric symptoms become marked. Icterus, which is absent only in extremely rapid cases, is obstructive from intrahepatic changes; the stools are acholic and the jaundice increases with the second stage.

In the second stage, (*a*) the *hepatic dulness lessens* to one-half or one-quarter of its former dimensions and first in the left lobe. The liver is small, flabby and falls back toward the spine so that in some cases there is no liver dulness. The liver dulness may remain normal if death is rapid, as from hæmatemesis (Leube), if the liver is cirrhotic or if it is adherent to the abdominal wall. The liver is *tender and painful,* and pain is occasionally referred to other parts of the abdomen. The region of the liver may appear sunken and the liver may feel flabby or may pit to the finger (Leube). (*b*) The *spleen is enlarged* in 66 per cent.; it is not enlarged when perisplenitis, profuse hemorrhage or diarrhœa is present. (*c*) *Vomiting* is *almost invariable,* the vomitus consisting of mucus, bile and finally blood; it is accompanied by hiccough, a dry brown or white tongue, sordes and constipated, uncolored stools. (*d*) The *nervous symptoms* may come on gradually with headache and dulness or very abruptly with *delirium,* anxiety, *meningeal symptoms,* grinding of the teeth, trismus, wide pupils, amaurosis, cerebral vomiting and convulsions (in 33 per cent. of adults and almost constantly in children). The nervous symptoms are due to hepatic insufficiency (hepatargia), the degenerated liver being unable to protect the nervous system, as it does in health, against various ptomaines and leukomaines. (*e*) The *urine is decreased* or even suppressed. *Bile pigment, albumin,* casts and epithelial cells are found. Unusual products are found in the urine, due to the breaking down, *autolysis, of the liver cells; leucin and tyrosin* occur most frequently, though neither constant nor pathognomonic; sarcolactic acid, oxyamygdalic acid, peptone and albumose are also found. The urea is decreased and may be entirely absent. The ammonium compounds, which normally constitute 2 to 5 per cent. of the nitrogen excretion, are increased, even to 17 or 20 per cent., because they are needed to neutralize the increased acid products in the blood and because the liver cells cannot convert ammonium into urea. The uric acid and xanthin are increased. Indicanuria may be noted. Strangely, glycosuria is rare, though the glycogenic function of the liver can hardly be normal. Bouchard, Hervouet, Brouardel and Chauffard speak of urinary crises with increase in the amount of urine voided and in its nitrogenous constituents. (*f*) *Fever* may occur early in the disease, but the temperature is usually normal or subnormal at the height of the process. The *pulse* may be at first slow, but later becomes *very rapid. Dyspnœa* is almost invariably present. (*g*) *Hemorrhages* occur in the majority of cases, largely from the stomach and into the skin. Bowel hemorrhage is less frequent, as are those from the nose, mouth and genital mucosæ. Uterine hemorrhages occur if pregnancy exist. Hemorrhages in the retina are frequent, together with white flecks

due to tyrosin deposit and fatty change. Punctate hemorrhages into the serous membranes are common autopsy findings. Erythematous eruptions, muscular pain and articular swellings may occur. In a case seen by the writer herpes was the first symptom. Leukocytosis is infrequent.

Course.—The clinical course is rapid; 50 per cent. of cases die between the fifth and fourteenth day and 30 per cent. between the third and fifth week. The second stage usually lasts one and a half to three days, rarely more than a week, but may be longer in the rare cases which recover. The more abrupt and severe the cerebral symptoms the more rapid is the course. Pregnancy hastens the issue. Remissions may be observed; temperature may develop during the death agony. The pulse becomes very rapid, the breathing difficult and irregular and the symptoms of nervous excitation are followed by paralysis of the brain centres.

Diagnosis.—The diagnosis is based upon (*a*) the *icterus,* (*b*) *nervous symptoms* and (*c*) *shrinking of the liver.* Diminution of the liver may be simulated by tympanites or by the colon lying over the liver; it may not shrink when previously cirrhotic or when bound to the abdominal wall by adhesions, even though the liver cells atrophy. The urine shows distinct disturbances of metabolism, which are valuable diagnostically with the above mentioned symptoms. Confusion may occur in *icterus gravis* or in the so-called bilious typhoid, pneumonia, recurrent fever, puerperal fever, yellow fever, phosphorus poisoning or Weil's disease; most of these affections are febrile, while acute yellow atrophy is almost always afebrile; the intensity of the icterus, shrinking of the liver, severe cerebral symptoms and the urinary findings are diagnostic. The greatest difficulty in diagnosis is *acute phosphorus poisoning,* for which Quincke gives the following differentiation (Leube maintains that the etiology alone is distinctive):

Acute Yellow Atrophy	Acute Phosphorus Poisoning.
One to two weeks of moderate symptoms from icterus.	Acute gastro-intestinal symptoms; then icterus on the third day.
Icterus intense.	Icterus less.
Liver sensitive, smaller. If colon is distended, entirely obliterating the liver dulness, the urinary symptoms are suggestive.	Liver sensitive and swollen.
Liver cells greatly degenerated, and but little increase in the fat (5 per cent.).	Very extensive fatty degeneration (30 per cent.).
Sudden cerebral symptoms with alternating irritation and depression one to two days before death.	Cerebral symptoms more of depressive order.
Urine; oxyamygdalic acid, leucin, tyrosin, more abundant and constant.	Sarcolactic acid (though peptone, sarcolactic and tyrosin are found in both diseases).

Acute Yellow Atrophy	*vs.*	Acute Phosphorus Poisoning.
Hemorrhages smaller and fewer.		Hemorrhages more numerous.
Duration of one to two weeks.		Duration shorter.

Admitting that acute yellow atrophy may regress, recovery usually indicates error in diagnosis. From *simple atrophy* of the liver differentiation is easily made by the general marasmus and the absence of icterus and of nervous and urinary symptoms. *Cirrhosis* is differentiated by absence of ascites and the presence of a larger spleen, firmer liver and collateral circulation; ascites in acute yellow atrophy has occurred in but 8 instances (Tileston) (*v.* page 695).

Treatment.—Treatment is wholly expectant and entirely negative in its results. The heart depression, vomiting and nervous excitement are treated as in typhoid (*q. v.*).

PORTAL CIRRHOSIS.

Synonyms.—Laennec's cirrhosis, alcoholic or atrophic cirrhosis.

Definition.—Portal cirrhosis is a fibrosis of the liver, characterized (*a*) etiologically by alcoholism; (*b*) anatomically by induration around the portal vein radicles enclosing numbers of liver lobules, whose cells degenerate; and (*c*) clinically by portal obstruction (dyspepsia, hæmatemesis, enlarged spleen and ascites) and often by signs of hepatic insufficiency.

Classification.—The classification of cirrhosis may be made upon an etiological, pathological or clinical basis. From a practical standpoint the chief points are included in the following classification:

(1). Capsular or Glissonian.	Portal vein syphilis. Chronic perihepatitis.
(2). Vascular.	*Portal vein* cirrhosis, first in clinical importance; its leading type is the alcoholic, atrophic type of Laennec. (A sub-type is a hypertrophic cirrhosis, like the atrophic save that the liver remains large.) *Hepatic vein* cirrhosis, occurring in stasis (cyanotic induration), and, according to French authors, with portal vein cirrhosis.
(3). Biliary Cirrhosis.	Hanot's disease.
(4). Mixed Cirrhosis.	(Portal vein and biliary cirrhoses combined.)

Of these, two main forms are conspicuous—the portal and biliary.

Etiology.—(*a*) Sixty to 75 per cent. of cases are found in *men* between *forty and fifty years* of age. Musser (1889) collected 685 cases in children. (*b*) *Alcoholism* is the chief, though not the sole cause; cirrhosis may be due to whisky, wine, beer or absinthe, especially in sedentary individuals; alcohol is absorbed by the gastric and intestinal veins and is conveyed to the portal vein radicles, where its toxic effects are expended on the liver cells or interstitial tissue,

but in what sequence it is not known. Of late the role of alcohol has been disputed, but it remains a striking though perhaps an indirect factor. (*c*) *Syphilis* is a cause, especially in the young, in whom the spirochætes are conveyed by the umbilical vein to the liver. (*d*) *Other infections,* as malaria (especially where it is endemic), typhoid, tuberculosis, etc., are possible causes; experimentally cirrhosis has been caused by the Bacillus pyocyaneus, Bacillus coli and staphylococcus; and lactic, acetic and butyric acids; local mycotic necrosis is thought to be followed by fibrosis; in this group the toxins probably enter by the hepatic artery. (*e*) *Spices,* curry, drastics, lead, phosphorus, arsenic, hæmochromatosis, anthracosis (Welch), silicosis (Adami) and possibly gastro-enteric autointoxication are exceptional etiological factors. It may be associated with gout, diabetes, rickets or other constitutional affections. There may be *multiple causes, e. g.,* syphilis plus malaria or alcoholism. In some cases no cause is discoverable.

Pathology.—The essential change is fibrosis around the branches of the portal vein. Some consider that the connective tissue growth is primary, causing, as it contracts, atrophic or fatty alteration of the liver cells, sometimes with pigmentation; others maintain that the cells are first injured and are replaced by fibrous tissue. Whatever the pathogenesis, the connective tissue forms and surrounds several lobules at a time (*multilobular cirrhosis*). Histologically the cirrhosis is *atrophic,* and this point is emphasized first, because the size and weight of the liver may vary. Some clinicians maintain that the liver is first large (the so-called hypertrophic stage) and later is shrunken. Undue importance is attributed to size alone, for a normal-sized or enlarged cirrhotic liver is histologically atrophic. An actually atrophic liver may be large from (*a*) fat deposit, (*b*) passive congestion, (*c*) compensatory parenchymatous hyperplasia or (*d*) active congestion. The *weight* is *as often increased as decreased,* ranging from 2,000 gm. (65 ounces) to 1,000 gm. or less. Its *edge is rough; i. e.,* granulations can be felt which represent the remaining relatively normal liver tissue. Regeneration of the parenchymatous cells is active but somewhat disorderly, irregular and unstable, the cells again wasting. The *surface* is sometimes smooth, and microscopic examination may be necessary for confirmation. Granulations also occur in fatty liver, pylephlebitis and passive congestion. Its *consistence is increased.* The liver is *gritty* to the knife; its color is yellow (cirrhosis), perhaps green or brown. The *connective tissue is increased* and prominent, encroaching on the parenchyma. Injections of the organ through the portal vein are not successful; *i. e.,* there is portal obstruction, which causes many of the clinical symptoms. There is a lengthening and narrowing of the capillary vessels; this and the increased arterial pressure in the organ account for the ascites, aside from the connective tissue hyperplasia (*v.* DIFFERENTIAL PATHOLOGY on pages 677–678).

General Clinical Picture.—The disease often exists for several years

without symptoms; this latency is marked by enlargement of the liver, and is due to compensatory cellular hyperplasia. In the minority of cases, swelling, pain or active hyperæmia of the liver and dyspepsia, fever or icterus are observed; they last a short time only and are by no means characteristic. With marked shrinking there are symptoms (*a*) of general *disturbance of nutrition,* as constipation, emaciation (which is often not noticed because the abdomen remains obese) and an earthy, sallow look to the skin, in which dilatation of the small arterioles (toxæmia or alcoholism), dryness and hemorrhages are also observed; (*b*) of *portal congestion,* as early hæmatemesis, epigastric pressure, ascites, large spleen and disturbed breathing, circulation and digestion; (*c*) of *intoxication,* resulting from functional failure of the heart, liver and kidneys; (*d*) of *complications,* as hemorrhage, diarrhœa or the so-called cholæmia.

Symptoms.—1. The Liver.—The liver is *enlarged about as often as it is decreased in size;* the left lobe suffers most and earliest contraction. The liver may be found to be high, as a result of paresis of the diaphragm, due to perihepatitis. Its *edge is hard,* but often cannot be felt, even after tapping, on account of its shrinkage or because the bowels cover it. The *edge is uneven;* its granulations may be confused with the small islets of fatty tissue in the abdominal wall, due to unequal wasting of the adipose tissue. *Palpation* of the liver is *the only certain way* of determining its lower limit, the results of percussion being decidedly unreliable. In infantile forms of portal cirrhosis the liver and spleen are larger than in adult types.

2. Portal Stasis.—This results from obstruction to the return portal circulation. (*a*) The peritoneum is thickened, œdematous and the seat of transudation, *i. e., ascites,* which occurs in 85 per cent. when the patient dies of the cirrhosis itself and in 50 per cent. when he dies from other causes. It is more common in small than in large livers. Some writers hold that toxæmia and peritonitis are more potent factors than portal obstruction. *Ascites* usually *appears first without general œdema,* though later the legs may become swollen as the ascites compresses the inferior vena cava. Pre-ascitic œdema of the legs or abdominal wall may develop from cardiac or renal complications, involvement of the cava by perihepatitis or from thrombosis of the iliac veins. Under ascites its physical signs will be more closely considered. The amount of fluid may be enormous; Liebermeister reports a case in which he withdrew nine gallons.

The fluid is usually amber-colored, seldom red from admixture of blood or very rarely turbid from fat (adipose ascites) or lecithin (chyliform ascites); the two latter forms also contain nucleo-albumin. The *specific gravity* is 1,008 to 1,015, with 0.6 to 1 per cent. albumin. Though ascites is rarely absent in advanced atrophic cirrhosis, the patient may die before ascites develops—for instance, from hemorrhage, which is dangerous when there is no ascites (Leyden). An extensive though slowly developing *collateral circulation* may permanently relieve an earlier ascites. It may disappear after

hemorrhage, diarrhœa or carcinoma of œsophagus. (*b*) *The spleen* is from two to six times its normal size, which is due to the early toxæmia and the later stasis. The *splenic tumor* is present in 75 per cent. of cases and is a measure of the degree of cirrhosis. Its capsule is hard and thickened (sometimes preventing splenic enlargement), the trabeculæ are thicker and a systolic murmur is sometimes heard (Bouchard and Leudet). (*c*) *Gastro-intestinal catarrh* is due to stasis and alcoholism. Pain after eating, especially in obese persons, a coated tongue, vomiting, singultus, constipation, piles, diarrhœa toward the end of the course and meteorism also occur. (*d*) *A collateral circulation* may be evidenced by enlargement of the veins about the navel (*cirsomphalos* or *caput Medusæ*). Prominence of the epigastric veins occurs regularly, but results from compression of the inferior cava. *Anastomoses* occur (i) between the middle and inferior hemorrhoidal veins (passing by the hypogastric to the cava) on the one hand, and the superior hemorrhoidal (passing by the superior mesenteric to the portal vein) on the other hand; (ii) between the coronary veins of the stomach on the one hand and the œsophageal and phrenic veins on the other, which empty into the vena azygos; (iii) between the veins in the ligaments of the liver and new adhesions and the phrenic veins; between the veins of the *ligamentum teres* and the veins of the abdominal wall and epigastric veins, which may sometimes be felt and may produce murmurs (Bamberger and von Jaksch); (iv) the mesenteric, peritoneal and parumbilical veins also communicate with those of the abdominal wall. A well-developed collateral circulation is both a favorable and an unfavorable symptom; on the one hand it *relieves* the *portal stagnation* and probably gives the over-taxed liver a better opportunity to perform its functions; on the other hand, the dilated collateral vessels are likely to *rupture* and they convey directly into the general circulation *toxic substances* which the liver normally destroys or alters; this produces, according to Stockton, the frequent arteriosclerosis, granular kidneys and more acute toxæmia, which resembles uræmia. (*e*) *The kidneys are congested,* degenerated and often show hypertrophy. The urine is decreased in amount, darker and of higher specific gravity, because the arterial tension is low in cirrhosis. More urine is often secreted during fasting than during digestion (opsiuria), which is the converse of normal conditions. The *urobilin may be increased* two-fold. The chlorides are decreased, as they accumulate in the ascitic transudate. The urea is often decreased, but the *ammonium compounds* (as in acute yellow atrophy) are often increased, less from interference with the liver functions than from increase of acid products in the blood (acidosis), which the ammonium salts seek to neutralize. *Albuminuria* may result from stasis or nephritis (*v. i.*). Blood may be found in the urine as a result of stasis in the bladder. Glycosuria is uncommon save in the pigmentary cirrhosis (see Diagnosis). Alimentary glycosuria may be produced by administration of 3 ounces of grape sugar with a meal.

3. General Symptoms.—There is considerable *loss in weight,* some *anæmia* and a sallow, *earth-like color.* Malnutrition is explained (*a*) by the inability of the liver to handle the carbohydrates, proteids, etc., and (*b*) by the diversion through the collateral circulation of toxins into the general circulation, the liver being the normal barrier against toxins injurious to the nervous system. In three cases with well-developed collateral circulation, scurvy developed which the writer interpreted as toxæmic. It was once thought that bile salts or pigment entered the blood and produced "*cholæmia,*" or that substances were retained which the bile normally excreted, producing "*acholia*"; now the term *liver insufficiency* (*hepatargia*) is more often employed. Toxæmic nervous symptoms include adynamia, headache, itching, delirium, stupor, convulsions, paralysis, contractures or coma. The writer twice observed Kussmaul's breathing. Much depends upon the activity and integrity of the kidneys, which may for a time maintain adequate excretion.

Complications.—1. Hemorrhage.—(*a*) *Cachectic* capillary hemorrhage from the nose, mouth, lungs or stomach is less frequent than it is in biliary cirrhosis. Epistaxis occurs from dilatation of the vessels on the anterior part of the septum. (*b*) Of *mechanical* congestive hemorrhages from the œsophagus, stomach, bowels, kidneys, bladder or uterus, those from the *œsophagus* are most important. Zenker found œsophageal varices in 20 per cent. of cirrhosis autopsies. Their rupture causes *hæmatemesis* (which occurs in 23 per cent. of cirrhosis cases), chiefly in adults and rarely in children; if very profuse, blood may come up without vomiting; in one of Osler's cases the blood amounted to 10 pounds; it often causes blood in the stools. It may seep out slowly and form dark clots; it is usually attended by less collapse than in hemorrhage from gastric ulcer but acute anæmia or slight fever may follow it. Preble, in a careful study of the subject, concludes that: (i) Hemorrhage occurs chiefly in atrophic forms. (ii) In 33 per cent. the first hemorrhage is fatal; in 66 per cent. hemorrhages occur over a period of several months to eleven years. (iii) In 33 per cent. a diagnosis of cirrhosis was possible at the time of the first hemorrhage. (iv) Œsophageal varices were present in 80 per cent. and in over half of these macroscopic rupture was detected; yet (v) fatal hemorrhage may occur without macroscopic rupture and is due to a simultaneous rupture of many capillaries. (vi) Hemorrhage may be the first symptom of cirrhosis, but other signs are usually found first. (vii) The symptoms of cirrhosis were typical in but 60 per cent. of cases of œsophageal varix.

2. Peritonitis.—(*a*) *Chronic retracting* peritonitis (in 15 per cent., Gratia) may occur with shortening of the mesentery and intestines; according to Botazzi it prevents absorption of the ascites. (*b*) *Tuberculous* peritonitis (20 per cent.) may occur with fever, pain and often an hemorrhagic exudate. It usually follows the ascites of cirrhosis and hastens the fatal outcome. One would think that stasis would tend to prevent the development of tuberculosis.

3. Nephritis.—Nephritis occurs in 33 per cent. of cases.

4. Heart.—The heart is "arteriosclerotic" in 60 per cent.; chronic myocarditis, endocarditis, fatty degeneration and dilatation of the right ventricle often occur. The arterial tension is low in cirrhosis.

5. Lungs.—In the lungs bronchitis, emphysema and in 15 per cent. pulmonary tuberculosis occur; the latter is rare in infantile cirrhosis. Right hydrothorax occurs from stasis in the vena azygos and right intercostal veins; right-sided pleurisy, due to extension of perihepatitis through the diaphragm, occurs in 10 per cent. of cases.

6. Brain.—Pachymeningitis or apoplexy may occasionally occur.

7. Fever.—*"Intermittent hepatic fever"* may result from hepatargia (hepatic intoxication), perihepatitis or complications. The temperature is usually subnormal in advanced cases, though terminal temperature and leukocytosis are observed in terminal infections.

8. Icterus.—In atrophic cirrhosis jaundice is a genuine complication; it is due to duodenal catarrh or a radicular cholangitis; few pass through the disease without a muddy yellow areola under the eyes. If present it is incomplete. Its frequency is 15 per cent.

9. Anasarca.—Anasarca results from pressure on the cava by an extensive ascites, from perihepatitis with cicatricial involvement of the cava and from complicating cardiac or renal disease and marasmus.

10. Other Complications.—These are rarer; for instance, pylethrombosis, clubbed fingers, alcoholic multiple neuritis or delirium tremens; acute yellow atrophy and venous thrombosis seldom occur.

Course and Prognosis.—The clinical course is essentially chronic and seldom exceeds two or three years, but its duration is more often months than years; the maximum is fifteen years. All acute cases must be stricken from the category of cirrhosis. *Compensatory factors* include (*a*) the collateral circulation, though this may throw toxins directly on the nervous centres and produce symptoms like uræmia; (*b*) hyperplasia of the liver cells, which may produce veritable adenomata. As Hanot puts it, the diagnosis is based on the condition of the connective tissue, but the prognosis depends on that of the liver cells. (*c*) The kidneys may compensate to a small degree. Hale White holds that ascites is ominous and that the patient rarely survives the second tapping; this does not conform to the author's experience, though ascites is a late symptom; the author has seen numerous cases which survived frequent tappings, and he has felt justified in excluding perihepatitis, which White considers explanatory of these cases. The prognosis is relatively favorable in subjects under forty years with large livers and small spleens. Recovery is possible, especially if the diagnosis is made early. In advanced stages death is the usual outcome, although the author has seen relative recoveries lasting for five or six years follow adhesions from multiple paracentesis.

Death results from hepatargia (cholæmia, acholia), marasmus, hemorrhage, ascites or intercurrent terminal infections; exhausting diarrhœa or vomiting; cardiac, renal or other complications (*v. s.*).

Differential Diagnosis.—Two classes of conditions require differen-

tiation: those in which the liver is large and those in which it is small.

1. When the Liver is Large.—(*a*) The *fatty liver* is soft and presents no evidences of portal stasis. (To avoid repetition the disease is differentiated later, in tabular form on page 695.) (*b*) The *amyloid liver* is hard; the spleen, kidneys and alimentary mucosæ are almost always involved, icterus is very rare and occurs only when amyloid glands at the *porta hepatis* produce mechanical icterus; anæmia is common. (*c*) *Leukæmic* deposits in the liver cause hepatic intumescence, but the blood examination is decisive. (*d*) Some swelling of the liver may result from *simple icterus,* but the history, absence of portal stasis and shorter course are distinctive. (*e*) *Passive hyperæmia* from heart or lung disease produces an evenly enlarged tender liver, subicterus and cyanosis. (*f*) Cancer of the liver is usually clearly marked by hard and nodose enlargement, by detection of the primary growth (*e. g.,* in the stomach), by icterus and cachexia; in some cases early cirrhosis may simulate cancer, as in a patient in whom Kahler diagnosticated cancer, though the later course proved that cirrhosis was present. (*g*) In *syphilis* of the liver the history, knobs and depressions, the more rapid course, greater perihepatitic pain, the icterus (in 33 per cent.), the findings in other parts and the results of treatment usually make the diagnosis clear (see Syphilis). (*h*) *Pericarditic pseudocirrhosis,* described by Pick (1896), occurs in young, rheumatic and cardiac subjects and is characterized by symptoms which on the one hand resemble extreme passive congestion of the liver and on the other a capsular cirrhosis; the liver is large and firm, there is recurrent ascites without œdema of the extremities and little or no icterus. Its mechanism is disputed; contracting pericarditic adhesions may perhaps cause dilatation of the right auricle and vena cava inferior, thereby distending the hepatic vein system; or possibly the inflammation extends down the cava inferior to the liver; the liver, however, is permanently and disproportionately congested. (*i*) The *enlarged liver in Banti's disease* is cirrhotic, but the spleen is enlarged first and there is early splenic anæmia, while the cirrhosis and hæmatemesis occur later.

2. When the Liver is Small.—The following possibilities should be considered: (*a*) *Syphilis,* characterized by stellate scars, nodes and pain. (*b*) *Passive congestion;* the nutmeg liver may atrophy, but anasarca is observed first and usually there are primary and sufficiently characteristic heart findings. (*c*) Simple *marantic atrophy* is of little clinical interest; the liver is small, there is no portal stasis and arteriosclerosis is found elsewhere. (*d*) *Pylethrombosis* is characterized by a rapid onset with swelling of the spleen, severe or repeated hæmatemesis, dilated veins and rapid recurrence of the ascites after tapping; it is due to some special cause, as pressure of a tumor, nearby ulcers, suppuration or gall-stones. (*e*) Perihepatitis has a very much slower course than has cirrhosis; it lasts for years with intermissions and very often results from peri-

carditis. (*f*) In *chronic peritonitis* the fluid is turbid, often hemorrhagic, with high specific gravity and many leukocytes; it is characterized by absence of symptoms of portal vein stasis; there may be pain, fever and, in the tuberculous form, positive results from inoculations; for the differentiation of chronic peritonitis see pages 731, 732 and 733. (*g*) The peculiar *pigmentary cirrhosis* of hæmochromatosis (diabète bronzé) was first described by Troisier (1871), was named by Recklinghausen and was particularly elaborated by Hanot, Chauffard and Letulle. Its symptoms are threefold: (i) cirrhosis (which causes the hemorrhages, indurated liver and enlarged spleen), (ii) diabetes and (iii) pigmentation of the skin, which somewhat resembles Addison's disease. It is thought that some toxic substance destroys the red blood cells (hæmolysis), and so operates on the cells of the liver, pancreas and other organs that they transform the soluble blood pigment into insoluble hæmosiderin and iron-free hæmofuscin. These pigments are found in the muscles, intestine, heart, liver and pancreas, whose cells become pigmented, degenerated and necrotic; the pigment passes into the connective tissue of the part. The essential lesions are pigmentation of the liver, skin and pancreas, with the fibrosis of the liver and of the islands of Langerhans in the pancreas, which latter causes the diabetes. Diabetes has occurred in all but five of the French cases, but seems less common in those reported in this country. In Futcher's series of 35, 33 were males.

Treatment.—1. Causal Therapy and Prophylaxis.—These have but limited application, save in preventing further injury to the affected liver by syphilis and by the use of alcohol. All forms of liquors or medicinal tinctures are absolutely interdicted, but too frequently with no effect. Syphilitic forms are greatly benefited by appropriate treatment, which some clinicians think may help all forms of cirrhosis.

2. Medicinal Treatment.—This is of little value, for cirrhosis cases come under observation only after contracting connective tissue has already formed. Naunyn has used ten grains of ammonium chloride three times daily, but it does not arrest cicatrization. Rolleston believes that potassium iodide and ammonium chloride are beneficial in acid intoxication (acidosis); he never gives arsenic and salicylates.

3. Diet.—The diet should exclude rich proteids, for meat is less well tolerated than milk. Skimmed milk is best for it contains but 1 per cent. of fat. Rich or highly seasoned foods are to be avoided.

4. Symptomatic Treatment.—The same treatment which is instituted in chronic gastritis or alcoholism (*q. v.*) may be indicated. Flatulence should be met by calomel in fractional doses, then by salol 10 grains after each meal, or possibly by betanaphthol 10 grains, though it may cause renal irritation. Diarrhœa should not be checked until the bowels have been thoroughly cleansed, for it is eliminative. Unusual splenic intumescence may be treated by purg-

ing, for it is often a forerunner of hæmatemesis. Hæmatemesis should be treated by absolute rest and by rectal feeding, as in gastric ulcer (*q. v.*); calcium chloride 3ss to j may be given by rectum for two or three doses. Pain is controlled by local heat, calomel, ammonium chloride and salines. Nervous symptoms necessitate the same treatment as uræmia, namely, laxatives, sweats and transfusions, to which sodium bicarbonate may be added as in diabetes, for the acidosis.

5. Ascites.—Ascites is seldom benefited by the cardiants, sweats, diuretics or purges. Early repeated punctures may induce adhesions and consequent collateral circulation (see page 733).

6. Operation.—Talma and Van der Meulen (1889) recommended operative measures, which were later amplified in various ways, to induce a collateral circulation; these consist of sewing the omentum to the abdominal wall, rubbing the surface of the liver until it is abraded to promote adhesions and other similar measures. Montprofit, in 224 operations, found 30 per cent. cured. Bunge collected (1905) 274 operations of the Talma-Drummond type; the immediate mortality of the operation was 3.6 per cent.; of the surviving cases, 14 per cent. were somewhat improved, 30 per cent. recovered and 56 per cent. were not improved. Perhaps better results will be recorded if operation is resorted to early in the disease, but it must be recalled that the ascites and collateral circulation involve vital as well as mechanical considerations. How the operation is beneficial is still a matter of doubt; its mechanical collateral side is known; perhaps lessening of venous tension in the portal circuit helps the liver cells to a better functional activity.

BILIARY CIRRHOSIS.

Definition.—A cirrhosis occurring chiefly in adolescence and characterized by fibrosis of a mono- and intralobular type, enlargement of the liver without contraction, splenic enlargement, chronic icterus without gross obstruction of the bile vessels, paroxysmal fever and by absence of portal stasis. It is known as *Hanot's disease,* for it was first clearly described by him in 1876. The term hypertrophic cirrhosis is unfortunately ambiguous.

Etiology.—Its etiology is obscure. Alcoholism is not a factor. Sometimes it apparently results from (*a*) *infection,* either an ascending *cholangitis* due to the Bacillus coli or pneumococcus or an infection through the blood stream. (*b*) *Age.* Most cases develop between the twentieth and thirtieth years of age; it is rare in persons over forty; a considerable percentage occurs in the young, especially in India (Gibbons, Ghoses). (*c*) *Sex.* Over 80 per cent. of adult cases are *males;* in the young the sexes are equally involved.

Symptoms.—The onset is usually characterized by the early development of icterus and less often by malaise, muscular asthenia, fever, splenic pain, dyspepsia, diarrhœa or abdominal pain.

Cardinal Symptoms.—1. The *liver* is *uniformly enlarged* and may weigh five to ten pounds (2,000 to 4,000 gm.). Its surface is smooth and its edge is fairly even, though somewhat blunt. Its pathological characters will be considered under diagnosis. Its dulness extends from the fourth rib in the nipple line to the umbilicus or even to the iliac crest; it visibly bows outward the right costal arch and possibly by its weight drags down the right shoulder. The gall-bladder is not enlarged for there is no obstruction of the large bile-ducts. The liver progressively increases in size, often paroxysmally with fever, pain over the liver and increase of the icterus. It never shrinks, except when combined with portal cirrhosis.

2. *Icterus develops early* and is *constant, permanent and characteristic.* It varies in degree, though it is usually quite intense. It is due to obstruction of the smaller bile vessels; it is not certain that it is due to over-secretion of bile (polycholia) or of bile pigments (polychromia). In extreme cases the skin becomes green or brown. It is frequently accompanied by itching, xanthelasma and eczema.

3. The *spleen is hypertrophied,* is usually increased relatively more than the liver, averaging one to two pounds (400 to 1,000 gm.) in weight, but sometimes actually outweighs it (2,600 gm.), especially in infantile forms. It is hard, smooth and evenly increased in all its elements and dimensions. It is often clearly visible and is distinctly palpable.

Other Symptoms.—*Digestive.*—The condition of the tongue and appetite is variable. Nausea, vomiting, dyspepsia and tympanites are uncommon. Diarrhœa is frequent. The normal or diarrhœal movements contain bile in over 90 per cent. of cases.

Nutritive.—Nutrition is often maintained for years, despite the icterus, but wasting subsequently appears. Development is retarded in young subjects and infantilism may be as marked as in cretinism or congenital syphilis. Clubbed fingers, from hyperplasia of the soft tissues, is not an uncommon development; Gilbert and Lereboullet collected 40 cases in 1901.

Urinary.—The urine is highly colored, bile-stained and is usually increased in amount; its freezing point is high and it may contain urobilin, indican or albumin.

Circulatory.—The pulse is not slow; the heart is often dilated and may reveal a systolic murmur; dyspnœa is frequent from pressure on the diaphragm by the swollen liver and spleen; the blood shows secondary anæmia, decreased coagulability and sometimes moderate leukocytosis. Arthritis and multiple neuritis are rare complications.

Diagnosis.—The following table shows the essential pathological and clinical points of contrast with the portal type:

PORTAL (ATROPHIC) CIRRHOSIS.——*vs.*——BILIARY (HYPERTROPHIC) CIRRHOSIS.	
1. *Etiology;* alcoholism; over forty years of age.	Infection; under forty years.
2. *Liver,* large or small (possibly both in succession); shrinks often.	Always large and to greater degree. Never shrinks in pure forms.
Granulations; larger.	Small; very fine.
Yellow.	Dark, icteric, green.
3. *Connective tissue* and inflammation ensnaring several lobules (multilobular or annular), and around portal vein radicles.	Begins in lobule (monolobular or insular) around smaller bile radicles (cholangitis), in early stages at least, later less clear.
Distinction between connective tissue and lobules sharp.	Not sharp.
Connective tissue, firmer, more fibrous; more elastic tissue.	More delicate, reticular, richer in cells, embryonal.
Scar-like and contracts.	Does not contract (elephantiasis hepatis).
4. *Liver-cells,* early fatty, degenerate, disappear.	Seldom fatty, remain normal for long time, may regenerate, more pigmented.
5. *Portal system* cannot be injected by portal vein.	Easily injected.
6. *Bile vessels.* (Proliferating bile vessels in biliary form are not characteristic, and are probably merely compressed rows of liver cells.)	
7. *Icterus* infrequent, a complication; when present, usually catarrhal.	Constant throughout course, though the stools contain bile.
8. *Ascites* rarely absent in advanced cases.	Absent in all (except mixed forms).
9. *Collateral* circulation—usual.	Never.
10. *Hæmatemesis,* frequent, early, mechanical.	Very rare, late, cachectic or toxæmic.
11. Constipation usual.	Diarrhœa quite frequent.
12. Duration of two to three years.	Five to ten years.
Death results from hæmatemesis, nephritis, tuberculosis, peritonitis.	Rare; death from hepatargia (so-called cholæmia) with fever, nervous symptoms, delirium, typhoid state, coma.

It is a rare affection and a diagnosis of biliary cirrhosis is too often made. If the cardinal features under its definition are borne in mind it can usually be separated with ease from the following affections: (*a*) In *hepatic cancer* the subject is older and more cachectic, the course is shorter, there is no splenic tumor, the primary growth, *e. g.,* in the stomach, may be recognized, the liver often presents irregularities, the neoplastic obstruction of the bile duct produces acholic stools and obstruction of the portal vein causes ascites. (*b*) Multilocular *echinococcus* (*q. v.*) also produces icterus and enlarged liver and spleen, but is exceedingly uncommon. (*c*) *Fatty and amyloid* liver are unattended by jaundice and in amyloidosis the

enlargement of liver and spleen is usually associated with albuminuria, diarrhœa, œdema and cachexia. (*d*) *Obstruction* of *the bile ducts* may produce an enlarged gall-bladder (*v.* Icterus); the stools are usually but not necessarily acholic (*e. g.,* in stone of the common duct). The spleen is not large. In this connection it may be stated that some recognize a (so-called) biliary cirrhosis due to chronic obstruction of the duct, as described by Charcot and Gombault in 1876; simple mechanical obstruction never causes fibrosis, though it may follow ascending cholangitis. The liver is far less large in this obstructive type and is seldom hard. (*e*) In hæmochromatosis and pigmentary cirrhosis icterus is absent and glycosuria is present in about 85 per cent. of cases. (*f*) For *Banti's disease* see *diagnosis of portal cirrhosis.* (See Differentiation, page 695.)

Treatment.—The treatment is that of portal cirrhosis; no means are known which stop the formation of connective tissue. A generous diet may be given. Cold and dampness are especially to be avoided. Calomel in small doses (grain $\frac{1}{60}$ to $\frac{1}{40}$ *t. i. d.*) may be given for weeks at a time and the salicylates and simple salines are sometimes beneficial. Operative relief by drainage of the gall-bladder is suggested, but in the reported cases the true biliary cirrhosis is often confused with obstructive icterus; operation might be helpful in cirrhosis due to ascending infections.

ABSCESS OF THE LIVER.

Suppurative hepatitis may be considered as a local affection, though it is sometimes a part of septicopyæmia (*q. v.*). Virchow found it in 1.5 per cent. of autopsies. Ninety-seven per cent. occur in males.

Varieties.—1. In the *septic type* (55 per cent. of cases, Bärensprung and Luda) the hepatic artery is the usual carrier of infection; the primary focus is usually in the external parts, especially the skull (Hippocrates). It may result from osteomyelitis, external inflammations, pulmonary abscess or gangrene, putrid bronchitis and, less often, from ulcerative endocarditis. Liver abscess occurs in 15 per cent. of cases of surgical septicopyæmia (Bärensprung). Infected thrombi in the peripheral veins may become detached, infarct the lungs and thence reach the arterial circulation. In rare cases infective material may fall back from the cava into the hepatic vein, known as retrograde embolism. The bacteria are those enumerated under sepsis—the pyogenic organisms, pneumococcus, colon and typhoid bacilli.

The *symptoms* of septic abscess are not distinctive because, first, they are obscured by the general toxæmia and, second, the multiple abscesses when scattered through the liver usually produce few decisive local symptoms. The liver enlargement is uniform and generally indistinguishable from the cloudy swelling of sepsis; fluctuation is rare; the portal circulation is rarely obstructed; pain and tenderness are usually present; jaundice is inconstant; the spleen is

swollen from sepsis; the course is rapid. The septic type may occasionally, and the traumatic often, consist of a single large abscess.

2. The *second variety* results from infection through the tributaries of the *portal vein* (31 per cent.) (Kelsch contends that 85 per cent. of 500 liver abscesses were due to dysentery). It has been known since Morgagni's time and is clinically the most important type. Any ulcerative or suppurative affection in parts containing these branches may be causal, for instance, amœbic dysentery (the most frequent cause), appendicitis (in 6 per cent. of its fatal cases), neglected hemorrhoids, gastric ulcer or cancer and disease of the bile vessels. Pyogenic organisms, the Amœba dysenteriæ, foreign bodies (as fish-bones, pins and parasites) may enter by the portal circulation. Pus organisms may enter and infect tuberculous, echinococcic or actinomycotic foci, of which latter 40 cases are recorded. The form of lesion varies; (*a*) there may be the *solitary* or tropical *liver abscess,* which will be considered separately, or (*b*) there may be suppurative pylephlebitis, which is less a suppuration of the liver substance than an extension of inflammation in the portal vein itself, following in a dendritic fashion through all of its intrahepatic ramifications; its branches are distended and present small accumulations of pus, which may appear as yellowish foci through the capsule, but show more clearly on section as portal vein suppuration; the whole liver may be involved or certain areas may be exempted by total thrombotic occlusion of the larger branches. In some cases the purulent thrombophlebitis may be traced back to the original lesion in the stomach or intestine; the liver is enlarged but the diagnosis is rarely more than a probable one.

3. The *third variety* is infection by the *bile vessels* (10 per cent.), in which an ascending cholangitis (*q. v.*), often calculous in origin, produces multiple small abscesses through the liver substance; this is rather similar in appearance to the pylephlebitic form. These three varieties account for 96 per cent. of cases of liver abscess.

4. The infrequent *fourth variety* is infection in infants through the *navel.* 5. The *fifth variety* is direct *involvement* of the liver *by contiguity,* which may occur in suppuration of the gall-bladder or ulcer of the stomach.

I. Solitary or Tropical Abscess.—The abscess is solitary in 75 per cent. of cases; in 11 per cent. there are two and in 14 per cent. more than two foci. Twenty to 30 per cent. of cases of amœbic dysentery, by far the most common cause, are complicated by liver abscess. Amœbic abscess may even occur without intestinal ulceration, though amœbæ are found in the stools; in 5 out of 27 of Futcher's cases there were no intestinal symptoms. Over 50 per cent. of tropical abscesses operated on in England by Manson contained amœbæ. Shiga's bacillary dysentery is rarely a cause. Single abscess may occasionally occur from trauma, pyæmia, typhoid or in hydatid cysts. It occurs chiefly in the tropics, though seen in the Southern states and sometimes in the North. In the tropics it is thirty-five times as frequent in Europeans as in the natives; alcoholism and a rich diet are par-

ticularly predisposing factors; 97 per cent. occur in males and 50 per cent. in those between twenty and thirty years of age.

Pathology.—Amœbæ reach the liver by the portal vein and cause necrosis by their toxins; this is followed by suppuration. The smaller, sometimes miliary, foci contain glairy, gelatinous, translucent fluid; the larger foci contain white, yellow, green, reddish (like anchovy sauce, Budd) or chocolate-colored fluid, which may be serous, bloody or viscid. Lafleur and Councilman consider that the absence of leukocytes in the wall and contents is pathognomonic of amœbic abscess. Red disks and necrotic liver cells are frequent. As Kartulis pointed out, the pus is usually sterile. The amœbæ are more abundant in the abscess wall than in its contents and may be found in the vessels outside of the abscess, necrosing the liver cells. In contrast to other abscesses, the walls of the amœbic type are soft, ragged and produce but little granulation tissue or fibrous encapsulation.

Symptoms.—Absence of all symptoms (latency) occurs in 13 per cent. of cases. Symptoms may be classified as (1) septic, (2) hepatic and (3) other symptoms and complications.

1. Septic Symptoms.—(*a*) *Fever* is the most important; it is remittent or intermittent, is less often continuous and in chronic cases there may be a normal or subnormal register. (*b*) *Rigors* and sweats, even independent of fever (Lafleur), are frequent. (*c*) *Leukocytosis* occurs in 50 per cent. only. In 43 uncomplicated dysentery cases, the leukocyte count was above the average count of the abscess cases in 23.4 per cent. (Futcher). (*d*) The *pulse* is small and frequent. (*e*) Progressive *emaciation* is usual, and the facies is a combination of hectic, depression, subicterus and cachexia, which is difficult to describe. (*f*) The urine is febrile and may contain albumin or albumose.

2. Hepatic Symptoms.—The *liver* is enlarged from the abscess and from sepsis. The *abscess* in 70 per cent. is located in the *right lobe;* Sérégè by injections has shown that venous blood from the pancreas and intestine is conveyed to the right lobe (whence the right lobe is chiefly involved in intestinal ulceration or cancer), while the blood from the stomach and spleen finds its way to the left lobe. The *enlargement* is more often *upward* than downward, wherein abscess differs from cancer and resembles echinococcus. The swelling is often localized, occasioning a *dome-like dulness on the upper border* of the liver (Frerichs); in other cases there is bulging of the lower interspaces or of the hypochondrium. In very large abscess the entire organ is greatly swollen, as in one reported abscess which contained 2⅓ gallons of fluid. When it occurs below the ribs, fluctuation in the liver and, less often, œdema of the abdominal wall may be noted. The liver consistence may be increased at first. (*b*) *Pain is usually* present at some time in the clinical course and is due to increased weight or to capsular tension and inflammation; perihepatitis or pleurisy may cause a friction rub from fresh inflammation,

or adhesions in more chronic cases. The patient usually lies on the back or right side to avoid the pain caused by sagging of the liver which occurs in the left lateral decubitus. Pain is present in the right shoulder in 17 per cent., being reflected from the phrenic filaments on the liver convexity along the phrenic nerve to the acromial branch of the fourth cervical nerve, with which the phrenic nerve is connected; in rare cases the deltoid may waste; the left shoulder is seldom the seat of referred pain. (*c*) *Tenderness* over the liver is usually circumscribed, but sometimes general. The right rectus muscle is frequently tense.

3. Other Symptoms; Complications.—(*a*) *Icterus* occurs in 16 per cent. of cases; it may be slight from sepsis or compression of some bile ducts within the liver, or it may be complete from catarrh of the common duct. (*b*) The *spleen* is *seldom enlarged,* except from a causal septicopyæmia. (*c*) A dry spasmodic *reflex cough* may be noted (*tussis hepatica*). (*d*) *Rupture* may be the first sign of abscess of the liver. In Waring's 300 cases, 56 per cent. remained intact, 16 per cent. were operated on and 28 per cent. ruptured. (i) It occurs *into the lungs* in 43 per cent. (average of the figures of Waring and Cyr); abscess of the lung results more often from direct rupture than from indirect metastasis by the hepatic vein. The patient presents signs of lung abscess (*q. v.*) and expectorates chocolate-, anchovy- or cream-colored pus containing amœbæ, often liver and lung tissue, hæmatoidin crystals and perhaps bile. Death may be caused by profuse hæmoptysis. (ii) Rupture into the *pleura* (15 per cent.) causes empyema. Pleurisy, both fibrinous and serous, may also occur without rupture, resulting from infection passing through the lymphatics of the diaphragm; a right-sided friction rub, serothorax or empyema should always suggest the possibility of an hepatic etiology. (iii) Rupture into the peritoneum (15 per cent.) causes localized or diffuse peritonitis. A subacute serofibrinous peritonitis rarely results, but the author has seen this form, as well as an encapsulated serous effusion, cover the convex and anterior surfaces of the liver. (iv) Rupture into the *bowels* (colon 2 per cent. and duodenum 1 per cent.) causes pus to be voided, sometimes in large amounts, by the stools. (v) Less frequent are ruptures into the stomach, pericardium, pelvis of the kidney, portal or hepatic vein, inferior cava, bile vessels or abdominal wall. (*e*) Less frequent complications are pyloric obstruction (from abscess of the left lobe), pseudorheumatism or clubbed fingers (from toxæmia), cerebral abscess and intestinal obstruction.

Diagnosis.—Diagnosis is based on (*a*) an *etiological factor;* (*b*) symptoms of *sepsis;* (*c*) *hepatic signs,* localized enlargement, pain and tenderness; the *x*-rays may be useful when there is bulging of the convexity covered by the lung; (*d*) exploration with the *aspirating needle,* which may be dangerous; the author has seen, as a result of puncture, two deaths from peritonitis and one from an enormous hemorrhage into the peritoneal cavity in a case of abscess with in-

tense icterus; puncture should be performed only when an operation can be done immediately after pus is found; pus is far less often found in multiple small abscesses than in the large solitary form.

Differentiation.—*Five types* of liver abscess are often described: (*a*) The latent, (*b*) typhoid (see table, page 42), (*c*) malarial, intermittent, (*d*) phthisic, and (*e*) a type declared by rupture, either pulmonary, peritoneal or pleural.

Subphrenic abscess simulates pneumothorax (*q. v.*) rather than liver abscess, because of the presence of tympany; in its etiology and treatment it resembles liver abscess (see Localized Peritonitis). In *gall-stones,* with or without suppuration, the fever usually occurs in paroxysms separated by fairly long intervals, jaundice (if present) is apt to become more intense with the fever, the history is different and marasmus is rare. Abscess of the abdominal wall shows no respiratory excursion in the exploratory needle introduced into it. Differentiation from *pleurisy, echinococcus* and *cancer* of the liver is considered under these topics and also in the differential table (page 695).

Prognosis and Treatment.—Ninety-three per cent. of unoperated cases die from unfavorable ruptures (peritoneal, pleural, pulmonary), sepsis, marasmus and amyloid disease, which may occur after weeks or months. It is possible for an abscess to desiccate or to heal after breaking into the lung or bowel, but this cannot be depended upon. The treatment is solely operative; 40 per cent. reach the operating table *in extremis;* Solonoff (1903), in a collection of 1,094 cases, estimates the mortality at 30 per cent.

TUMORS OF THE LIVER.

Tumors of the liver are chiefly *carcinomata. Benign tumors* possess little clinical interest, as fibroma, lipoma, teratoma, adenoma or angioma, which latter is more frequent in the liver than in any other organ. Single adenoma may develop from the liver cells or bile ducts; multiple adenomata occur chiefly in cirrhosis. Three cases are reported of adrenal "rests." *Sarcoma* is rarely primary, there being hardly twenty clear cases on record; it may develop from the connective tissue, endothelium of the lymph or bloodvessels or Kupfer's cells, as a single primary tumor, multiple primary tumors, diffuse infiltration or possibly as a primary melanotic growth, which, however, is almost always secondary to melanotic sarcoma of the eye or skin.

Cancer of the liver occurred in 2.7 per cent. of Virchow's autopsies, and ranks third among internal cancers; the usual order of frequency is cancer of the uterus, stomach, mamma and liver; Eichhorst's order is uterus, stomach, mamma, œsophagus and liver. (*a*) *Primary cancer* is 20 to 48 times as infrequent as secondary cancer; 66 per cent. occur in males. It originates from the liver cells (88 per cent.) or bile vessels (12 per cent.). The main forms are observed (i) as a sharply outlined massive local swelling (in 23 per cent. of Eggel's

163 cases reported in 1901); (ii) as sharply demarked multiple nodes of moderate size (in 65 per cent.) of which one of the tumors is primary and the rest secondary; (iii) as diffuse infiltration, fusing with the parenchyma (in 12 per cent.); (iv) there is a fourth form, which develops in hepatic cirrhosis. The writer has seen primary cancer develop from an hepatic cyst. Metastases occur in 66 per cent. (*b*) *Secondary cancer* results from primary cancer in the stomach (33 per cent.), in the colon (12 per cent), œsophagus, pancreas, gall-bladder, uterus, ovaries and mamma, by metastasis or extension by contiguity; cancer cells may enter by the portal vein and hepatic artery or much less frequently by retrograde metastasis through the hepatic vein; the lymph vessels do not carry cancer infection, as their current flows from within to without the liver. Their structure is that of the primary type.

Symptoms.—1. Hepatic Symptoms.—(*a*) The *liver enlarges rapidly;* it may increase to six or eight times its normal size and may weigh 30 to 40 pounds. Its *edge is uneven* though fairly sharp; on its *surface are lumps* which may present a central umbilication. *Great unevenness* is more characteristic of secondary than of primary tumor, in which the liver is more uniformly swollen. The *enlargement* is chiefly *downward,* though the liver is sometimes high because of ascites, tympanites or adhesions. Nodules in the falciform ligament may be felt near the navel or linea alba. Sudden increase in size occasionally results from hemorrhage due to icterus. There is no enlargement when the nodules are few and small, and the liver is smaller than normal in 10 per cent. of cases (liver cirrhosis plus malignant adenomata). (*b*) *Hepatic pain* is *more conspicuous than in any other liver lesion.* It may be caused by capsular tension, increased weight, perihepatitis, pleuritis or, less frequently, from rupture of tumor particles into the bile passages, which may simulate the pain of gall-stones. (*c*) The *liver is* often *tender.* (*d*) Exceptionally pulsation may be transmitted from the aorta or there may be a venous hum over the liver.

2. Cachectic Symptoms.—These are adynamia, marasmus, anorexia, secondary anæmia, leukocytosis (in any carcinoma, but especially in hepatic), insomnia, stupor and illusions. Cachexia probably results from the katabolic toxins elaborated by the tumor, and possibly also from hepatic insufficiency (*v.* Cirrhosis). Fever may result from the tumor alone, from cholangitis and from secondary or terminal infection. The rare peripheral neuritis and some of the urinary findings may be referred to cachexia. Rarely there may be no cachexia.

3. Compression Symptoms.—(*a*) *Jaundice* occurs in over 50 per cent. and more frequently in secondary than in primary cancer. It usually results from mechanical pressure on the larger bile ducts by the tumor or carcinomatous glands. Pressure on the ducts within the liver often produces moderate jaundice and some bile then appears in the stools; complete icterus may result from concomitant catarrhal

or calculous obstruction, especially significant when there is no ascites. Exceptionally the tumor grows into and along the bile ducts, thereby totally obstructing them. (*b*) *Ascites* is present in 50 per cent. of cases. It is usually moderate in degree and results from pressure on the portal vein or its larger branches, peritonitis or portal vein thrombosis (pylethrombosis); occasionally it is due to the cirrhotic form of cancer, growing of the tumor into the hepatic or portal vein, rupture—perhaps with syncope and great hemorrhage—of a cancer nodule into the peritoneum, or rarely to plugging of the liver capillaries by the pigment of a melanosarcoma (Hektoen and Herrick). A collateral circulation may develop. Friction from perihepatitis may be noted. The fluid is usually serous or hemorrhagic, less often adipose, chylous or opaque (*v.* CIRRHOSIS) and very rarely contains melanin (sarcoma). (*c*) *Other compression symptoms* include splenic tumor from marantic pylephlebitis or pressure on the splenic vein (14 per cent.), hydronephrosis, vomiting from pyloric obstruction, dyspnœa from pressure on the heart and lungs and œdema from pressure on the cava inferior.

4. OTHER SYMPTOMS.—The cervical glands are seldom swollen unless there is coincident gastric cancer. Carcinomatous nodes at the navel or in the skin over the liver are due to regional extension. In liver cancer combined with cirrhosis any cirrhotic symptom may occur. The urine shows increased destruction of the tissue nitrogen; the nitrogenous extractives are increased from the normal 0.6 or 0.8 per cent. to 13 or 23 per cent. Acetone and diacetic, lactic, beta-oxybutyric acids, indican, leucin and tyrosin are not infrequently present. The urine is decreased, high-colored and is often icteric. Albuminuria is due to stasis and toxæmia (Teissier speaks of hepatogenous albuminuria). Melanuria (sarcoma) is sometimes detected by adding to the urine a solution of ferric chloride; the pigment exists in the blood as melanin or melanogen, the latter of which is precipitated as the urine stands, or by the addition of some oxydizing agent as nitric acid; melanuria nearly always indicates involvement of the liver by a melanotic tumor and is often confounded with indicanuria and alkaptonuria.

Diagnosis.—The chief features are rapid enlargement of the liver, irregular tumors, pain, icterus, ascites, cachexia and in 50 per cent. of cases the recognition of the primary tumor. Other causes of liver enlargement must be considered as in cirrhosis. (See also DIFFERENTIAL TABLE, page 695.) Icterus with ascites is always suggestive. In some cases liver involvement is not suspected.

DIFFERENTIATION.—(*a*) *Cirrhosis* (see both forms). (*b*) *Syphilis* (*q. v.*) produces smaller, harder tumors, of less rapid growth, more often associated with splenic tumor and albuminuria, much less often complicated by ascites or icterus and responding to mercury and iodide. (*c*) The *amyloid liver* is equally large and causes confusion only when complicated by gummata; amyloidosis of the spleen, kidneys and intestines usually co-exists with amyloid liver. (*d*)

Hydatid cysts develop more slowly and *earlier in life* and cachexia appears late if at all. The rare multilocular echinococcus produces enlarged liver, ascites and icterus, but the spleen is also enlarged; puncture may give characteristic findings. (*e*) *Liver abscess* is seldom accompanied by icterus and ascites; puncture often excludes those doubtful cases of cancer in which there are fever, leukocytosis and semifluctuation of the necrotic neoplastic nodes. (*f*) In *congested liver* the causal heart or lung disease, the symmetrical liver swelling, other signs of stasis, diffuse tenderness and subicterus, together with cyanosis, are distinctive, though the writer knows of four cases of nutmeg liver in which stasis caused more local than general hepatic enlargement. (*g*) The *corset liver* can be easily differentiated if the general symptoms, such as cachexia and the compression symptoms of cancer, are duly considered. (*h*) *Cancer of the stomach* (*q. v.*) is readily differentiated by its peculiar chemism and signs of stagnation; gastric cancer may cause hepatic involvement. (*i*) *Renal or adrenal tumors* are covered by the inflated colon, encroach less upon the thorax, evert the ribs less, have (less or) no respiratory excursion and often are separable from the liver by an intervening strip of tympany, which occurs in but 13 per cent of hepatic tumors.

(*j*) *Differentiation between primary and secondary tumors* is difficult, yet somewhat irrelevant, as the causal growth leading to hepatic metastasis is latent in 50 per cent. Great caution should be exercised in diagnosticating primary liver tumor; the following characteristics are subject to many exceptions: the solitary tumor, its rapid growth, infrequent jaundice and ascites, less emaciation, more rapid course and death in less than four months. Secondary tumors cause death within seven months from cachexia, ascites and intercurrent disease, as pneumonia.

Treatment.—Treatment is entirely palliative for the pain and ascites except when, in very exceptional cases, early operation (for primary growths) can be performed. Lücke reported the first recovery, which afterward proved to be a gumma; his operation was followed by a few others, the cases of Nothnagel, Keen, Mayo Robson, Bardeleben, D'Urso, Israel, Hochenegg, von Bergmann and others, which included recovery from cancer, sarcoma and malignant adenoma.

ECHINOCOCCUS CYSTS OF THE LIVER.

The parasite will be considered under Animal Parasites and we will here consider its hepatic localization, which constitutes 60 per cent. of echinococcus disease; the adult parasite occurs in the intestine of the dog, wolf, fox and jackal; its cystic form occurs in man alone.

Symptoms.—*Fully half the cases* of liver echinococcus are clinically *latent.* Cases with symptoms show the following *liver findings:* (i) A *cyst on the lower border* or anterior surface may produce a visible, palpable, round and *smooth bulge* which everts the right costal arch

or much less often distends the left lobe. In some cases cysts in the right lobe lead to compensatory hypertrophy of the left lobe, which may equal the size of a normal liver. The tumor is as large or larger than an orange. It is tense, elastic, semi-solid and on percussion is dull; it may fluctuate and give the "hydatid thrill" of Briancon and Piorry, a vibration which is elicited by laying three fingers over the cyst and tapping with the middle one; the thrill is not a frequent finding and may also occur in ascites and sarcoma. The cysts rarely contain gas, due to the Bacillus coli. In large cysts (36 to 70 pints are reported to have been found), the liver may tip so that the right lobe is lower and the left higher than normal. (ii) *Cysts on the convexity* crowd the diaphragm upward, may cause symptoms resembling those of pleurisy (*q. v.*), produce a dome-like dulness, as in cancer and may compress the lung or even the heart. (ii) *Liver pain* and *tenderness are rare* without suppuration, which may induce perihepatitis; pain, as in cancer, may be referred to the right shoulder. Pleuritic pain follows rupture into the pleura. (iv) *Exploratory puncture* (Récamier and Cruveilhier) is dangerous, because severe nervous toxæmia and cardiac collapse may follow, as described under echinococcus of the lung and pleura, where also the finding of pus, the characteristic membrane, chemical contents and hooklets are enumerated.

Rupture may occur into the lungs (in 11 per cent. of liver echinococcus); it may simulate phthisis or gangrene of the lungs; cysts may rupture into the pleura, peritoneum, kidney, stomach, bowel, bile passages, pericardium, portal or hepatic vein and cava inferior; heart embolism or external rupture may occur.

Pressure symptoms are rare, as icterus (5 per cent.), ascites, albuminuria, *caput Medusæ* or œdema of the legs. Dyspnœa may result from pressure on the lungs or vomiting from pyloric stenosis. In some cases there is a distaste for fat or meat. Hemorrhage from the alimentary tract is rare. The blood is usually normal, though the eosinophiles are usually increased in nonsuppurating cysts. Urticaria may occur after puncture or rupture or without either incident.

Diagnosis.—Diagnosis depends on (*a*) the history of the case, including contact with dogs; (*b*) the smooth, round tumor; (*c*) the thrill; (*d*) slow development; (*e*) the infrequency of constitutional disturbance, pain and fever, the signs predominating over the symptoms.

Differentiation.—(*a*) From *other liver cysts;* the small cysts in cirrhosis and obstructive icterus are of no clinical importance. Simple serous cysts may, in exceptional cases, hold several (even 13) pints; they rarely cause symptoms, rupture or hemorrhage. Diffuse, congenital, cystic disease may cause dystocia; in adults also the cysts are probably congenital or developmental in origin and are associated with cystic kidneys, in 4 to 27 per cent. of cases (Lejars, Luzatta and Johnson). Moschcowitz (1906) collected 85 cases of non-parasitic cysts of the liver; they are usually multiple and sub-

capsular and are often associated with other congenital anomalies, such as hare-lip, spina bifida, hypospadias or supernumerary fingers. He concludes that (i) they are always associated with congenitally aberrant bile ducts; (ii) they are embryonal "rests"; (iii) the cysts are due to inflammatory hyperplasia of these ducts or to congenital obstruction in them. Hoffman reported 18 operated cases. (*b*) *Malignant disease* causes cachexia, more pain (than does echinococcus), multiple tumors, ascites and icterus. (*c*) *Syphilis of the liver* (*q. v.*) more often causes albuminuria or splenic tumor and responds to antisyphilitic remedies. (*d*) From *pleurisy;* the *x*-rays may show the localized tumor on the liver convexity; but the upper line of pleuritic dulness is much less often convex upward; there may be thoracic symptoms, as hæmoptysis, when there is rupture into the lung; careful examination of the sputum or aspirated fluid shows the diagnostic hooklets. (*e*) In rare cases a pendulous, pedunculated, movable cyst (v. Frerichs) may resemble a pancreatic cyst, enlarged kidney, gall-bladder or ovarian cyst. Rupture into the bile passages may simulate the pain of gall-stones. (*f*) Liver abscess is usually distinguished with ease, though one may remain in doubt in cases of suppurating cysts; the eosinophiles are not increased in liver abscess; both conditions are strictly surgical. (See table, page 695.)

Prognosis.—The prognosis depends largely on whether surgery is invoked. Cysts may, however, calcify or indurate spontaneously. According to Cyr, the mortality of rupture into the peritoneum is 90 per cent., into the pleura 80, bile ducts 70, bronchi 57, stomach 40, intestines 15 and externally 10 per cent.

Treatment.—When drainage by the trochar is performed (Hippocrates), it is said that 45 to 77 per cent. of cases recover. A few die suddenly from a toxalbumin, formed when the parasite dies; it causes urticaria, convulsions, cardiac failure and other symptoms similar to those produced by myrtilotoxin in mussel-poisoning. *Direct surgical intervention* is attended with the least risk. *Prophylaxis* is necessary, such as the burning of slaughter-house offal and avoiding close contact with dogs.

Multilocular or Alveolar Echinococcus.—This form is due to another parasite (see ANIMAL PARASITES); it is extremely infrequent and only about 100 cases are recorded.

Symptoms.—The right lobe of the liver alone is involved in 65 per cent. of cases and the left alone in 10 per cent. The liver is fibrosed, nodular and seldom fluctuates. This rare affection is attended by splenic tumor (90 per cent.), icterus (80 per cent.), dyspepsia, ascites and, less commonly, by fever, polyuria, collateral circulation, gastro-intestinal hemorrhage, late emaciation (cachexia) and metastases in the heart and lungs by way of the lymph or bloodvessels; death may occur after years from cholæmia or hemorrhages. The diagnosis is very difficult, though it is sometimes aided by exploratory puncture and the most frequent confusion is with cancer of the liver. The only hope of recovery is in extirpation, which has been successfully done by Terillon, Bruns and others.

FATTY LIVER.

The term "fatty liver" (*hepar adiposum*) includes fatty infiltration and fatty degeneration; as a distinction between them is of more speculative than clinical interest, they may be considered together.

Etiology.—(*a*) *Obesity* and (*b*) *decreased oxygenation* from carcinosis or any other cachexia, rickets, profound or pernicious anæmia, phthisis, cardiac stasis and kindred causes may produce fatty liver. (*c*) *Toxæmias,* as tuberculosis, the acute specific infections, infantile diarrhœa, acute yellow atrophy and suppuration, (*d*) *poisons,* as phosphorus, alcohol, arsenic or chloroform, and (*e*) *local diseases* of the liver, as cirrhosis or nutmeg liver, are etiological factors.

Pathology.—The fat, which is normally found in the liver up to 2 to 5 per cent. of its weight, may amount to over 40 per cent. The *liver* is *enlarged* and may weigh as much as 12 pounds or 4,500 gm. Its *surface is smooth,* its edges are rounded, its *consistence is decreased* and its color is yellow. On section it is anæmic, yellow and smooth, though the lobules may be slightly prominent. The color is yellow when oleic acid predominates and pale when there is more palmitin and stearin. Slight quantities may be detected only on microscopic examination. When cut with a warm knife the fat may be seen on the blade. Microscopically, fat is seen which, *in fatty infiltration,* is located principally in the periphery of the lobules and obscures the liver cells; when the fat is dissolved by ether, the normal liver cells are clearly seen. *In fatty degeneration,* fat forms at the expense of the liver cells, *i. e.,* the percentage of albumin is decreased. The statement that fatty infiltration occurs in large globules and fatty degeneration in smaller ones is not wholly correct. Pathologists are inclined to consider infiltration of more importance than degeneration.

Symptoms.—(*a*) *The positive findings* are as follows: the liver is *symmetrically enlarged* and *smooth* and rarely extends below the navel. Its *edge is rounded,* soft and can usually be palpated, even in obese persons. (*b*) The *negative findings* are absence of pain, tenderness, icterus, splenic tumor, ascites or gastro-intestinal hemorrhage.

The *diagnosis* is easily made and liver stasis, amyloid degeneration and cirrhosis can be readily excluded (see page 695). The treatment is that of the causal factors.

AMYLOID LIVER.

Amyloid degeneration was first clearly described by Rokitansky.

Etiology.—(*a*) *Ulcerative tuberculosis* of the lungs or bones causes over 50 per cent. of amyloidosis. (*b*) *Inveterate syphilis,* particularly ulcerative and osseous types, produces 25 per cent. of amyloid disease. (*c*) *Chronic suppuration* has become uncommon since the introduction of asepsis and antisepsis. Krawkow produced amyloid degeneration experimentally by inoculations of the staphylococcus. (*d*) As exceptional causes, intestinal ulcerations, exhausting diar-

rhœas, rickets, leukæmia, pseudoleukæmia and ulcerating neoplasms may be mentioned. Seventy-eight per cent. of cases occur in males (Frerichs).

Pathology.—Amyloid degeneration is much less frequent in this country than in Europe. Its *localization,* in order of frequency, is in the spleen, kidneys, liver and intestines. The liver is uniformly enlarged and may weigh 13 to 16 pounds (5 to 6,000 gm.), even in a child. It is hard, smooth and very pale. Its edges are more blunt than normal, though somewhat sharp. On section the general appearance is waxy, speckled, resembling raw ham, the edges are slightly translucent and the lobules are indistinct. If a thin slice is placed in diluted Lugol's solution, the amyloid areas assume a brown color (Virchow), the name (amyloid) referring to this starch-like reaction; the waxy substance, however, is a proteid and reaches the liver by the hepatic artery, whence it is first deposited in the middle zone of the lobule, and later infiltrates the central and peripheral zones. It is essentially an infiltration, affecting primarily the capillaries of the hepatic artery. Slight lesions may necessitate the microscope for detection. Amyloid liver may co-exist with gumma, cancer or cirrhosis.

Symptoms.—The large, tense, symmetrical, painless, smooth liver, with rounded edges, is characteristic. Its dimensions may attain those of cancer. The onset and development are usually slow, but acute amyloid has developed within a month. *Splenic* amyloid *enlargement* is usually also noted, as well as *albuminuria* from renal amyloidosis (*q. v.*). In only 0.8 per cent. of amyloid degeneration is the liver alone involved (Fehr). *Gastro-intestinal symptoms,* as nausea, vomiting and diarrhœa, may result from amyloidosis of the alimentary capillaries. In advanced cases *cachexia, œdema* and ascites may develop. Hepatargia is rare.

Diagnosis.—Diagnosis depends on (*a*) the recognition of a *cause;* (*b*) the characteristic, smooth, *symmetrical enlargement,* which is less hard than that of cirrhosis; (*c*) the associated *amyloid disease* of the *spleen, kidney* and alimentary tract; (*d*) the *absence of icterus* (which very rarely results from amyloid portal glands) and of *portal stasis* (ascites is an infrequent, late symptom and results from renal implication); and (*e*) the small amount of urobilin in the stools. *Differentiation* from cirrhosis (*q. v.*) and cancer (*q. v.*) presents no difficulties. Gummata in the liver plus amyloid disease may cause confusion. No diagnosis is possible in the exceptional cases in which the liver is slightly enlarged, because it is only slightly diseased, or in those cases in which splenic tumor and albuminuria are absent. Other diffuse and focal hepatic diseases are differentiated on page 695.

Prognosis.—The prognosis of pronounced cases is unfavorable, though light grades may exceptionally regress (Budd, Bamberger, Frerichs, Graves).

Treatment.—Treatment is unavailing; it is solely that of the causal condition and of the symptoms as they arise.

ANOMALIES OF FORM AND LOCATION OF THE LIVER.

In *situs viscerum inversus* the liver lies on the left side, which in pathological conditions may lead to serious diagnostic error. Fenger and Billings mention a case of icterus and gall-stones in situs inversus which was diagnosticated as acute yellow atrophy!

Tilting of the Liver.—Tilting of the liver (ante- and retroversion), *dislocation downward* by pleuritis, pneumothorax or tumors, or *upward luxation* require but passing mention.

The liver may be found in the thoracic cavity as a result of congenital malformation or severe trauma.

"Wandering Liver."—Wandering liver (Hepar migrans s. *mobile*) occurs in women (88 per cent.), usually after several pregnancies. Its *causes* are those of enteroptosis, with which it usually occurs, *viz.,* flaccid abdominal walls, possibly congenital laxness of the suspensory ligaments, tight lacing or very rarely from traction, as by a tumor. Clark and Dolley collected 118 cases; one case had a mesohepar attached to the diaphragm and absence of the coronary and right lateral ligaments.

Symptoms.—Symptoms may be entirely lacking. In 5 per cent. the onset is acute (Graham), though usually gradual. *Subjectively,* vague pain or oppression over the liver, gastro-intestinal disorders or nervous symptoms may be present. *Objectively,* the liver is lower and more mobile than normal; there is sometimes cardioptosis, usually gastroptosis and in 20 per cent. nephroptosis. Its notch can be distinctly felt, often also its convex surface and sometimes the tense, stretched ligaments. It can be replaced. The entire liver, in extreme instances, may be below the costal arch, and tympany, due to the bowel, may exist between it and the lung. In a recent case there was tympany between the left costal arch and the prolapsed spleen and tympany between the right arch and the liver, even though the patient was five months pregnant. In very rare instances there may be pain similar to that of gall-stones; ascites or splenic tumor results from torsion of the portal vein.

Treatment.—The *treatment* is (*a*) *mechanical,* which consists of replacing and supporting the liver with appropriate binders or pads; (*b*) *symptomatic,* with due consideration of the neurasthenic manifestations; (*c*) *dietetic,* in order to increase the body-weight and (*d*) *operative,* in which the liver is sewed to the diaphragm or abdominal wall. In 39 cases hepatopexy cured about 75 per cent.

Corset Liver.—The corset liver is found in 25 per cent. of female cadavers (Leube), but much less frequently here than abroad. Corsets cause pressure-atrophy of the liver substance, followed by induration, deformity and peritoneal thickening; these changes are most conspicuous and usual in the right lobe. The liver may be merely constricted, dislocated upward or the part below the constricting zone may hang loosely as upon a hinge; this may cause diagnostic confusion with floating kidney, enlarged gall-bladder or other tumors.

The gall-bladder is dilated in 60 per cent. of cases, though icterus is infrequent. Subjective manifestations are often absent, though there may be a sense of tension in the part. Sometimes paroxysmal syncope and vomiting may occur, due to congestion of the snared-off portion of the liver. Physical examination often determines the condition, especially when, by careful palpation, the hinged and hard part can be proven directly continuous with the liver's known margins. Respiratory excursion is naturally imparted to it in most cases. Confusion arises when the intervening gut causes a narrow zone of tympany between the hinged segment and the liver proper, but even then careful palpation frequently demonstrates its continuity with the liver. *Congenital tongue-like lobes* may be confused clinically with corset liver; they are often cirrhotic and relatively often the seat of malignancy, suppuration or syphilis. Corset liver is regarded as a disposing factor in gall-stone formation. Treatment is seldom possible nor is it important, though prophylaxis and perhaps resection should be mentioned.

AFFECTIONS OF THE BLOODVESSELS OF THE LIVER.

I. Active Hyperæmia of the Liver.—A degree of arterial hyperæmia in the portal circulation is physiological during digestion. An accentuation of this condition is common in heavy eaters and drinkers and acute infections, in which it may be combined with venous hyperæmia. Heat is probably not the cause of "tropical hyperæmia." The alleged vicarious hyperæmia from suppressed menstruation, the menopause or diseases of the internal female genitalia is based wholly on theory. The symptoms resemble those of passive hyperæmia (*v. i.*), *viz.,* some hepatic enlargement, moderate discomfort in the right hypochondrium and also, it is alleged, slight icterus. Treatment is seldom indicated, save in cases of dietetic excesses, where gastro-intestinal symptoms obtain. Then a simple, moderate or milk diet and calomel and saline purgation are indicated.

II. Passive Hyperæmia of the Liver.—**Etiology.**—Passive congestion of the liver results from various kinds of stasis, the stress in all of which is felt in the efferent hepatic (sublobular) veins within the liver. It results (*a*) from *cardiac insufficiency,* caused by valvular diseases, particularly mitral, myocardial maladies or pericardial lesions; (*b*) from *pulmonary* stasis, due to emphysema, chronic bronchitis, asthma, induration or obliterative pleurisy; (*c*) from stasis caused by *spinal deformity,* aneurysm, mediastinal tumor, *perihepatitis* or pressure on the cava inferior.

Pathology.—The essence of passive congestion is over-filling of the sublobular or the *hepatic venous circulation,* malnutrition of the liver cells (*fatty degeneration*) and in some advanced cases, *increased connective tissue.* The liver is usually *symmetrically enlarged, firmer* and a darker red than normal, though hepatic hyperæmia lessens after death. The capsule is often thickened or œdematous. On section

the so-called *nutmeg appearance* is noted, but seldom over the entire liver; it is not pathognomonic of passive congestion. The *central veins* with their radiating offsets are *dark,* cyanotic and sometimes sclerotic from stasis, while the periphery of the lobule is yellow-white from cloudy or fatty change (malnutrition or impaired oxygenation). Long-standing congestion results in atrophy and pigmentation of the cells in the periphery of the lobule (Virchow's *red atrophy*); there is usually increased connective tissue with fine granulations, the *atrophic cyanotic liver* or cardiac cirrhosis of the French; fibrosis is probably for the most part relative or apparent, for the liver parenchyma atrophies; true cirrhosis may co-exist with cardiac disease.

Symptoms and Diagnosis.—(*a*) The *causal heart* (or lung) lesion has its appropriate symptoms. (*b*) The *liver* is *evenly enlarged,* both on percussion and palpation, sometimes also on inspection; if ascites is present thrusting palpation with the finger-tips usually outlines the organ; the liver may reach to the navel or even lower and may evert the ribs. Its *edges are firmer* than normal, somewhat rounded, *sensitive* (from capsular tension) and *descend with inspiration;* the incisure is clearly felt. If there is relative tricuspid insufficiency, a systolic expansile pulsation is detected by palpating with both hands, which are separated from each other with each systole. This is readily differentiated from the non-expansile pulsation which is transmitted to the liver from the aorta. (*c*) The *hepatic intumescence varies* greatly, as the heart responds to rest or to digitalis, or fails in compensation. (*d*) The *skin* shows *cyanosis* and is also *tinged with slight icterus;* to the experienced eye it has a characteristic appearance, which is probably due to a static catarrh of the finer intrahepatic bile radicles. (*e*) For other symptoms, see Signs of Stasis under Valvular Disease. The spleen is seldom enlarged save from cardiac infarcts or complicating liver cirrhosis. Hæmatemesis is infrequent. Albuminuria and gastro-intestinal symptoms are referable to the heart. *Ascites* is usually *late* in onset and moderate in degree; the earlier statement, that an ascites, which is disproportionately marked in comparison with the general œdema, indicates the atrophic (indurated) nutmeg liver, is incorrect, because a cardiac ascites sometimes occurs without general œdema. Toward the end of the course there may be signs of renal and hepatic insufficiency.

Differentiation is seldom complicated, for the fundamental heart lesion, the evenly enlarged, tender liver, which varies in size from time to time, and the slight icterus with cyanosis are usually unmistakable. Certain features may sometimes mislead, as the occasional unequal distribution of the liver changes, which may suggest tumor; the tenderness in two cases which the author saw was thought to be suppurative. (See page 695.) The treatment is that of uncompensated valvular disease (*q. v.*).

III. Pylethrombosis—Pylephlebitis.—Thrombosis or phlebitis of the portal vein is practically always *secondary* to *compression or ob-*

struction, as liver cirrhosis, with which 33 per cent. of pylephlebitis cases occur; to cancer, ulceration, abscess or *inflammation* of contiguous structures, gall-stones, cholangitis and chronic proliferative peritonitis; and to *diseases of the intima,* as phlebosclerosis and syphilitic endophlebitis; all cases are promoted by slowing of the portal current. Parasites and embolism are rare causes. Pylephlebitis and thrombosis occur chiefly in males over forty years of age. A fatal obliterating endophlebitis exists with symptoms very closely resembling those of atrophic cirrhosis; Hess in 1905 collected 23 cases.

Symptoms.—These may be entirely absent because of collateral compensation through the hepatic artery or Sappey's accessory portal vein. The *onset* is usually sudden, with symptoms of *acute portal stasis.* The *spleen is enlarged* in 75 per cent., unless fibrous changes in its pulp or capsule prevent its swelling. *Ascites* develops in 66 per cent. and rapidly recurs after tapping. *Gastro-intestinal* symptoms are usual, as *hemorrhage* (44 per cent.), intense *sudden epigastric pain,* vomiting, diarrhœa or sometimes acute intestinal obstruction. The *portal vein* is sometimes *varicose* below and is generally *obliterated* at and above the point of obstruction (pylephlebitis adhæsiva); this leads to *shrinking of the liver,* particularly if the hepatic artery is sclerotic, in which rare event the entire liver may be hemorrhagically infarcted. The liver is said to become lobulated, but this probably results from antecedent syphilis. Symptoms of hepatargia (hepatic insufficiency) and alimentary glycosuria may develop. Œdema of the abdominal wall, development of a collateral circulation and icterus are uncommon.

Diagnosis.—Diagnosis is difficult, unless the onset is typically sudden, in which case pylethrombosis may be considered. Save for their acuity, the findings are those of atrophic cirrhosis; a caput Medusæ rather indicates occlusion of the portal radicles than of the portal trunk. The ascites may suggest carcinoma or peritonitis or the hemorrhage may simulate gastric ulcer or hepatic cirrhosis.

Course and Treatment.—Death may occur in a few days or after months or six to twenty years. Treatment is ineffectual, save in early syphilitic cases. Citric acid (3j) may be given every few hours to lessen thrombosis. Talma's operation may be considered.

Pylephlebitis suppurativa has been considered under liver abscess. It is a pyæmia of the portal vein; metastasis or extension occurs in the main portal trunk and its branches from infection of its radicles of origin; ulcerations and inflammations in the gastro-intestinal tract are its cause in 75 per cent. of cases; appendicitis causes about 40 per cent. (Dieulafoy's "appendicular liver") and gall-stones, infected piles and pancreatic or umbilical suppuration cause most of the other cases (*v. s.* Liver Abscess, Second Variety).

The *symptoms* are those of the *original disease,* those of *sepsis* and those of *portal stasis.* In very rare cases the thickened vein may be palpated. The spleen is enlarged, chiefly from sepsis, though sometimes from stasis. The liver is tender and somewhat enlarged, ic-

DIFFERENTIAL TABLE OF LIVER DISEASES

	SIZE.	EDGE.	SURFACE.	CONSISTENCE.	ICTERUS.	ASCITES.	SPLEEN.	PAIN.
ACUTE YELLOW ATROPHY.	Very small.	Even.	Smooth.	Soft.	Intense and invariable.	None.	Enlarged in 66 per cent.	Usual.
PORTAL CIRRHOSIS.	Somewhat decreased. (Sometimes normal or enlarged.)	Sharp (possibly finely nodular).	Smooth (possibly finely granular).	Hard.	Seldom (15 per cent.).	Usual in 50 to 85 per cent.	Usual (75 per cent.).	Inconstant.
BILIARY CIRRHOSIS.	Very large.	Sharp.	Smooth.	Hard.	Constant.	None.	Constantly; very large.	None.
ABSCESS.	Enlarged (mostly right lobe or on convexity).	Uneven, if abscess is on lower part.	Nodular.	Soft, even fluctuating.	Seldom (16 per cent).	None.	None (save from sepsis).	Common.
TUMORS.	Greatly enlarged.	Nodular.	Nodular.	Hard.	Frequent (50 per cent.).	Frequent (50 per cent.).	Rare (in 14 per cent.).	More than in any other liver lesion.
HYDATID.	Enlarged (right lobe).	Usually even.	Nodular.	Semi-fluctuating.	Seldom except in multilocular (80 per cent.).	None (save in multilocular).	None (save in multilocular 90 per cent.).	Seldom (except in multilocular).
FATTY LIVER.	Moderately enlarged.	Even.	Smooth.	Soft.	None.	None.	None.	None.
AMYLOID.	Greatly enlarged.	Sharp yet more rounded.	Smooth.	Hard.	None.	None (except late from cachexia or nephritis)	Usual.	None.
PASSIVE CONGESTION.	Moderately enlarged; may shrink late.	Rounded.	Smooth.	Firmer than normal.	Subicterus plus cyanosis.	Some from cardiac stasis.	None.	Tenderness usual.
SYPHILIS.	Enlarged (save in rarer atrophic type).	Nodular or lobulated.	Nodular.	Hard.	In 33 per cent.	Seldom (save from atrophic type).	Frequent.	Frequent.

terus occurs in over 50 per cent. and the usual signs of sepsis prevail. The diagnosis is always difficult, but is based on *symptoms* suggesting liver abscess (though *without the findings* of solitary abscess) on the one hand and those of sepsis on the other. The treatment is practically futile, being supportive and rarely, if ever, operative.

IV. Affections of the Hepatic Artery and Vein.—These are chiefly of pathological interest. *Infarction* of the hepatic artery with complete necrosis of the liver is very rare.

Aneurysm (*q. v.*) can seldom be diagnosticated. Pulsation is unknown; pain is nearly always present, icterus and a tumor occur in the majority of cases, ascites is uncommon and death usually results from gastro-intestinal hemorrhage.

Thrombosis of the hepatic veins is very infrequent, only 12 cases being reported. It may follow perihepatitis or, less often, compression by a neoplasm or fibrous obliteration of the inferior cava. Ascites and splenic tumor sometimes result. Suppurative thrombosis is always secondary to liver suppuration. Retrograde embolism (Heller) is mentioned under liver abscess.

V. Syphilis of the Liver (see SYPHILIS).

DISEASES OF THE GALL-BLADDER AND BILE VESSELS.

ICTERUS.

Icterus or jaundice is a symptom of various affections of the liver and bile vessels; it usually results from obstruction to the bile flow and is characterized by staining of the skin, mucosæ and urine by retained bile pigments.

Etiology.—Most cases of icterus are due to *obstruction* within or about the liver and biliary system; but slight obstruction is necessary because the bile is secreted under low pressure. (*a*) *Hepatic diseases.* It is (i) frequent in acute yellow atrophy, biliary cirrhosis, multilocular echinococcus, carcinoma, passive congestion and syphilis; (ii) infrequent in atrophic cirrhosis, unilocular echinococcus and abscess; (iii) absent in fatty and amyloid liver and in simple pylephlebitis. (See page 695.) Pick, Liebermeister and Minkowsky believe that occasionally functional liver disturbance may produce icterus (parapedesis of bile, "diffusion-icterus" or paracholia) which is analogous to transient albuminuria (in its relation to Bright's disease). (*b*) Diseases of the *biliary tract,* as (i) catarrhal, croupous or purulent inflammation; (ii) calculous obstruction; (iii) congenital and (iv) cicatricial occlusion; (v) malignant growths; (vi) parasitic obstruction by ascaris, distoma or hydatid vesicles. (*c*) It may result from

disease in *contiguous structures,* as (i) duodenal ulcer, cicatrix, catarrh or cancer; (ii) tumors of the colon or lymph glands; (iii) tumors or inflammation of the pancreas, stomach, kidney, omentum, mesentery and peritoneum, and (iv) rarely from visceroptosis or aneurysm. (*d*) *So-called hæmatogenous* or *urobilin icterus.* It was formerly maintained that toxic and toxæmic conditions, breaking down the blood cells (hæmolysis), cause icterus (cytohæmolytic icterus). It is now thought that most of these cases are essentially obstructive (thickening of the bile or cholangitis of the smallest radicles), though the obstruction of the small bile ducts within the liver is not complete. Eppinger maintains that the smaller bile radicles dilate and sometimes rupture, letting the bile into the lymph vessels which absorb it; mechanical obstruction lies in the capillaries blocked by fibrin or bile thrombi. The jaundice in these cases is slight, bile pigment (bilirubin) is not found in the urine and the stools are bile-stained. This type occurs (i) in various infections, relapsing fever, septicopyæmia, etc., (ii) in pernicious anæmia, and (iii) in toxic conditions, poisoning by phosphorus, chloroform, etc.

Icterus neonatorum may be considered here. (*a*) The *physiological* type occurs in 66 per cent. of children; it appears on the second day, lasts two weeks and is never fatal; its cause is not clear, though hæmolysis, increased blood pressure within the liver and persisting patency of the ductus venosus are suggested. (*b*) The much rarer *pathological* type may be caused by sepsis, syphilis, Winckel's disease, or still more exceptionally by congenital obstruction, gall-stones (10 cases collected by Skormin), acute yellow atrophy (7 cases) or catarrhal icterus (but 3 cases recorded). Glaister reported a family in which 6 cases of jaundice developed after birth. Krannhals recently published 26 cases of congenital "family icterus," most of which had an enlarged spleen.

Symptoms.—Bilirubin, found normally in the bile, enters the blood in biliary obstruction, by the lymph vessels and possibly also by the bloodvessels and produces the following symptoms:

1. Skin and Mucosa.—Staining of the skin and mucosæ occurs in three days. The scleræ, skin and mucosæ are yellow and in chronic cases dark brown. The skin itches because of its dryness or the action of the bile on the nerves. Urticaria or xanthoma may occur.

2. Urine.—The urine contains bilirubin before icterus is seen in the skin. Adding nitroso-nitric acid to the urine in a porcelain dish or on filter paper gives the Gmelin play of colors, of green, yellow, violet and red. If the urine is agitated the yellow color appears in its foam. Confusion is possible with melanuria, hæmaturia, urobilinuria or staining of the urine with rhubarb, picric acid, santonin, senna, etc. The subject of *urobilinuria* and urobilin-icterus has caused much discussion; urobilin is formed in the intestine by the reduction of bilirubin, whence it is absorbed and excreted by the urine; it never stains the skin and icterus results from bilirubin alone. There are three classes of cases; (*a*) in complete biliary

obstruction with icterus only bilirubin is found in the urine, for none of it reaches the intestine to be converted into urobilin; (*b*) in partial obstruction bilirubin is found in the blood and urine and also some urobilin, which is produced in the intestine from the bilirubin by intestinal fermentation; (*c*) in slight obstruction there is no bilirubin but much urobilin in the urine. *Albuminuria* may occur or *cylindruria* alone and the hyaline casts and desquamated epithelium are then bile-stained. The *sulphates,* normally 14 to 25 per cent. of the sulphur output, may be increased to over 40 per cent. *Indicanuria* is often marked and is due, like the sulphate increase, to greater intestinal putrefaction.

3. The Digestive Tract.—The tongue is coated, the breath has a disagreeable odor and the mouth may have a bitter taste, due to toxæmia. There may be an especial aversion to fats and sometimes to proteids and there may be nausea. The *abdomen is distended,* which is thought to be due to the absence of the antiseptic action of the bile, though the importance of this action has been overestimated. Starchy food is not wholly absorbed, albumin digestion is somewhat impaired and fat is found in large amounts in the stools (*steatorrhœa*). The function of the pancreas in this respect is far more important than that of the bile. Vegetable are absorbed better than animal fats. The normal amount of fat found in the fæces is 7 to 10 per cent., and this is increased in icterus to 50 or 80 per cent. The *stools* are *clay-colored, constipated* and *stinking;* they may shimmer when the fatty soap crystals are abundant; they are also *dry* (the bile in the fæces normally amounts to nearly a quart a day). If the obstruction is incomplete—as in many intrahepatic diseases—the stools are merely somewhat lighter than normal; a nitrogenous diet may impart a brownish color to the fæces. The fæces may appear deceptively dark if voided with the urine which lends a yellow color to their surface.

4. Cholæmia.—Cholæmia (hepatargia or hepatic insufficiency) may finally develop. It probably results from injury to the liver cells which fail to destroy the usual intestinal toxins and those formed in excess by the increased putrefaction. Its symptoms resemble acute yellow atrophy, *i. e.,* nervous excitation followed by stupor, irregular breathing or cutaneous and other hemorrhages. Most subjects of icterus are depressed, irritable, sleepless and somewhat emaciated. The hemorrhages are thought by Mayo Robson to result from coincident pancreatic involvement (diminished lime salts or the glycerine formed by fat necrosis).

5. Other Symptoms.—(*a*) The *heart* rate is *slow,* particularly in catarrhal forms or recent cases and in the absence of pain, fever and intoxication; it may fall to 21 (Frerichs); it has been explained by degeneration of the heart muscle or action of the bile-salts on the intracardiac ganglia, but it is probably caused by vagus stimulation, for atropine will raise the rate (Weintraud). The arterial pressure is low and the capillaries wide. A functional heart murmur may

develop; this is usually caused by myocardial weakness; Potain holds that the lung capillaries contract reflexly, thereby increasing the work of the right heart and causing its dilatation. The *respiration* rate is *decreased,* even to 7 a minute; the temperature is usually normal. (*b*) The *blood* shows the bile stain and in severe cases delayed coagulation, anæmia and leukocytosis. The blood may give a partial Widal reaction (typhoid cholecystitis?). (*c*) The *liver cells* may suffer mechanically and chemically from bile stasis and may be smaller, altered in form, pigmented, necrotic and bile-stained. The gross and minute anatomy varies with the cause. The bile vessels are often dilated, sometimes thickened and occasionally ruptured. The condition of the gall-bladder will be discussed under gall-stones and pancreatic cancer. As stated under biliary cirrhosis, it is questionable whether simple biliary stasis ever causes induration of the liver. (*d*) The spleen is occasionally enlarged, especially in children, from infection or portal stasis, but splenic tumor is no part of bile stasis. (*e*) *Nearly all tissues, secretions and excretions are bile-stained.* The muscles and nervous tissues (except in the severe type of icterus neonatorum) are seldom icteric. (*f*) Xanthopsia, yellow coloring of objects seen, is fairly common and is due to the action of the bile pigment on the nervous tissues. The explanation of nyctalopia and hemeralopia is uncertain. (*g*) Of xanthelasma multiplex 23 cases were collected by the London Pathological Society in 1882 Futcher in 1905 reported three cases; 80 per cent. of reported cases occur in chronic icterus (in women more frequently than in men), due to gall-stones, stricture of the bile-ducts, cirrhosis, hydatids and cancer. They are often symmetrical, often begin on the eyelids or the folds and creases of the skin, in one case were found in the bile ducts, rarely disappear and are probably due to some toxic substance. Few cases are probably hereditary.

Diagnosis.—A diagnosis is easily made. Icterus is not noticed in artificial light and daylight is absolutely essential for its certain detection. It is seldom confused with anæmia, cachexia, Addison's disease or the pigmentation of vagabondism. Pingueculæ on superficial examination are often mistaken for scleral staining.

Prognosis and Treatment.—The prognosis and treatment cannot be considered, as icterus is but a symptom. The details depend on the basic affection. (See Catarrhal Jaundice for treatment in general.) The patient may recover after four years (Budd); cases of icterus lasting six years (Murchison, Barth, Bosnier), ten to twenty-five years (Legendre, Galliard) or even fifty-two years (Masing) have been reported.

CHOLELITHIASIS.

Gall-stones are found in 8 per cent. of autopsies. Among the many literary contributions to this subject are those of Naunyn, Kehr, Riedel, Lange, Robson, Keen, Fenger, Murphy and Mayo.

Etiology.—1. Immediate Cause.—The causes are (i) catarrhal

inflammation of the gall-bladder or bile vessels and (ii) stagnation of bile. Bacteria play an important role, especially the typhoid (found in 33 per cent.) and colon bacilli; they have been found in calculi, and cholelithiasis has been experimentally produced by their inoculation; the Bacillus typhosus may live seven years in the gall-bladder.

2. Predisposing Factors.—(*a*) *Age;* 75 per cent. of cases occur in persons *over forty years* of age and only 1 per cent. in those under twenty years. (*b*) *Sex;* 70 to 80 per cent. occur *in women,* in whom pregnancy, menopause, lax abdominal walls, enteroptosis, constipation, sedentary life and lacing are regarded as promoting factors; Bollinger and Bother found that 40 per cent. of women who have gall-stones also have the corset liver. According to Naunyn, gall-stones are found in 25 per cent. of autopsies on women over sixty years; 90 per cent. of women affected have borne children. (*c*) *Other possible but only accessory factors* are *sedentary habits, obesity,* luxurious living, insanity, cancer of the stomach and foreign bodies in the biliary tract; Kehr holds that stone-formation is induced by long intervals between meals. Cholelithiasis is more common in cool than in warm climates.

Properties of Gall-stones.—(*a*) *Site;* they are almost invariably formed in the gall-bladder; very rarely do calculi develop in the intrahepatic ducts as in McArthur's recent case. (*b*) *Size;* they range from the size of sand or gravel to that of an egg; a calculus 12 inches in length is recorded; the heaviest weight reported is over four ounces (135 gm. Ritter). (*c*) *Form;* they are usually round and rarely spinous; when multiple they are polygonal and facetted. Intrahepatic calculi may be cylindrical or branching like coral (Cruveilhier). (*d*) Their *number* averages a dozen; according to Riedel 44 per cent. are single; Otto reports 7,802 stones in one case. Chopart instances a case in which the liver could scarcely be cut because of the numberless stones in the intrahepatic ducts. (*e*) *Consistence;* gall-stones can usually be indented by the finger. (*f*) The *time required* for their growth is about six months. (*g*) *Chemistry;* the most common form consists of *cholesterin* and *bile-pigment* (bilirubin and biliverdin) with some lime as a cementing substance; these calculi are small, numerous, usually yellow, laminated and contain about 75 per cent. of cholesterin. *Cholesterin* stones are not common; they are translucent, become opaque when dry, are light in weight and color and are combustible; they present a crystalline fracture like that of camphor, they dissolve in ether and alcohol, from which the crystals are precipitated and produce a red tinge on addition of chloroform and sulphuric acid; cholesterin comes from the bile or the inflamed mucosa of the gall-bladder. *Pigmentary stones* are uncommon; bilirubin imparts a brownish and biliverdin a greenish tint to them. *Calcium carbonate* stones are rare, are grayish in color and heavy. Stones in the liver are constituted of calcium and bilirubin. Traces of magnesium, bile acids, fatty acids, iron and copper have been found. The nucleus may consist of mucin, bacteria, epithelium,

blood-clot, cholesterin, calcium, bile pigment or rarely of foreign bodies.

Symptoms.—Kehr, Riedel and Paulsen assert that 95 per cent. of cases of gall-stones have no symptoms, especially in elderly subjects with atrophic gall-bladders. Stones may be felt or may crepitate in the gall-bladder, or be found in the fæces in old people with lax tissues, without any subjective symptoms. The writer believes that latency is less common than above figured and that *gastric symptoms are exceedingly common.*

Biliary Colic.—An *attack* embraces several important elements. (*a*) Colic is usually *sudden* in onset and very frequently begins at night. The pain is great and many women state that it is more severe than labor pains. It begins in the right upper abdominal quadrant and may radiate to the back, navel, hypogastrium, left side and even to the arms, legs, testes or chest. Irregular or large stones cause the greatest agony, controlled only by large or repeated hypodermics of morphine. The pain usually endures for a few hours and may soon recur. The cause of biliary colic is *mechanical,*—the attempt of the stone to migrate produces the spasm of the cystic and common ducts during its passage—or *inflammatory,* Kehr maintaining that cholecystitis starts the stones moving by the exudation it induces. The pain is less or shorter if the ducts are patulous. The author has seen violent colic result from this mechanism, as well as from cholecystitis without stones. It is thought that quiescent gall-stones may be started onward by palpation of the gall-bladder, trauma, muscular exertion, pressure by corsets, particularly after reduction of flesh, menstruation or delivery. (*b*) *Reflex nausea* and *vomiting* are frequent; Kehr thinks that they indicate cholecystitis (Mierzkowski found the bile sterile in only 22 per cent.). Vomiting relieves the pain and this frequently causes an erroneous diagnosis of gastralgia. Torsion of the cystic duct may cause vomiting but not colic. (*c*) *Chills and fever* occur in 66 per cent. and indicate bacterial inflammation. (*d*) The *gall-bladder* is *always tender,* there being hyperæsthesia over the ninth costal cartilage or over a point two-thirds of the distance between it and the navel. In 33 per cent. the gall-bladder is palpable. The right rectus muscle is rigid from peritoneal involvement. Boas and Gruh describe a right-sided hyperæsthetic zone which lies between the spine and posterior axillary line and at the level of the lower dorsal or upper lumbar vertebra. (*e*) *Icterus,* so often emphasized as a diagnostic point, is present in but 15 per cent. (Riedel and Kehr). It may be calculous, infectious or compressive. (*f*) *Other symptoms* of the paroxysm are rapid pulse, sweating, prostration, concentrated urine, constipation, abdominal retraction and, after the seizure, marked prostration. In some cases hysterical manifestations may be precipitated. Attacks of migraine are not infrequent (Kehr). There may be reflex coughing or reflex contraction of the pulmonary capillaries, which imposes extra work on the right heart and causes its dilatation. The patient may suffer actual col-

lapse and in nine cases death has occurred in the attack (Naunyn). Glycosuria, albuminuria, leukocytosis and splenic swelling are inflammatory in origin. The stone may fall back into the gall-bladder or pass into the bowel if it measures under 1 cm. It is detected by washing of the stools through a cloth; confusion with lumps of olive oil, given to relieve the colic, is unnecessary. The passage of a round calculus suggests, but does not prove, that it is the only one. Stones passing into the bowel may be dissolved, unless coated with cholesterin; this may explain why stones are not more often found in the fæces. The *great importance of gall-stones lies in their complications.*

Complications.—Complications may be grouped as *mechanical* or *inflammatory,* but as this involves descriptive repetition, both forms are combined topographically. Such complications as liver abscess or pyæmia may develop from "latent" stones.

1. Gall-bladder.—Mechanical complications include pressure on the duodenum or pylorus and rupture of the gall-bladder, which, however, are generally inflammatory; pressure on the common duct; hyperchlorhydria and hypersecretion; and cancer of the gall-bladder (*q. v.*). Inflammatory complications are more serious; all forms and sequences of cholecystitis (*q. v.*) may develop, as hydrops, empyema, gangrene, ulceration, deformity, hemorrhage, atrophy, calcification or liver abscess; also pylephlebitis, adhesions producing pyloric obstruction with hyperchlorhydria, dyspepsia and sometimes hemorrhage into the stomach; adhesions causing duodenal obstruction; and peritonitis, either local (near the gall-bladder or subphrenic abscess) or diffuse; Courvoisier reported 49 cases of the localized and 70 of the diffuse type. A friction rub may be heard over the gall-bladder a day or two after the colic. The distended gall-bladder rarely exceeds the size of the fist, is pyriform, can be moved laterally and moves with the liver, from which it may seem separated by a groove; it may be sometimes confused with appendicitis, the kidney, ovarian cyst or ascites and has even been found in a femoral hernia.

2. Cystic Duct.—Mechanical impaction causes icterus by spasm or catarrh of the common duct (in 12 per cent., Riedel) or by directly compressing it. The duct may become twisted, stenosed or obliterated.

3. Common Bile Duct.—Its mechanical occlusion is usually but not invariably preceded by colic; impaction occurs oftenest at its lower end (50 per cent. Courvoisier or 67 per cent. Robson), and is caused most frequently by one stone; 88 have been found (Mayo Robson). Permanent occlusion by stone in the common duct or in the cystic duct pressing upon it causes marked icterus, usually without sepsis. The occlusion may be partial or intermittent, by a ball-valve action of the stone, described by Osler and Fenger, which allows some bile to enter the gut and, unfortunately, some bacteria to enter the duct, thus adding bacterial to mechanical injury. In isolated cases there is no icterus but usually the icterus is sudden in

onset and slow in subsidence. In partial or intermittent obstruction there is often the *intermittent hepatic fever,* first described by Charcot and later clearly defined by Osler. Budd well compared it to urethral fever. During the paroxysms the fever rises to 103 or 105°, often with chills, sweats, leukocytosis, vomiting and increased tenderness, pain and icterus. The spleen may swell, and also the liver from cylindrical or saccular dilatation of the channels and cholangitis. Osler correctly insisted that these symptoms occur without suppurative cholangitis, though with infection. The gall-bladder is not enlarged, but is usually shrunken, following *Courvoisier's law;* Courvoisier (1890) found the gall-bladder enlarged in 92 per cent. of non-calculous obstruction of the common duct and shrunken in 80 per cent. of calculous obstruction; this is caused by infection (Hanot); Eichlin, in non-calculous obstruction, found the gall-bladder dilated in 87 per cent. and contracted or normal in 6.5 per cent. each; in calculous obstruction the gall-bladder was contracted in 66 per cent., normal in 19 per cent. and dilated in 15 per cent. In some cases suppurative cholangitis may complicate stones of the common duct, with remittent fever (rather than the above described intermittent fever), enlarged liver, septic symptoms and more intense and more rapidly fatal course. Mechanical dilatation may distend all the bile-vessels which may be seen under the capsule; ascending cholangitis may cause subcapsular abscess. Cicatricial stenosis of the common duct is rare; pylephlebitis may develop. Malaria is excluded by examination of the blood; malignancy will be considered under tumors of the pancreas, though in obstruction of the common duct the nutrition is generally little impaired. Practically, special stress may be placed on (*a*) the periodic fever, (*b*) the icterus sudden in onset and subsiding slowly, and (*c*) tenderness (without pain over the liver).

4. Ampulla of Vater.—Calculous retention in the ampulla may obstruct the flow of the pancreatic juice, though the accessory duct may carry it into the intestine; retention may allow bile to enter the pancreatic duct, and result (*a*) in acute pancreatitis, caused by bile in the pancreatic ducts (Opie); (*b*) chronic induration of the head of the pancreas resembling malignancy (Riedel, Robson, Barling); (*c*) pancreatic fibrosis with pancreatic calculi due to stasis and infection; or (*d*) rarely glycosuria.

5. Intestinal Obstruction (*q. v.*).—Two hundred and fifty cases have been collected. The stone usually ulcerates its way into the bowel from the biliary channels or gall-bladder. The obstruction is often incomplete and may not occur for some time, sometimes receiving new layers of magnesia or phosphates. According to Courvoisier, the obstruction is in the ileum in 66 per cent., duodenum in 21 per cent., ileocæcal valve in 10 per cent. and sigmoid in 3 per cent.; 50 per cent. of cases die.

6. Fistulæ.—Courvoisier assembled 499 cases. The external form constitutes about 50 per cent. of cases; they are most often lo-

BREWER'S DIAGNOSIS OF DISEASES

PATHOLOGICAL CONDITION.	PAIN.	FEVER.	VOMITING.	JAUNDICE.	TUMOR OF GALL-BLADDER.
I. CALCULOUS DISEASE.					
(*a*) Stone in healthy gall-bladder, ducts free.	No.	No.	No.	No.	May be present from large number or size of stones.
(*b*) Stone in healthy gall-bladder, cystic duct temporarily obstructed.	May be absent; generally present during obstruction; paroxysmal.	No.	May be present when colic occurs.	No.	May be present from distention.
(*c*) Stone impacted in cystic duct.	No.	No.	No.	No.	Present; may attain large size.
(*d*) Stone in hepatic duct.	Frequently present; irregular type.	Occasionally present.	May be present during pain.	Frequent.	No.
(*e*) Stone in common duct; acute obstruction.	Present; acute paroxysmal radiating to back.	Generally present.	Present	Present.	No.
(*f*) Stone in common duct; movable; chronic.	Periodic attacks of acute radiating pain.	Present with chills and sweats.	Present.	Present; intermittent.	Rarely present.
(*g*) Stone in common duct; impacted; chronic.	May be absent; frequently present early; may be intermittent; variable.	May be present; variable.	Often present.	Present; progressive; may vary in intensity.	Rarely present.
II. INFLAMMATORY DISEASE.					
(*a*) Cholecystitis subacute.	Present; paroxysmal during periods of cystic duct closure from stone or swollen mucous membrane.	Present during attacks of colic.	May be present.	No.	Present during attacks of cystic duct obstruction.
(*b*) Cholecystitis acute.	Acute paroxysmal radiating pain; extending to back and shoulder; may be very severe.	Present with chills and sweats.	Present; often severe.	No.	Present; tenderness; often muscular rigidity.
(*c*) Cholecystitis chronic (empyema of gall-bladder).	Severe radiating pain at first; may disappear later; tendency to recur.	Present; severe at first, may diminish later.	Present at first.	No.	Present; with tenderness; may attain large size.
(*d*) Cholecystitis in previously diseased and contracted gall-bladder.	Present; often severe; paroxysmal.	Present; often with chills and sweats.	Present.	No.	No (occasionally present, due to pericystic exudate).
(*e*) Cholangitis of hepatic and common ducts.	May be absent; generally present when obstruction exists, or severe infection; tenderness and pain over liver in intrahepatic cholangitis.	Present; chills; sweats; severe prostration; general sepsis.	Present.	Present; variable.	No.
III. NEW GROWTHS.					
(*a*) Carcinoma of gall-bladder.	No; may occur late in disease.	No.	No.	Present late (portal glands).	Hard, irregular, movable tumor at first, later diffuse infiltration.
(*b*) Tumor of cystic duct.	No; may occur late.	No.	No.	No.	Present when obstruction exists.
(*c*) Tumor of hepatic or common duct.	No; may occur late.	No.	No.	Present; progressive.	May be present from distention with bile.
(*d*) Tumor of neighboring viscera producing chronic obstruction of common duct.	No; may occur late.	No.	No.	Present; progressive; may become extreme.	Present; generally from distention with bile.

OF THE GALL-BLADDER AND DUCTS.

URINE.	STOOLS.	LIVER.	SPLEEN.	ASCITES.	REMARKS.
Negative.	Normal.	Not enlarged.	Not enlarged.	No.	Generally discovered by accident; often unrecognized.
Negative.	Normal.	Not enlarged.	Not enlarged.	No.	All symptoms promptly relieved as soon as obstruction removed.
Negative.	Normal.	Not enlarged.	Not enlarged.	No.	Hydrops of gall-bladder often unrecognized.
Contains bile pigment at times.	May be clay colored if obstruction occurs.	Frequently enlarged.	Not enlarged.	No.	Diagnosis extremely difficult; symptoms generally due to coexisting cholangitis.
Contains bile pigment.	Clay colored.	Not enlarged.	Not enlarged.	No.	Symptoms rapidly disappear when stone passes papilla.
Contains bile pigment.	Clay colored.	May be enlarged during attack.	May be enlarged from pressure of stone on vein.	No.	"Fievre intermittente hépatique" of Charcot resembles malaria; all symptoms disappear during interval.
Contains bile pigment.	Clay colored.	Enlarged.	May be enlarged.	No.	Condition may remain for many years; may only be jaundice with digestive disturbances and loss of weight; history of previous attacks (?) Ascites may be present from pressure of large stone or possibly from hydræmia.
Negative.	Normal.	Not enlarged.	Not enlarged.	No.	Tenderness over gall-bladder; tendency to recurrence; generally associated with stones in gall-bladder.
May contain albumin and casts.	Normal.	Not enlarged.	May be enlarged (sepsis).	No.	May follow typhoid or other septic diseases; onset often sudden; rapid development of severe symptoms resembling appendicitis; may be necrosis of walls of gall-bladder with perforation; local or general peritonitis.
Negative.	Normal.	Not enlarged.	Not enlarged.	No.	Frequently follows acute cholecystitis; occasionally becomes quiescent, presenting practically no symptoms.
Negative.	Normal.	Not enlarged.	May be enlarged (sepsis).	No.	Generally tenderness over gall-bladder area, but no tumor; local peritonitis; diagnosis often extremely difficult.
May contain bile pigment, albumin and casts.	May be clay colored.	Enlarged.	Enlarged (sepsis).	No.	Often follows severe infections of gall-bladder; generally associated with stones in common or hepatic ducts; severe sepsis; generally fatal in virulent infections (streptococcus).
Negative (at first).	May be clay colored late.	Enlarged late in disease.	May be enlarged late (pressure on vein)	Present late.	Digestive disturbances, progressive loss of weight and asthenia; cachexia; rapidly fatal.
Negative.	Normal.	Not enlarged.	Not enlarged.	No. (?)	Very rare; both benign and malignant growths have been reported; diagnosis difficult.
Contains bile pigment.	Clay colored.	May be enlarged.	Not enlarged.	May be present late.	Very rare; diagnosis difficult.
Contains bile pigment.	Clay colored.	Enlarged.	May be enlarged late.	Present late.	Malignant tumors most common; chronic interstitial pancreatitis from previous infection of biliary passages may remain after cause has disappeared; enlarged portal glands; Hodgkin's disease.

cated near the navel and about 40 per cent. of them heal. In the duodenal form (28 per cent.) the fistula is between the duodenum and gall-bladder and less often between the common duct and the gall-bladder. In 13 per cent. there is a fistulous opening into the colon. Courvoisier and Graham collected 34 cases of bronchial fistula. In rare cases stones enter the stomach (five cases recorded, Snively, 1903), portal vein, hepatic artery, kidney, urinary bladder (200 gall-stones evacuated in the urine in the case of Barraud-Palletan), pericardium, retroperitoneal tissue, vagina or even the pregnant uterus.

7. Renal Calculus, Glycosuria.—In 700 cases of gall-stones Kraus found complicating renal calculus in 6 per cent., glycosuria in 7 per cent., diabetes in 4 per cent. and gout in 2 per cent.

Diagnosis.—The diagnosis is easily made when colic, vomiting, tenderness of the gall-bladder and icterus are present; icterus is absent in at least 80 per cent. of all cases and the *x*-rays are uncertain.

1. The Colic.—This is most apt to be confused with (*a*) *gastralgia,* for both conditions are relieved by vomiting; due regard for other symptoms of gall-stones and the habit of doubting every diagnosis of gastralgia will save many mistakes. (*b*) A diagnosis of *hepatic neuralgia* should always be made with reserve, though pain near the liver does occur in neurotics. (*c*) *Gastric or duodenal ulcer* (*q. v.*) and (*d*) hyperchlorhydria and hypersecretion (*q. v.*) are easily distinguished. (*e*) *Lead colic* has the characteristic gingival lead line, obstinate constipation and often neuritic manifestations. (*f*) *Tabetic crisis* is attended by the Argyll-Robertson pupil, absent knee-jerk and ataxia. (*g*) Renal colic (*q. v.*) is distinguished by pain reflected down the ureter, hæmaturia and pyuria and Dietl's crisis in floating kidney, by bimanual examination of the abdomen. (*h*) *Mucous colic* (colitis) is differentiated by the characteristic stools and neurasthenic symptoms. (*i*) *Appendicitis* is characterized by its four cardinal symptoms (*q. v.*); Ochsner finds 10 per cent. of his appendicitis cases complicated by gall-stones and 33 per cent. of his gall-stone cases by appendicitis. (*j*) *Acute pancreatitis* (*q. v.*) which may complicate gall-stones, causes greater collapse and more frequent intestinal obstruction. (*k*) *Febrile syphilis* may simulate gall-stones (Riedel) as may also (*l*) the abdominal crises occasionally observed in angioneurotic œdema.

2. Other Affections of the Gall-bladder and Bile Ducts (see Brewer's Table on pages 704 and 705).

Prognosis.—The prognosis is difficult to formulate. Stones may pass without recurrence of colic and the passage of smooth calculi may justify some hope that they are the only ones. The first attack is usually the worst. Fever, infection, cancer and obstruction of the common duct and intestine must be covered in forecasts to patients refusing operation. Riedel held that 10 per cent. recover spontaneously under expectant treatment, while in 90 per cent. of cases surgical intervention is indicated.

Treatment.—1. Prevention.—Exercise and deep breathing aid the flow of bile. Lacing is a factor to be regarded. Plenty of water increases the secretion of bile. Digestive derangements should be corrected. Alcohol is contra-indicated. The predisposing gall-bladder inflammation seldom comes under the physician's care, but in suspicious cases, sodium salicylate, gr. x to xv t. i. d. given alternate weeks, acts as a cholagogue—increasing the bile two or threefold—and antiseptic; hexamethylenamine (urotropin), 3j per diem, is excreted by the bile (Crowe). Mercurials seem to stimulate bile formation less than to aid in its evacuation. Potassium iodide and mild salines (sodium phosphate, 3ss to j) are beneficial.

2. Colic.—Hypodermics of morphine must be given, gr. ¼ being combined with nitroglycerine gr. $\frac{1}{100}$ and (for the first injection in the attack) with atropine gr. $\frac{1}{100}$; repeated doses of morphine and nitroglycerine are often necessary. Inhalation of chloroform is indicated in severe cases until the morphine acts, but none of these drugs should be left in the possession of any patient. A full hot bath and hot fomentations over the liver are valuable accessories. Drinking warm water facilitates vomiting, which in turn relieves the pain.

3. Medical Treatment.—This in no way influences the *solution of gall-stones.* Durande's solvent (oil of turpentine 1 part to ether 4 parts) is useless and, like chloroform, is only antispasmodic. Olive oil only facilitates their final passage. Salicylates, calomel, salines (*v. s.*) and various "cures," as at Carlsbad, Vichy, Kissingen, Las Vegas or Bedford Springs, are useful in quieting concomitant or causative inflammation. Local massage is obviously injurious.

4. Surgical Treatment.—The author believes that gall-stones should be operated on when recognized, thus saving time, suffering and danger. The following *indications* are given by Kehr: "(*a*) The latent state ensues in many cases of cholelithiasis after rest-cures or alkalies and in certain cases is permanent, particularly in the so-called cases of chronic recurrent cholecystitis. But I doubt whether an absolute cure will frequently occur, *i. e.,* the passage of all the stones, by medical means. We should never endeavor to force a passage of the stones; it is much better to seek to render them quiescent in the gall-bladder and quiet the inflammation. The continuous use of hot applications for weeks, with rest in bed and Carlsbad waters, will give the best results. (*b*) Riedel's theoretical justification for early operation—the removal of the calculi while they remain in the gall-bladder—holds good now as ever, as in many cases an early operation is the only salvation for the patient against such grave dangers as perforation, cholæmia and carcinoma. Universal application of early operation, however, in general practice, is absolutely impossible. (*c*) I decline operative interference if the attacks run a mild course and in the intervals when complete latency exists, *i. e.,* entire absence of sensitiveness in the region of the gall-bladder. (*d*) Acute obstruction of the choledochus is to be treated

medically. Should cholangitic symptoms appear or should the jaundice induce loss of strength and absolute loss of appetite operation should be performed. (*e*) Operation is necessary when frequent colics occur, even without jaundice or the passage of stones, which damage the general health, deteriorate the earning capacity and destroy the pleasure of living. (*f*) Cases of jaundice which are always accompanied by the passage of stones belong to the physician; should the attacks multiply, the patient become vitally depressed and no hope be present for the passage of all the stones, the propriety of the operation is established. (*g*) Hydrops, empyema of the gall-bladder and pericholecystitic abscesses belong to the province of surgery. (*h*) Surgical treatment should not be postponed too long after the failure of a Carlsbad cure. (*i*) Gall-stone patients who have become addicted to the use of morphine must be operated upon. During the after-treatment the best opportunities are afforded for curing the opium habit. (*j*) Treatment of carcinoma of the gall-bladder can only be of lasting benefit if the case is operated upon early, but everyone dreads early operation and late operations are not of much use. (*k*) Those suffering with chronic jaundice not dependent upon stone in the common duct or incurable disease of the liver must be operated upon at the latest three months after the appearance of the icterus, for it not infrequently occurs that in place of a suspected carcinoma of the head of the pancreas a curable chronic interstitial pancreatitis will be found. (*l*) The decision for operation will be rendered easy for both physician and patient upon demonstration of tumor of the gall-bladder, a swollen liver and the appearance of jaundice and fever. We may also operate on cases without demonstrable local lesions of the gall-bladder and liver which exhibit severe and continuous symptoms which do not prove amenable to internal medication. We find in such cases, particularly often in men, adhesions of traumatic origin without stones. (*m*) The sequelæ of cholelithiasis, purulent cholangitis, abscess of the liver, peritonitis due to perforation, subphrenic abscess, pyloric and duodenal stenosis and gall-stone ileus must be treated surgically. (*n*) Concluding: To present general indications for operation is not readily possible. One must decide each case on its own merits. Men, particularly corpulent ones, do not bear the operation well. Women who have borne children lend themselves particularly well to this surgical procedure. If possible one should refrain from operating in cases of diabetes, arteriosclerosis, chronic nephritis and diseases of the lungs and heart."

In Mayo's 1,500 operated cases the mortality was but 1 per cent. in 416 uncomplicated cases, while those complicated by suppuration or cancer brought the average mortality to 5 per cent.; the death rate in operations on the common duct was 11 per cent. These figures appear to the writer as the best possible argument for early operation. Cholecystotomy was attended by a 2.5 per cent. mortality and it is therefore the better operation, as cholecystectomy entailed

4.3 per cent. of fatalities. In Kehr's last 500 cases (1904) the mortality was 3 per cent. in uncomplicated cases and 13 per cent. with cancer and cholangitis; stones were left in 2.5 per cent., hernia developed in 3 per cent. and adhesions and inflammatory colic occurred in 5 per cent.

CHOLECYSTITIS.

Etiology.—Inflammation of the gall-bladder may occur before, after, with or without inflammation of the bile passages. (*a*) *Predisposing factors are gall-stones* (especially large ones which cannot pass, which cause 65 per cent. of cholecystitis and cholangitis (Dominici) or 90 per cent. in other statistics), bile stasis, trauma, foreign bodies or previous attacks. (*b*) The *exciting cause* is *bacterial;* the colon, typhoid, pyogenic, pneumococcus or other organisms are responsible. They may ascend the bile channels or enter with the blood current. Inflammation from typhoid infection (Louis and Andral) may occur without actual typhoid or may develop twenty years later (Osler).

Symptoms.—The symptoms vary with the intensity of infection, which is almost synonymous with the type (simple, membranous, suppurative, phlegmonous, ulcerative or gangrenous). In light forms the symptoms may not be recognized; this corresponds to post mortem adhesions in cases with no history of cholecystitis. In other cases, perhaps more severe ones, symptoms are obscured by the causative typhoid or by gall-stones. In the average case there are (*a*) *pain over the part,* which may be severe or paroxysmal and may radiate toward the appendix or back. (*b*) *Tenderness* as in gall-stones; it is often first general and then local and the right rectus is tense; it is clearly elicited by a thrust over the part during inspiration. (*c*) *Fever, increased pulse* rate, *vomiting* or nausea; fever is absent in mild infections; in typhoid, cholecystitis may be confused with typhoid relapse (Musser). There may be *colic.* (*d*) There may be a *palpable gall-bladder.* Its contents may be purulent (empyema cystidis felleæ) or serous (hydrops cystidis felleæ). Hydrops may enlarge the gall-bladder to the size of the fist or in extreme cases so that it weighs 50 to 60 pounds, contains thirty to forty quarts and fills the entire abdomen; experienced observers have mistaken such cases for ascites; the fluid is alkaline or neutral, serous or mucoid. If very tense the gall-bladder may not fluctuate or if very lax may not be palpable (see Floating Kidney). There may be pus in the gall-bladder without enlargement. In chronic cases it may shrink. (*e*) *Severe sepsis* may intervene in diffuse suppurative cholangitis or pylephlebitis; this is evidenced by septic fever, chills, leukocytosis, bacteriæmia, swollen liver and spleen, nephritis and ulcerative endocarditis. Icterus occurs in 33 per cent. (See pages 704 to 705.)

Prognosis.—Many cases with cholangitis die in spite of operation. Mild cases may subside, but recurrence and formation of gall-stones are frequent. Under expectant therapy the outlook is serious in severe cases, for perforation, localized or general peritonitis, adhe-

sions (in 75 per cent.) and duodenal or pyloric obstruction may result.

Treatment.—Rest, milk diet, local heat and morphine may suffice for light cases, but in the more severe infections drainage is indicated.

CHOLANGITIS, CATARRHAL ICTERUS, CONGENITAL OCCLUSION.

Suppurative Cholangitis.—Suppurative cholangitis usually is diffuse, co-exists with suppurative cholecystitis and results from gall-stones with infection. Infection is usually *hæmatogenous* through the hepatic artery or the portal vein, but may be ascending from cancer or stone in the tracts. The symptoms of cholangitic sepsis have been described under cholecystitis. Drainage is indicated. (See page 704.)

Acute Catarrhal Jaundice.—Acute catarrhal jaundice (cholangitis catarrhalis, icterus simplex seu gastroduodenalis) results from gastroduodenal catarrh which causes swelling with obstruction of the papilla and probably catarrhal inflammation of the lower portion of the common duct; few cases come to autopsy, though a small plug of mucus usually occludes the opening. Eppinger found swelling of the lymphoid follicles in that part of the common duct which lies in the wall of the bowel. Its usual causes are practically those of acute gastritis or enteritis. Favoring factors include passive congestion (cardiac or portal), acute infections, toxic causes (as phosphorus), nephritis and other diseases of the liver or bile tracts (cirrhosis, cancer, stones, parasites). Epidemic forms occur or forms which seem to be a separate type of infection (*v.* Weil's Disease).

Symptoms.—*Gastro-intestinal catarrh* occurs first in many cases and after a few days to a week or so *icterus* appears, with its usual symptoms, sudden decolorization of the stools and bright yellow staining of the skin and urine (*v.* Icterus). However, the physician must not expect to always find an antecedent gastroduodenitis, because a primary infectious catarrh of the bile ducts is equally frequent. Other findings are inconstant; the liver is not enlarged at first, but may become slightly swollen later; there is no pain over the liver or ducts, emaciation if present is slight, the spleen and gall-bladder are seldom enlarged and the *diagnosis* depends entirely on the mode of onset in young, healthy subjects, sequence of symptoms and exclusion of other causes of obstruction. In advanced life catarrhal jaundice is uncommon and gall-stones and cancer are always to be thought of first. See pages 704, 705.

Prognosis.—The prognosis is almost always good, the jaundice lasting four to eight weeks. The author has seen cases clear in a week and others last five months, though a course of over four to eight weeks renders the diagnosis uncertain. Cases with fever may run a prolonged course. Death rarely occurs. Cases of fatal hemorrhage (Toelg and Neusser), and in the aged, of exhaustion (Leichtenstern) and of suddent death with sudden heart-failure are reported.

Treatment.—(*a*) *Causative* factors, as gastritis or cardiac failure, should be appropriately treated; dyspeptic symptoms yield most readily to gastric lavage; fractional doses of calomel at night followed by a mild saline in the morning, should be given; but it should be remembered that active catharsis augments or indeed initiates catarrh.

℞ Acidi hydrochlorici diluti ..... ʒj.
Tincturæ nucis vomicæ ..... ʒiv.
Tincturæ gentianæ co. .....q.s. ad ʒiv.
M. et S.—One teaspoonful after meals in half a glass of water.

Calomel was formerly given because it produced green "bilious stools"; this color is not due to bile but to mercuric sulphide. (*b*) In the *diet* fats are particularly to be avoided, as neither the stomach nor intestine can digest them; tea, coffee, alcohol and coarse foods should be interdicted; skimmed milk is given and well-cooked carbohydrates are gradually added to the diet; toast, eggs and mealy soups may be given; meat is often poorly tolerated, seemingly because of exclusion of the pancreatic secretion. (*c*) As long as acute dyspepsia exists, *rest in bed* is indicated, in order to maintain nutrition on the lowest possible diet. (*d*) The bowels should be regulated by *copious injections* of water which are thought to incite peristalsis and therefore dislodge the obstructing mucus plug. (*e*) The *itching* is allayed by 1 per cent. carbolic solution, the use of which requires care, as the urine sometimes becomes cloudy; acetanilide, gr. v q. i. d.; potassium bromide, ʒss, well diluted, by rectum once or twice daily; an occasional hypodermic of pilocarpin hydrochloride gr. ⅛, warm baths to relieve the dryness of the skin and calcium chloride gr. x to xx t. i. d. for a few days are indicated.

Chronic Catarrhal Cholangitis.—Chronic catarrhal cholangitis rarely follows the acute form but more often results from occlusion of the common duct by calculus, cancer, stricture or pressure from without (*v. gall-stones, pancreatic cancer and icterus*).

Congenital Occlusion of the Bile Ducts.—Sixty-three cases are recorded (Lavenson). Rolleston thinks that some poison initiates cholangitis, which results in a mixed cirrhosis and obliteration of the ducts. Lavenson holds that the ducts are congenitally atresic. Icterus develops within a month after birth and death results in a few weeks or months—nearly always within a year—from cholæmia or hemorrhages.

TUMORS OF THE GALL-BLADDER AND BILE VESSELS.

I. Cancer of the Gall-Bladder.—Tumors other than cancer are rare and of purely anatomical interest, as papilloma, fibroma, lipoma or adenoma. Rolleston states there are but 9 recorded cases of sarcoma. Fütterer (1901) collected 268 cases of cancer, which is certainly more frequent than these figures indicate.

Etiology.—The most striking etiological factor is *gall-stones,* pres-

ent in 70 per cent. (Musser) to 91 per cent. (Courvoisier); it is estimated that 9 per cent. of cases of gall-stones result in cancer of the gall-bladder, but this seems a very high percentage. Eighty per cent. occur in women, caused by lacing (Fütterer); the average age of subjects is fifty-eight years.

Pathology.—The tumor may be scirrhus or medullary, but histologically is usually of the columnar type (adenocarcinoma); the spheroidal and squamous types are rare.

Symptoms.—(*a*) *Gall-stones* which irritate the mucosa and cause most cases, usually produce no symptoms, though previous colic may be noted in the history. (*b*) A *tumor of the gall-bladder* is palpable in over 66 per cent., first as a smooth oval swelling and later as a larger and more nodular one; it is usually due to the growth, for the gall-bladder itself is generally shrunken. The fundus is the usual point of origin, being most irritated by calculi; much more rarely the neck of the gall-bladder or least frequently the cystic duct is the point of origin. The tumor appears below the edge of the liver with which it moves and with which it is consecutive on palpation, percussion and inflation of the colon. There may be local discomfort and, in rare cases, severe pain like that of gall-stones. (*c*) *Cachexia* appears with all its attendant symptoms.

Complications.—*Secondary growths* in the liver occur in 58 per cent. by lymphogenous routes or by invasion from contiguity and sometimes cause hepatic enlargement; from the liver extension by the hepatic veins may occasion systemic metastases. *Icterus* occurs in 69 per cent. (Musser) from malignant or inflammatory involvement of the bile ducts; cholæmia is a common termination. Using Musser's figures, growths in the peritoneum occur in 12 per cent., in the lungs or pleura in 10 per cent., in the abdominal lymph glands in 16 per cent. and ulcerative fistula into the colon in 10 per cent. Warthin records adrenal metastases with pigmentation like that of Addison's disease and with vitiligo. Pressure on the pylorus may cause its stenosis, vomiting or dyspepsia. *Ascites* occurs in 25 per cent. and is due to mechanical compression of the portal vein or to malignant peritonitis. Suppurative cholecystitis and cholangitis may develop and the case then terminates by sepsis, ulcerative endocarditis, etc. The differential *diagnosis* is considered under gall-stones. The clinical course averages six months. *Treatment* is merely palliative, unless the affection is diagnosticated early enough for operation or is accidentally found in operating for other conditions. Removal of the gall-bladder cancer has resulted in a few permanent cures (Hochenegg, Robson, Warren).

II. Tumors of the Extra-Hepatic Bile Ducts.—Rolleston states that 80 cases are reported. These tumors are usually small, show little tendency to infiltrate and are cancers of the columnar type. The common duct is most often affected, the hepatic duct less frequently and the cystic duct least frequently; these tumors usually occur between the years of sixty and seventy and two-thirds occur in males.

The salient symptom is intense *icterus,* which develops early, increases steadily, occurs in nearly all cases, causes icteric necrosis of the liver cells in the central zone (Fütterer) and precipitates early cholæmia before metastases or cholangitic sepsis have time to develop. The gall-bladder is almost always dilated anatomically in tumors of the common duct and is felt clinically in over 50 per cent. Pain is not common, though gall-stones may be simulated (*q. v.*). Ascites, dyspepsia and gastro-intestinal hemorrhages from cholæmia may develop. Differentiation from duodenal or pancreatic tumors (*q. v.*) is usually impossible. *Palliative treatment* consists of draining the gall-bladder to relieve the icterus; radical treatment is extirpation of the deeply located tumor (Kehr, Halsted).

DISEASES OF THE PANCREAS.

ACUTE PANCREATITIS; FAT NECROSIS.

Etiology.—*Infection* ascending from the intestines is the leading cause. (*a*) *Calculus obstructing the common duct,* by allowing bile to regurgitate into Wirsung's duct, may cause acute pancreatitis (Opie), especially when the gall-bladder, the natural reservoir for obstructed bile, is shrunken. Egdahl (1907) found gall-stones in 42 per cent. of acute fat necrosis of the pancreas. According to Quénu and Duval (1906) 118 cases are recorded in which pancreatitis occurred in the course of cholelithiasis. Flexner concludes that bile salts may initiate inflammation and fat necrosis, even when there is no infection. Though the accessory duct of Santorini may carry off the pancreatic juice when there is obstruction of Wirsung's duct, it is too small for much compensation in 66 per cent. of cases (Opie); (*b*) *metastatic infection* is an infrequent cause; in one case the author observed acute pancreatitis in which an acutely inflamed ulcer of the leg was the atrium of infection; (*c*) in some cases suppuration or *ulceration in contiguous tissues* is causative.

Predisposing Factors.—(*a*) *Age and sex;* in Körte's collection 90 per cent. occurred in *males beyond middle life.* (*b*) *Obesity;* (*c*) *alcoholism;* (*d*) trauma; (*e*) gastro-intestinal catarrh; and (*f*) parturition in 6.6 per cent. of Peiser's collection of 128 cases are factors.

Symptoms.—The clinical picture was clearly described and defined by Fitz (1889), though cases were reported earlier (Oppolzer). The *onset is sudden* in adult stout males who have enjoyed previous good health or who have a history of alcoholism or of gall-stones.

1. *Epigastric pain* is the *initial symptom;* it is very sudden and severe, is often agonizing, may be constant or paroxysmal and may be confused (or coincident) with gall-stone colic, though it is even more

intense; it is diffusely epigastric, to the right or left of the median line, or rarely over the head of the pancreas. The author has seen radiation into both axillæ. The intensity of the pain is referable to pressure on the cœliac plexus (neuralgia or possibly neuritis cœliaca). Much less often the pain is gradual in onset.

2. *Epigastric tenderness* is usual, often over the head or sometimes more over the tail of the pancreas, whence the inflammation may extend to the left pleura with tenderness and friction, as in one of the author's reported cases. The recti are tense and the epigastrium is swollen.

3. *Vomiting* is early and severe and brings up food, mucus and sometimes blood.

4. *Collapse* soon follows; it is often very severe, threatens immediate dissolution and too often prevents surgical interference. It is due to pressure on or actual inflammation of the closely contiguous cœliac plexus and semilunar ganglia. There may be marked *cyanosis.* In one of the author's cases there were relapsing shock with cyanosis and a pulse of 140, which was so weak that no surgeon would operate during the two weeks before recovery. Collapse usually causes death in 2 to 4 days in unoperated cases.

5. *Fever* is not constant; there may be no elevation or an irregular rise to 103 or 104 degrees. Chills are most common in late suppuration.

6. The *pancreas can rarely be felt* because of its deep position and because the epigastrium is tense and tympanitic.

7. The *bowels* are constipated, simulating obstruction, though flatus is passed. Operation or autopsy at this stage shows an acute pancreatitis, with swelling, a variegated yellow-red or black color, exudation of fibrin, pus cells, blood (hemorrhagic pancreatitis) and areas of acute *fat necrosis* (Balser, 1882). Fat necrosis is caused by the fat-splitting steapsin (Langerhans, Hildebrand, Dettmer and Flexner), which produces glycerine and insoluble fatty acids from the pancreas and contiguous adipose tissues, *i. e.,* omentum, mesentery and peritoneum; the glycerine is absorbed, the fatty acids are precipitated with lime as opaque white areas of necrosis and some of the ferments occasionally escape into the blood, producing toxæmic symptoms or necrosing fatty tissue elsewhere, as in the pericardium or skin.

8. If the patient lives the *symptoms of localized peritonitis* develop in the upper half of the abdomen and sometimes more to the left side; a pus sac may fill the lesser peritoneal cavity; the localized pain may become general from diffuse fat necrosis. In one of the author's cases great dyspnœa attended diaphragmatic pleurisy.

9. *Septicopyæmic symptoms* may appear, as rigor, hectic fever, delirium, pleurisy, hiccough, icterus, diarrhœa and loss of weight and strength; in one case the author saw acute splenic tumor, acute nephritis and universal swelling of the lymph nodes.

9. Acute *glycosuria* is not common, though present in one of the author's cases.

In the third week *rupture* into the colon may occur; this is attended by lancinating pain, intestinal hemorrhage, even passage of the pancreas by the bowel (Chiari) and relief from all symptoms.

Suppurative types, of which Körte collected 46 cases, may begin gradually, without acute gangrene, fat necrosis or hemorrhage. The symptoms are less acute and severe than those of the acute type. The suppuration is localized or there is a diffuse purulent infiltration; it may cause pylephlebitis, liver abscess, subphrenic abscess, burrowing of pus into the peritoneum, loin (which Körte considers rather characteristic), peripheral tissues or alimentary tract. Protracted suppuration is said to cause glycosuria, skin pigmentation and anasarca. Körte found 40 cases of *acute gangrenous pancreatitis.*

Diagnosis.—The cardinal features are (*a*) the sudden onset, (*b*) violent epigastric pain and tenderness, (*c*) vomiting, (*d*) collapse, (*e*) epigastric distention. The absence of indican under these circumstances is suggestive of a pancreatic lesion, because trypsin converts albumin into peptone, which in turn becomes partly converted into indol, the source of indican in the urine. Since leucin and tyrosin are formed in the normal intestine as a result of the action of the pancreatic juice upon proteids, Ulrich attaches much importance to their absence from the urine. Robson finds calcium oxylate crystals in 30 per cent. of all varieties of pancreatic disease.

The *pancreatic reaction* of Robson and Cammidge is a test for glycerine in the urine; it is complicated and still *sub judice.*

Differentiation.—(*a*) *Acute poisoning* is eliminated by the history and gastric contents. (*b*) *Intestinal obstruction* is less severe in onset; collapse is usually attended by distention and often by intestinal rigidity or peristalsis above the obstruction; indicanuria is usual; epigastric distention alone is unusual; in one pancreatic case, the author saw obstruction lasting nine days. Operation, indicated in either instance, may be necessary for diagnosis. (*c*) *Gall-stone colic* is sometimes hard to differentiate but is rarely as severe as pancreatic pain; collapse or epigastric distention is infrequent. (*d*) In *perforating* duodenal or gastric *ulcer* the later collapse, pain and the history are significant. (*e*) Mesenteric embolism and appendicitis must be considered.

Prognosis.—The malady generally evinces a severe progressive tendency. A fatal attack may be the last of several milder seizures. Fitz has found at autopsy fibrous tissue and crystals or granules of hæmatoidin which he regards as evidence of previous hemorrhage and inflammation. Death may occur within a few hours to three days from collapse, in two or three months from sepsis or in six months from diabetes (Fitz). Death at an even later period may result from sepsis or diabetes. The pancreas may slough into the intestine, as in Trafoyer's and Chiari's cases, with recovery.

Treatment.—Rectal feeding and stimulation, morphine hypodermatically and heat are indicated during the initial shock. Though early operation is dangerous because of shock and hemorrhage, the

author considers that it is indicated lest local extension or general toxæmia develop. In 21 cases (Quénu and Duval), 62 per cent. of the acute cases died after operation, 100 per cent. of the hemorrhagic and 45 per cent. of the acute suppurative cases.

CHRONIC PANCREATITIS.

Etiology and Pathology.—(*a*) *Gall-stones* in the common duct may induce chronic catarrh of Wirsung's duct, leading to chronic pancreatitis (Robson). (*b*) *Cancer,* gastroduodenal *catarrh* and *ulceration* (alcoholism, syphilis and pancreatic calculi) may produce the same results. In these instances *ascending infection* appears to be the potent factor, causing *interlobular fibrosis* (Opie), *i. e.,* fibrosis around the pancreatic lobules; diabetes is a late and uncommon sequence. (*c*) *In liver cirrhosis* and *hæmochromatosis* (see Diagnosis Portal and Biliary Cirrhosis) *an interacinar fibrosis* occurs, developing in the lobules, diffusely invading the islands of Langerhans and causing glycosuria (Opie); it does not result from obstruction of the common duct.

Symptoms.—Riedel, who first described the affection, and Robson state that chronic inflammation is found more frequently with increasing thoroughness of operations for gall-stones. Symptoms may be lacking. They vary with the cause. In catarrh due to gall-stones there is a history of *colic, jaundice* and sometimes *intermittent fever. Tenderness* in the epigastrium, a fulness above the umbilicus and loss of flesh are noted and if pancreatic symptoms predominate the pain passes from the epigastrium to the left side, to the scapular or renal regions. If gall-stones are not the cause, there may be merely aching or painful attacks which are not at all pronounced; pain may be absent. The gall-bladder may enlarge from obstruction of the common duct when the liver becomes enlarged and frequently cirrhotic (Robson). According to Robson marked *enlargement of the spleen* is frequent, bile is found in the urine in 60 per cent., *calcium oxalate crystals* in 40 per cent. and glycosuria in 5.5 per cent. (90 cases). Opie found glycosuria but once in 22 cases. Riedel, Körte, Robson, Moynihan and others have described a *tumor-like induration* of the head of the pancreas, which simulates cancer because of the emaciation and deep icterus it produces; differentiation may be impossible, even during laparotomy; it is more common in subacute than in chronic cases. As in tumor, excessively copious, pale stools, laden with fat and undigested muscle-fibers, are noted. Polyuria has been reported.

Differentiation from stone in the common duct and from cancer is very difficult.

Prognosis.—Without surgical intervention death is likely to occur from emaciation, cholæmia, diabetes or hemorrhagic diathesis.

Treatment. —Drainage of the gall-bladder or anastomosis is indicated. Robson operated on 62 cases with 13 per cent. mortality,

Quénu and Duval on 62 cases with 13 per cent. mortality and Moynihan on 14 cases without a death. Hemorrhage is a dreaded complication; calcium lactate, gr. xx to 3j t. i. d. before operation, is said to increase the coagulability of the blood.

PANCREATIC APOPLEXY.

Pancreatic hemorrhage has been described by Spiess (1866), Klebs (1870) and Zenker (1876); Anders (1899) collected 40 cases. The anæmias, syphilis, alcoholism, acute infections and local lesions of the pancreas seem to be *predisposing causes;* 66 per cent. occur *in men.* Doubtless many reported cases are acute hemorrhagic pancreatitis. *Sudden onset,* severe *collapse, epigastric pain,* intestinal obstruction, vomiting, restlessness and normal or subnormal temperature mark the affection; death sometimes occurs within a few hours. If the patient survives the initial stage suppuration may develop in the pancreas and tissues into which the hemorrhage burrows. Hemorrhagic cysts may form, which should not be confused with cysts into which secondary hemorrhage has occurred. The diagnosis from acute pancreatitis is rarely made and the treatment of both affections is identical.

LITHIASIS.

About 100 cases of sialolithiasis pancreatica are recorded. The *etiology* is probably like that of gall-stones, infection of Wirsung's duct; stagnation of secretion is a predisposing factor, whence the occasional association with gall-stones, pancreatic inflammation and tumor.

Symptoms and Diagnosis.—Confusion with gall-stones is often inevitable, for colic without icterus is indicative of either condition and their co-existence is not uncommon. The pain may radiate to the left side and shoulder; Lancereaux, Lichtheim, Leichtenstern, Minnich, Holtzmann, Moynihan and Kinnicutt have recognized cases. In some of them the stones were found in the fæces during life, in others at autopsy. Salivation is reported. In Lichtheim's case there was colic, glycosuria (found in 45 per cent of cases) and diarrhœa. The stones are single or multiple (even 300), white-gray, round, oval, irregularly outlined or branching, hard or friable and vary in size from that of almost impalpable gravel to that of a hazel-nut. They consist largely of calcium carbonate and phosphate. Shattuck described an oxalate calculus. Their sequelæ are (*a*) obstruction and inflammation of Wirsung's duct and (*b*) acute suppurative pancreatitis. The treatment in the cases which are fortunately diagnosticated is surgical; Moynihan recognized and successfully operated on a case. Gould operated on a case which did not recover.

PANCREATIC CYSTS.

To Nicholas Senn (1885) belongs the credit of establishing their clinical picture. Bessel-Hagen (1900) collected 149 cases.

Etiology and Pathology.—Probably many reported cases are extra-pancreatic, especially (*a*) the *traumatic* cysts of which Honigmann in 1905 collected 69 cases. (*b*) Duct obstruction (*ranula pancreatica,* Virchow) by calculi, strictures, parasites, tumors or inflammation in the smaller ducts is an etiological factor. (*c*) *Cystadenoma* is the cause of most cysts (Lazarus). (*d*) Cysts rarely result from an *old hemorrhage.* In all forms the cyst causes considerable parenchymatous atrophy. (*e*) *Sex.* Traumatic and inflammatory forms are most frequent in men and the cystadenomatous type in women. (*f*) *Age;* over 50 per cent. are found in the third decade of life, though a congenital form, sometimes with cystic liver and kidneys, is found (Richardson).

Symptoms and Diagnosis.—1. In *cysts,* (*a*) a *deep retroperitoneal* swelling is found, (*b*) the location of which is more or less *central,* but is *above the navel* in 87 per cent., where other cysts are most uncommon; it lies behind or between the inflated stomach and colon; it sometimes lies below the colon but rarely above the stomach; very large cysts (which contain 10 to 20 quarts) may fill almost the entire abdomen. A cyst may in rare cases develop in the tail of the pancreas, when it points toward the spleen or left kidney. (*c*) Its form is *round, oval* and *smooth;* (*d*) its consistence often *varies;* (*e*) it is *immobile* on palpation and on inspiration, or at the most can be moved slightly in a vertical direction. (*f*) The *fluid* is alkaline, 1,010–25 specific gravity, usually mucoid, blood stained in 82 per cent. (Körte), and in 45 per cent. contains one or more pancreatic ferments (diastatic, saponifying or tryptic) of which the latter is alone characteristic, for the other ferments occur in various exudates. The fluid sometimes contains fat and cholesterin; aspiration is dangerous, because the tension of the cyst is great and fat necrosis or peritonitis may result from leaking after exploratory puncture.

2. So-called *pancreatic symptoms* are rare, as emaciation, salivation, stools laden with undigested fat or muscle-fibers or glycosuria.

3. Symptoms of *peritonitis* or of *intestinal obstruction* may occur in traumatic or inflammatory types and after rupture. The author saw a case of Murphy and Wittwer in which the picture after rupture was exactly that of subacute tuberculous peritonitis; after two ruptures the case was successfully operated on. Rupture into the bowel is followed by diarrhœa and disappearance of the tumor.

4. *Pain* like that of cholelithiasis may be present, but it more often radiates to the left side, shoulder and neck. Pressure symptoms include dyspnœa, vomiting, icterus, emaciation and polyuria.

Differentiation from ovarian, renal and other cysts is largely a topographical question. Ovarian cysts have their pelvic origin and connection, but hydronephrosis is movable and originates in the

flank to which pancreatic cysts seldom penetrate. Fluid in the lesser peritoneal sac or mesenteric, omental and retroperitoneal cysts are difficult to distinguish. Cysts of the liver present respiratory excursion and other obviously hepatic symptoms.

Treatment.—Aspiration is never permissible. Enucleation of the sac is more difficult and more dangerous than simple drainage. In Bessel-Hagen's 149 cases the operative mortality was 6.7 per cent. Diabetes has followed operation (Bull and Krönig).

TUMORS OF THE PANCREAS.

Cancer of the pancreas constitutes 67 per cent. of all pancreatic diseases but possibly this estimate must be discounted because confusions with benign induration have been frequent until of late years. It occurs in 0.6 per cent. (Eppinger) to 1 per cent. (Soyka) of autopsies. In 132 cases of pancreatic tumors (Segré) 127 were cancer, 2 sarcoma, 2 cysts and 1 gumma. *Sarcoma* is usually secondary from contiguous lymphosarcoma in the retroperitoneal glands; 21 cases are reported.

Cancer is usually scirrhus, less often of the medullary, colloid or adenomatous type. It most often begins in the head of the organ. In some cases it may be secondary to pyloric cancer.

Symptoms.—(1) *Tumor* is the commonest and surest symptom, occurring in 25 to 50 per cent.; it lies *deep,* beside the spine at the level of the navel and between the pylorus and colon. It is tender, practically *immobile* and varies from the size of a walnut to that of a child's head, though small tumors are commonest; in some instances it may show propagated pulsation from the aorta or a bruit from its compression. Palpation should be made with the stomach and bowels empty. (2) *Icterus* occurs in 72 per cent., increases gradually and is more often *continuous* than remittent. In some cases there may be rigors and pain as in biliary calculi; occasionally, just before death, the growing anæmia seems to lessen the jaundice. Bard and Pic state the liver is usually rather small. (3) *The gall-bladder* is usually *enlarged* (Battersby, Bard and Pic); see *differentiation.* Cochez holds that it shrinks when the hepatic or cystic duct is obstructed. (4) *Pain* and *early vomiting* are usual. The pain is epigastric, severe and sometimes neuralgic (neuritis cœliaca) or radiating as in pancreatitis or lithiasis. (5) Cachexia is marked and rapid; great prostration, emaciation and a syncopal tendency occur in tumor as in other pancreatic lesions. The temperature is usually subnormal. (6) The *stools* may contain much fat (Kuntzmann, 1827, and Bright, 1833); in *steatorrhœa* the fatty acids and neutral fats are more abundant than the fatty soaps (Müller); its significance grows if diarrhœa and icterus are not present. The stools also contain many undigested muscle-fibers (*lientery*) and sometimes blood is present. (Sahli's test for albumin digestion is made by giving salol in divided doses in glutoid capsules; as the glutoid is not dissolved, no carbolic

acid appears in the urine.) (7) The *urine* may contain sugar; Mirallié holds that *glycosuria* occurs in 26 per cent., though in Robson's 15 cases it was absent. It is said to be an early symptom and to disappear before death or with the increasing icterus. *Albuminuria* is more frequent than glycosuria. The *indican is decreased.* Peptone, maltose (once, by v. Ackeren) and fat (Clark and Bowditch) have been found. Polyuria and bronzing of the skin are rare findings. *Complications* include pyloric or duodenal obstruction; compression of the aorta or cava; retention cysts of the pancreas; and extension by contiguity to the portal vein, colon, peritoneum, ureter or lungs.

Differentiation.—1. Chronic *pancreatitis* with icterus and tumor may not be differentiated even by laparotomy (Riedel), though ascites indicates neoplasm and recovery means inflammation. The pancreatic cachexia of French writers, occurring in many lesions of the pancreas, may be confused with the cachexia of cancer.

2. In *duodenal cancer* (*q. v.*) the growth is also fixed and is attended by icterus, but cancer above the papilla causes rather the picture of pyloric stenosis; in cancer below the papilla pancreatic juice may be recovered from the stomach.

3. *Liver disease,* with the frequent icterus, ascites and splenic enlargement, causes little difficulty in differentiation.

4. Calculus in the Common Duct.—*vs.*—Compression of Common Duct by Pancreatic Tumor.

1. History of colic usual.	Absent.
2. Tenderness of gall-bladder usual.	Absent.
3. Icterus, often remittent.	Permanent.
4. Liver less large than in—	pancreatic tumor.
5. Intermittent fever, and rigors.	Subnormal temperature.
6. Gall-bladder (see gall-stones) shrunken in 80 per cent.	Dilated in 92 per cent. (Courvoisier's law).
7. Slower course.	Very rapid emaciation.

According to Bard and Pic the icterus, enlarged gall-bladder, rapid emaciation, cachexia, subnormal temperature and normal or smaller liver of pancreatic cancer can only be confused with some few cases of gall-stones, cancer of immediately adjacent parts, primary cancer of the liver or bile-passages and primary duodenal or gastric cancer with early diffuse generalization in the liver. Kehr states that chronic obstruction of the common duct is cancerous in 70 per cent., pancreatitic in 20 per cent. and calculous in 10 per cent.

Treatment is largely symptomatic; morphine should be given for pain, the gall-bladder should be drained (cholecystotomy) or it should be sutured to the intestine (cholecystduodenostomy). Very few permanent recoveries follow extirpation.

DISEASES OF THE PERITONEUM.

ACUTE DIFFUSE PERITONITIS.

Acute inflammation of the peritoneum was at one time far more important as an independent affection than at present; we now regard it as chiefly symptomatic of some other disease, *e. g.,* appendicitis, salpingitis, puerperal sepsis, etc.

Etiology.—The *actual cause* is probably always *mycotic.* Some writers classify peritonitis as bacterial (especially suppurative forms), chemical (serous and sero-hemorrhagic forms) and mechanical (adhesive forms). The Streptococcus pyogenes (puerperal and traumatic forms) and Bacillus coli (forms due to intestinal lesions) are the two most frequent organisms. The staphylococcus, pneumococcus and tubercle bacillus are fairly frequent and less often the Bacillus pyocyaneus, proteus, anthracis, typhosus, aërogenes encapsulatus, the ray fungus, Bact. lactis, gonococcus, Amœba coli, etc. Generally speaking, mono-infections are less common than *mixed infections.*

Factors causing *lessened physiological resistance* include nephritis (in 10 per cent.), alcoholism, cardiac disease, scurvy, etc.; in other words peritonitis may be a terminal infection, fatal to reduced subjects.

Atrium.—Compared with pleurisy and pericarditis, peritonitis is seldom primary. Though it may follow inflammation of every abdominal viscus, it is most commonly a secondary manifestation of intestinal disease (*e. g.,* appendicitis) or of pelvic diseases in women. (*a*) Infection through the *alimentary tract;* appendicitis, bowel ulceration (typhoid, carcinomatous, tuberculous), bowel obstruction, perforation, traumatism and infarction may cause it. Gastric are less frequent than intestinal lesions and ulcer and cancer rank first. (*b*) The internal *female genitalia;* it may result from gonorrhœa (Nögerrath), extra-uterine pregnancy, puerperal infections and septic abortion; peritoneal infection from diseases of the male genitalia is rare. (*c*) *Liver and bile vessels;* local or general peritonitis may result; peritonitis is more common as a result of hepatic abscess and syphilis than of echinococcus, cancer, pylephlebitis or cholecystitis. (*d*) *Pancreas;* acute pancreatitis. (*e*) *Kidney* and bladder; pyonephrosis, calculi or ulcerations of the bladder. (*f*) Rarer causes are splenic affections, retroperitoneal adenopathies, spinal or costal caries, lesions of the thoracic duct or the abdominal parietes or extension through the diaphragm. (*g*) *Metastatic peritonitis* is not common, though observed in septicopyæmia and acute infections. (*h*) In *children* fœtal peritonitis has been observed by transplacental infection; in the new-born the umbilical vein may convey infection, though peritonitis occurs in but 8 per cent. of fatal cord infections. It occurs more commonly through the lymphvessels of the cord.

Age and Sex.—Most cases occur between the ages of fifteen and forty and women are more often affected than men.

Symptoms.—Symptoms are local and general.

1. Local or Abdominal.—(*a*) *Pain,* the usual initial symptom, is almost invariable, is continuous, begins near the navel and remains greatest there; a causal gastric ulcer may produce pain in the back, salpingitis is attended with early pelvic pain and appendicitis has its own initial localization, but the pain of diffuse peritonitis is felt at or below the navel. (*b*) *Tenderness* accompanies pain; the tenderness is deep rather than superficial; tenderness and pain are very rarely absent and are caused by inflammation; at the operation or autopsy both the visceral and parietal investments are injected, reddened, lustreless, covered with a thin film of fibrinous exudate which binds the intestinal coils together; not infrequently the peritoneum is dotted with minute petechiæ. (*c*) The *attitude* is characteristic; the knees are drawn up and the head lifted to relieve tension and, in marked cases, to keep the bedclothes from contact with the abdomen. (*d*) *Vomiting* is reflex; it occurs early and greatly aggravates the pain; the vomitus contains food and later bile; it may be green (the *vomitus herbaceus* of the older writers), rarely dark brown or fæcal. It is accompanied by redness and dryness of the tongue, great thirst and singultus which is a sensory phrenic symptom; toward the fatal issue vomiting and singultus cease. (*e*) The *abdomen* on inspection and palpation is at first *tense, retracted* and *scaphoid,* but *later* becomes *tympanitic* from paresis of the inflamed intestinal coils (Stokes) and decomposition of their contents; the diaphragm stands high and its movement is restricted; in some, especially muscular subjects, the abdomen may be retracted throughout the course. Exploration by percussion may elicit *dulness,* which is usually due to serous, seropurulent or perhaps serohemorrhagic exudation, which may be effused in small pockets between fresh adhesions or may fill the entire cavity, amounting to even 30 or 40 quarts and sometimes fluctuating to the hand; *as a rule* a *tympanitic note* prevails; in some cases dulness suggests the presence of fluid which, however, is not found on exploration; because of adhesions, the fluid changes little on change of posture. On auscultation a friction rub is sometimes heard (Beatty and Bright), chiefly over the liver. (*f*) *Constipation* is usual and results from inflammatory infiltration of the intestine (Stokes); in the puerperal (streptococcic) and in pneumococcic peritonitides, diarrhœa is common.

2 General Symptoms.—(*a*) *Shock* is a conspicuous symptom. Collapse may occur early and directly from perforation or later from toxæmia. The *pulse* is *frequent, hard, wiry* and about 120; later it becomes thready and runs to 140 or 170. The *facies Hippocratica* is pronounced, with its anxious expression, wrinkled, clammy, cyanotic, lead-colored skin, pointed nose, hollowed temples and sunken eyes. The voice whispers (*vox cholerica*) to avoid movement of the diaphragm and the *breathing* is costal, *shallow,* difficult and rapid. (*b*) *Fever* is usually but not always present. It may rise abruptly with a chill at the onset but follows no one type; it may be continu-

ous or remittent and there is usually elevation of temperature by rectum, even during collapse. The fever and pulse curves may run parallel in peritonitis with a slow course. (*c*) The *lungs and heart are crowded* upward by the abdominal distention. The apex-beat is high and further to the left as the heart lies more horizontally. (*d*) The *urine* is scant from lessened absorption and from cardiac weakness; albuminuria is frequent; *indicanuria* is constant from increased putrefaction in the paretic bowel; dysuria is usual; tenesmus, due to involvement of the vesical serosa, is less common than retention.

Course.—Though recovery is possible, the issue is fatal within two days in the *rapidly fatal toxæmic form* in which there is only roughening of the peritoneum without exudation; the slower cases rarely last more than five days. Consciousness is usually preserved to the end, though stupor or delirium occasionally intervenes; the pulse becomes thready, the breathing shallow and sighing, the superficial temperature is low and the patient succumbs to cardiac failure from toxæmia. In exceptional cases death occurs within a few hours or relative recovery results with encapsulation of pus. Treves found inhalation pneumonia in 17 per cent. of his cases and Tilger found serous pleurisy in 25 per cent.; of the latter, 63.3 per cent. were right-sided, 3.3 per cent. left-sided and 33.3 per cent. bilateral.

Types.—(*a*) *Peritonitis serosa* usually follows the fibrinous form; the fluid is yellow or light green, its specific gravity is 1,015 or more, it contains 2 to 3 per cent. of albumin and flocculi of fibrin, epithelial cells, red cells and leukocytes. (*b*) *P. purulenta* may occur in pockets or as a massive pyoperitoneum, amounting to 30 or 40 quarts. (*c*) *P. putrida* occurs in septicopyæmia and puerperal fever, but especially in perforating carcinoma; Obermeyer and Schnitzler have proven that fœtid peritonitis may occur without perforation. (*d*) *P. perforativa* is characterized by two groups of symptoms, (i) those of peritonitis and (ii) those of gas in the peritoneal sac (pneumoperitoneum, pneumatosis peritonei); there is often (but not always) the initial subjective sense of perforation, sudden pain, collapse and retracted abdomen; in four to twelve hours diffuse peritonitis sets in, unless death occurs from the initial shock; free gas may obliterate the dulness of the liver and spleen, though this also results from great tympanites without perforation; the liver dulness may be found only in the posterior axilla, but when the patient is turned on his left side the free gas entirely obscures it; the abdominal contents may be putrid or fæcal, though when due to perforating gastric ulcer (*q. v.*) there is seldom any odor (Czerny). The diaphragm is pushed to its highest possible level; a succussion splash may be elicited and the intestines may be compressed against the spine. It has been held that gas may develop in peritonitis without perforation through the action of the Bacillus pyogenes fœtidus and Bac. aërogenes encapsulatus, but pneumoperitonitis without perforation is rare. (*e*) *P. puerperalis,* usually streptococcic, is described under sepsis on page 127; it is characterized by moderate pain, enormous tympanites, diarrhœa

and the usual septic manifestations. (*f*) *P. pneumococcica* has been described by Sevestre, Moizard, Galliard and Dieulafoy. Jensen has collected 106 cases. Annand and Brown found 91 cases under 15 years of age; one-third were secondary to thoracic lesions; two-thirds probably were enterogenous; of the latter 84 per cent. were in girls. The lower abdomen is especially involved, thereby often simulating appendicitis, which it also resembles in its abrupt onset, pain, fever and vomiting; pneumococcic peritonitis is nearly always attended by early, continuous, fœtid diarrhœa. It often points at the navel, through which a creamy, greenish pus may rupture. Sometimes it ends by crisis; an encysted pus sac often remains. The inflammation is usually fibrinopurulent. The pneumococcus may be found in the blood. (*g*) The *streptococcic, colon* and *staphylococcic peritonitides* are described under appendicitis.

Diagnosis.—Peritonitis is usually diagnosticated with ease from the cause (appendicular, salpingitic, gastric ulcer, septic abortion), by the febrile onset, pain, tenderness, vomiting, distention, effusion and collapse. Puncture with an aspirating needle is dangerous. In certain cases only the fact of peritonitis can be established, perhaps even at operation, though the autopsy usually demonstrates a cause.

Differentiation is necessary (*a*) from intestinal *obstruction* (*q. v.*); (*b*) from the pain of biliary or renal calculi; (*c*) from acute *pancreatitis* (*q. v.*); (*d*) from *ruptured tubal pregnancy,* in which the menstrual history, pelvic localization and symptoms of acute anæmia and shock are present; (*e*) from *ruptured abdominal aneurysm;* or infarction of the superior mesenteric artery in which a cause for embolism, acute obstruction and bloody vomiting or diarrhœa with later peritonitis are often distinctive; (*f*) from acute *enterocolitis,* which is attended by less pain and tenderness and by more diarrhœal colic, tenesmus and collapse due to desiccation of the tissues and to toxæmia; (*g*) from *hysteria* (*q. v.*), which may simulate peritonitis, as it may resemble every other disease; if its presence is once suspected the other stigmata are usually definitive. (*h*) The *referred abdominal pain* of *thoracic disease* (pleurisy and pneumonia) is very seldom accompanied by tenderness, and is easily distinguished by examination of the chest, especially of its posterior parts. (*i*) Mild infections with much exudation may simulate *ascites* (*q. v.*).

Treatment.—*Surgical* treatment alone is of any value. In most statistics the percentage of recoveries is not large; Körte estimates it at 35 per cent., Haenel at 37 per cent. and Krogius (680 cases) at 28.5 per cent. The author has seen, in Murphy's service, 48 out of 57 consecutive cases of diffuse purulent peritonitis recover under operation, the last one being a case of streptococcic peritonitis; Murphy institutes drainage, puts the patient in Fowler's semi-erect posture and administers continuously enemata of normal salt at low pressure.

Palliative treatment is of little value. (*a*) The *opium* treatment only controls pain and though suffering must be relieved by drugs or

operation, the doses recommended by Alonzo Clark (more than 1,000 grains of opium in one week!) are now only of historical interest. Morphine, given hypodermatically, produces the best results. (*b*) The patient should have *absolute rest.* (*c*) Tait's method of *saline purgation* to increase osmosis is productive of more harm than good. (*d*) *Vomiting* is treated as in acute gastritis; the stomach should be washed out; all medication, food and water by mouth should be withheld and given only by rectum. (*e*) Tympanites should be treated by the turpentine stupe and by enemata containing emulsum asafœtidæ 3iij, spts. chloroformi 3j and ol. terebinthinæ 3ss.

CHRONIC DIFFUSE PERITONITIS.

Aside from tuberculous peritonitis, chronic diffuse peritonitis is uncommon. (*a*) The serous or serohemorrhagic form will be considered under ascites; (*b*) the diffuse adhesive form results from acute peritonitis and from tuberculous peritonitis; in this form the peritoneal sac is obliterated and at operation or necropsy the knife cuts directly into the intestine. (*c*) *Chronic proliferative peritonitis* may occur in peritoneal cancer or tuberculosis, chronic alcoholism, cirrhosis of the liver or cardiac disease. It may develop with or without adhesions, as a diffuse sclerosis of the peritoneum and as a subperitoneal proliferation. In its extreme forms it was described by Virchow (1885) as *peritonitis deformans.* The peritoneum is white, lustreless and diffusely thickened, though in some places more than in others. The omentum is retracted upward, so that it lies as a hard, transverse roll above the navel and the mesentery is indurated (mesenteritis retrahens), as described under tuberculous peritonitis. As the mesentery retracts it may gather the intestinal coils into a ball the size of a child's head, lying centrally or to the right of the median line. Here and there may be found multilocular serous encapsulations or there may be no serous exudation when the case comes under observation. There may be palpable tumor-like thickenings. The spleen, liver, stomach, cæcum or colon may be particularly indurated. In some cases it is but part of a multiple serositis (polyserositis, polyorrhomenitis), invading also the pleura and pericardium. Miliary fibromata, resembling miliary carcinosis or miliary tuberculosis, may sometimes form around parasitic ova or cholesterin crystals.

LOCALIZED PERITONITIS.

I. Suppurative Forms.—1. Subphrenic Abscess and Pyopneumothorax Subphrenicus.—Subphrenic suppuration was first diagnosticated by Barlow (1845) and was especially described by Leyden (1879). Maydl's monograph (1894) comprised 179 cases, upon which the following figures are based; sixty cases are described by Körte (1903).

Etiology.—(*a*) *Gastric ulcer* causes 19 per cent.; gastric cancer is

a less frequent cause. (*b*) *Appendicitis* causes 14 per cent.; (*c*) *trauma,* 14 per cent.; (*d*) *intestinal ulceration,* 7 per cent.; (*e*) cholangitis and cholecystitis, 11 per cent.; echinococcus, 7 per cent.; liver abscess, 10 per cent.; (*f*) *metastatic* abscess, 6 per cent.; (*g*) *perinephric* abscess, 6 per cent.; (*h*) occasionally, ulcerative lung lesions, 5 per cent.; (*i*) splenic infarct, abscess or echinococcus, diseases of the spine, ribs, pericardium or uterus are uncommon causes.

Symptoms.—Pus is ensacculated beneath the diaphragm (pyothorax subphrenicus), associated in 50 per cent. with gas formation (pyopneumothorax subphrenicus). The *subphrenic abscess* is usually right-sided, is limited by the suspensory ligament of the liver and may be intra- or extraperitoneal; in the latter instance the abscess is most often appendicular or perinephric and lies well back in the abdomen. Cases with simple pus formation are often difficult to localize; their symptoms are those of sepsis, with pain, tenderness or dulness on the liver convexity, which suggest liver abscess. The retroperitoneal forms give most dulness posteriorly, but in intraperitoneal forms it is chiefly located anteriorly; œdema of the chest wall may occur without suppuration; the *x*-rays may produce a subphrenic shadow.

In *pyopneumothorax subphrenicus* there are signs "of a cavity beneath the diaphragm, filled with gas and pus, pushing the diaphragm upward and simulating pyopneumothorax" (Leyden). (*a*) *Tympany* and *absence of vesicular murmur* and of vocal *fremitus* are found from the third rib downward, above which is the compressed lung and below which is (*b*) the *dulness of the liver* whose exposed surface and edge, dislocated downward, are easily percussed and palpated. Sometimes the tympany entirely covers the liver dulness. As in pneumothorax (*c*) *succussion* and, on change of posture, the *clearly shifting line of dulness* may be elicited. (*d*) *Puncture* evacuates gas and putrid pus; sometimes on a higher puncture serum is found in the pleura, for pleurisy develops in 66 per cent. of the cases. The needle ascends during inspiration and descends during expiration, which is the converse of the findings in pleural exudation (Fürbringer). If a manometer is attached to the exploring needle, a rise in the indicating column during inspiration and a fall during expiration denote a subphrenic lesion (Pfuhl). (*e*) *The x-rays* may show movement of the diaphragm and a shadow between it and the liver; in three cases the writer found no respiratory excursion. (*f*) *Perforation* into the lungs occurs in 16 per cent. (more often in extra- than in intraperitoneal forms), into the pleura in 19 per cent. and into the pericardium in 1 per cent. At the beginning and until perforation there is generally an entire absence of such thoracic symptoms as cough or sputum; this is of value in differentiation from true pyopneumothorax. (*g*) The *history* of the subphrenic pyopneumothorax is usually that of some *abdominal lesion* (*v.* ETIOLOGY).

Treatment is surgical. Körte's experience with 60 operations gives 66 per cent. of recoveries.

2. Suppuration in the Lesser Peritoneum.—This may result from

gastric or duodenal ulceration, acute pancreatitis, etc. Closure of the foramen of Winslow confines the pus; a tumor appears in the epigastric, umbilical or left hypochondriac region, bounded above by the stomach, which also in part covers it, below by the colon and behind by the pancreas and retroperitoneal tissues. Its relations are like those of pancreatic cyst; variability in its size occurs when the stomach is filled with gas and fluid. When gas and pus co-exist the diaphragm and liver are pushed upward. Treatment is surgical.

3. Other Forms.—The *appendicular abscess* has been described. The *pelvic abscess* occurs especially from tubal, but also from uterine and ovarian inflammation; it follows abortion, puerperal fever, gonorrhœa, tuberculous and pneumococcic peritonitis in young girls. Other types are suppuration around the gall-bladder, colon and sigmoid and in other rare localizations.

II. Adhesive or Indurative Forms.—Local thickening or adhesions may occur over any abdominal viscus but especially over the spleen, next over the liver and less commonly over the intestines; it may bind together two coils or form bands under which loops may become strangulated.

Perisplenitis is most common over chronic malarial, leukæmic and pseudoleukæmic enlargements of the spleen and less frequent over acute splenic tumors.

Perihepatitis may cover small areas of the liver like soldier's spots in the pericardium or may engross its entire surface. General perihepatitis was described by Budd (1852), by Curschmann (1884), who called it the "icing liver" (*Zuckergussleber*), and by Fagge, Hale White, Nichols, A. O. J. Kelly and others. It is frequently associated with deforming proliferative peritonitis. Nichols describes it as a hyaline degeneration, hyaloserositis. There are three groups of cases; (*a*) *multiple serositis* (polyorrhomenitis, Concato's disease); this borders closely on Pick's "pseudocirrhosis pericarditica." Kelly has assembled 27 cases in which perihepatitis was associated with adhesive pericarditis. (*b*) *Arteriosclerosis* and *interstitial nephritis* occurred in 86 per cent. in Hale White's series. (*c*) Rarer factors are syphilis, tuberculosis, malignancy and alcoholism. Its symptoms may be those of cirrhosis; it will be differentiated under ascites (*v. i.*).

III. Chronic Hemorrhagic Peritonitis.—This rare disease was described first by Virchow, who likened it to pachymeningitis hemorrhagica. It occurs chiefly in the pelvis and is most often circumscribed. Pachyperitonitis is characterized by hemorrhagic inflammation; from the laminæ of the resulting fresh connective tissue, deposited layer by layer, repeated fresh hemorrhages arise.

CARCINOMA OF THE PERITONEUM.

Primary malignant growths of the peritoneum are *endotheliomata*, and are very rare.

Cancer is most frequently secondary to cancer of the stomach, ovaries, intestines, pancreas and liver. Histologically it is that of the primary growth. Its gross forms are (*a*) *miliary carcinosis,* resembling miliary tubercles; (*b*) *vegetative;* (*c*) *ulcerative;* (*d*) *cystic* and (*e*) *infiltrative.* The primary tumor may be quite latent clinically, so that the peritoneal complication is apparently primary. The secondary growths spread by contact, the lymphatics and implantation of free particles. They may be felt in the omentum, mesentery and Douglas' *cul de sac* and in some cases may be confused with tuberculous masses. The diagnosis is usually reached by the symptoms and signs of the initial growth or by those of an ascites, which is serous, hemorrhagic, pseudochylous or adipose; its progress is rapid and attended by cachexia. Groups of cancer cells may be found in the aspirated fluid and secondary cancer may develop at the point of puncture. Differential features are tabulated under the next topic.

ASCITES.

Ascites (hydroperitoneum) is a symptom and sometimes the chief symptom of various diseases.

Etiology.—(*a*) It may result from *stasis,* of which the chief varieties are valvular and other *cardiac* lesions, pulmonary, mediastinal or pleural disease and *portal* stasis, due to disease of the liver or portal vein. Stasis causes increased pressure in the portal system, followed by malnutrition of the vessels and pouring out of serum. (*b*) *Hydræmia or cachexia* causes malnutrition and increased permeability of the vessel walls; it occurs in amyloidosis, chronic nephritis, marantic conditions, profound anæmias and occasionally in acute affections. (*c*) *Inflammation,* as simple, tuberculous, suppurative or cancerous peritonitis. (*d*) *Abdominal* tumors, leukæmic spleen, etc. In many cases more than one factor may operate; tuberculous peritonitis may complicate liver cirrhosis or ascites may be due to the hydræmia of nephritis, accentuated by weakness of the heart.

Symptoms.—1. Inspection.—A *symmetrical enlargement* of the abdomen in the average case presents rather more lateral than anterior bulging. The abdomen resembles that of a batrachian (*ventre de batracien*). The skin is pale, tense, striated, œdematous; the *pouting navel* and diastasis of the recti muscles are noted. A *caput Medusæ* may be noted in cirrhosis cases. Dilated veins over the ribs and upper abdomen indicate compression of the cava inferior. Sometimes the heart imparts systolic waves to the fluid.

2. Palpation.—Fluctuation (*ballottement*), due to the transmission of a fluid wave from side to side, is usually present but may be absent because of tense abdominal walls or great accumulation of fluid. Pseudofluctuation may be caused by accumulation of fluid other than ascitic, as intestinal contents, or by lax, obese abdominal walls; in the latter condition a third hand placed in the median line

of the abdomen will break the deceptive wave, which is due to superficial or parietal vibration. Fluctuation is felt in ascites even where great tympany exists. In women the *water-pillow* fluctuation (Landau) may be elicited *by vaginal examination* and Tripier remarks that the uterus seems remarkably movable. *Thrusting palpation* may disclose the outline of the liver, spleen or possibly some neoplasm even through the abundant fluid; it is performed by placing the fingers vertically on the abdomen and suddenly thrusting them inward, thus anticipating any rigidity of the muscles and pushing aside the ascitic fluid.

3. Percussion.—At least a pint to a quart (500 to 1,000 c.c.) of fluid must be present for clinical detection. With small effusions the patient should be examined in the genupectoral position (Leube). Elevation of the buttocks causes scanty fluid to gravitate to the flanks where its presence is more easily demonstrated. Classically, the *fluid* in dependent parts *gives dulness* and the *supernatant intestines* are located by *tympany* in the highest parts of the abdomen. These areas *vary with change of position,* always giving, in uncomplicated cases, *tympany above* and *dulness below,* in any posture the patient may assume. When the patient lies upon his back the tympanitic area is central and elliptical. This rule is void when the mesentery is retracted and does not allow the gut to float, in which event there is dulness over the highest areas on light percussion, though tympany can be elicited on deep percussion over the same area; adhesions between the intestinal loops prevent shifting of the fluid; two lines of tympany in the flanks mark the ascending and descending colon; when the gut is adherent anteriorly there is permanent tympany in that location and the author knows of an instance of ascites in which a physician perforated the intestines; immovable tympany in the flanks in ascites may be due to extensive adhesions of intestinal coils to the lateral abdominal parietes. Light percussion is employed in the determination of ascites, because heavy tapping elicits tympany from the adjacent or more deeply-situated intestine.

A large quantity of free fluid may, in very rare instances, exist without dulness. The author withdrew two gallons of ascitic fluid from a patient in whom no dulness could be elicited in any position.

Œdema of the subcutaneous tissues renders percussion of the abdomen difficult, as do fatty abdominal walls, fatty omentum and fatty mesentery. The manometric pressure in the abdomen amounts to 6 to 40 mm. of mercury.

Diagnosis.—Although ascites is usually one of the most easily demonstrable conditions, excellent clinicians have mistaken it for other affections. *Fæcal accumulations* cause dulness in either flank, simulating fluid, but the history of the case, the immobility of the dulness and the use of enemata or catharsis preclude error. Large quantities of *semifluid fæces* in the ascending and descending colon may closely resemble free fluid; on thrusting palpation a

splashing sound may be elicited over the colon and Küssner published a case in which fluid in the intestines was mistaken for ascites. Leube confused ascites with an enormously *dilated stomach.* Preperitoneal lipoma, hydronephrosis, hydatid cyst, pregnancy, dilated bladder, ovarian cyst and tympanites are frequently sources of error.

Rostan (1837) noted that tympany might be absent in ascites when the intestines contained little or no gas, a point sometimes forgotten in differentiation between ascites and *ovarian cyst;* the latter may thrust itself in between the coils of gut and simulate ascites; ovarian cyst usually produces a central flatness with tympany in the flanks, and neither the dulness nor tympany shifts with change of posture. Adami has collected 42 cases of fatty tumor (*fibrolipoma* and *myxolipoma*) in which ascites was very closely simulated.

Etiological Diagnosis.—1. Stasis.—In *cardiac disease* the dyspnœa develops early, but that caused by ascites pressing upward on the diaphragm is a late symptom; swelling of the feet usually antedates the ascites, though Strümpell (1883) pointed out that pericarditis and valvular disease may exceptionally produce ascites without anasarca. It must be borne in mind that a marked ascites may crowd the heart upward and outward. The *liver* is peculiarly an etiological factor in ascites (see Differential Chart on page 695). Portal ascites commences almost invariably in the peritoneum; the legs swell secondarily from pressure on the vena cava inferior or from cardiac weakness due to great ascites (Giovanni); the legs may swell disproportionately, cicatrices may involve the portal vein and cava simultaneously at the notch in the posterior surface of liver, and finally the legs may swell first. In some cases the ascitic fluid may lie between the diaphragm and the liver, which latter is then pushed downward and may appear large, though it is merely dislocated. An attempt should be made to exclude *perihepatitis,* which, according to Fagge, causes one death to every five fatal cases of cirrhosis, and is more apt to be attended by albuminuria (see Table given below).

2. Hydræmia. (Cachexia, Increased Vascular Permeability).—In renal disease the eyelids often become œdematous before other symptoms develop and the nephritic ascites is seldom great unless the liver and heart are involved. Cachectic ascites is not uncommon in leukæmia and kindred affections. Slight serous effusions occurring in acute infectious diseases in children may come under this title. The author has seen three instances of ascites in typhoid convalescence.

3. Peritonitis.—The specific gravity and the percentage of albumin in the fluid are important factors in differentiating between exudate and transudate. Three divisions are distinguished: (*a*) a specific gravity of 1,010 or lower indicates a *cachectic transudate, e. g.,* in nephritis, with less than 1 per cent. albumin. A very low specific gravity and percentage of albumin occur in amyloidosis. (*b*) A *hypostatic transudate* has a specific gravity which ranges be-

tween 1,010 and 1,014 and has between 1 and 3 per cent. of albumin. (*c*) An *exudate* has a specific gravity of more than 1,015 (or 1,018) and the albumin reaches 4 to 6 per cent. The lower strata are heavier and the specific gravity should be measured several times during the paracentesis. When there is less than 1 per cent. of albumin, no disease of the peritoneum or portal vein exists. Reuss' formula to compute the albumin percentage from the specific gravity is subject to an error of less than ¼ of 1 per cent.; the percentage of albumin = ⅜ (specific gravity minus 1,000) minus 2.8. A specific gravity of 1,017 to 1,020 positively indicates an exudate. There are certain exceptions to this rule, and certain confusions may arise; thus an ascites due to a carcinoma in the liver substance has a low specific gravity and albumin is present to 1 or 2 per cent. If a carcinomatous peritonitis develops, the specific gravity and percentage of albumin increase. An ascites due to nephritis has a low specific gravity but the figures rise when stasis due to weak heart or intercurrent inflammation develops. In many cases of simple transudation (ascites and hydrothorax), the characters of an exudate may develop, due to the long-continued irritation of the serosa.

Ascitic fluid always contains the constituents of the blood—albumin, globulin, urea, uric acid, sugar, etc. (*a*) Ascitic fluid due to stasis is clear yellow or yellow-green, opalescent and alkaline and microscopically contains some lymph cells, erythrocytes and peritoneal endothelium. (*b*) Exudates show a microscopic picture varying with the cause; fibrin threads, pus, blood, adipose, chylous or chyliform fluid, intestinal contents, cancer cells, tubercle bacilli, diplococci or pyogenic organisms, may be found. In exudates are found the polymorphonuclear leukocytes and some erythrocytes; lymphocytes and red cells are detected in tuberculous exudates (see page 532); the fluid must be examined at once or formalin should be added for the cells readily degenerate and shrink. Rieder, Dock and others describe cells in carcinomatous peritonitis in which asymmetrical karyokinetic figures appeared. Quincke states that red disks, lymph cells and peritoneal endothelium are found in every ascites.

Primavera differentiated between transudates and exudates by means of glacial acetic acid; a single drop will leave a white cloud as it falls to the bottom of a vessel containing an exudate, while its addition to transudates gives no reaction.

"Essential" ascites is usually tuberculous peritonitis.

Differentiation Between Serous and Tuberculous Peritonitis.—Chronic serous peritonitis is generally tuberculous. In tuberculous peritonitis (*q. v.*) there are usually temperature and emaciation and bacilli in the exudate. According to Tepret and Vierordt, chronic nontuberculous peritonitis occurs especially in women, the causes of which are Bright's disease, trauma, portal stasis, alcoholism and heart affections. The ascitic forms of nontuberculous peritonitis may pass into the membranous or adhesive variety (peritonitis de-

formans or pachyperitonitis). The diagnosis of simple nontuberculous peritonitis is most difficult to establish, even at autopsy.

Differentiation Between Tuberculous Peritonitis and Ascites.—Portal ascites is accompanied by other circulatory disturbances, as splenic tumor, piles and caput Medusæ. The spleen is rarely enlarged in tuberculous peritonitis. The specific gravity and percentage of albumin are almost final in determining between peritonitis and ascites. The fluid is generally clear in ascites and cloudy in peritonitis. Pain and fever usually indicate encapsulation. The liver is small in the terminal stage of atrophic cirrhosis, which is marked by various urinary changes; these are evidences of incomplete discharge of the hepatic function. In tuberculous peritonitis, the *hydrops ascites saccatus* gives an irregular form to the abdomen from meteorism produced by adhesions and from irregular nodules sometimes simulating carcinoma; it was described by Hippocrates. Free tuberculous exudations differ according to Thomayer, from simple ascites in that the tympany in tuberculous forms is often right-sided, due to retraction of the mesentery (mesenteritis retrahens) to the right side, carrying with it the small intestine. Mesenteritis retrahens is usually tuberculous or carcinomatous. The author recalls instances in which right-sided tuberculous masses pushed to the left both the tympany and the fluid. Hemorrhagic ascitic fluid occurs frequently in tuberculous peritonitis and rarely in cirrhotic transudate.

The "apparent tumors" of tuberculous peritonitis (Bamberger) are always suggestive where the nature of an ascites is in question. In two instances observed personally, the omentum was so retracted and adherent to the normal liver as to resemble closely the edge of a cirrhotic liver. Intestinal or pulmonary tuberculosis, adhesive pleuritis and meteorism from intestinal adhesions or an irregular form of the abdomen with a "doughy" feel on palpation, indicate tuberculous peritonitis. A good working rule is that a peritoneal exudate is tuberculous, when cancer, sarcoma and trauma can be excluded. Exploratory incision is often the only direct route to diagnosis and appropriate therapy. In the case of Heidenrich, in which the fluid was encapsulated, each of about 20 separate sacs was filled with fluid differing from that found in the others; in one was clear serum; in another paler fluid; in others, the effusion was brown, purulent, thick or sanguinolent.

Nichols gives a differential table, presented on the following page.

4. Tumors.—Tumors, other than peritoneal, as uterine fibromata, may produce hydroperitoneum. There is no relation between the size of the tumor and the amount of fluid transuded, because very small uterine myofibromata may excite considerable peritoneal effusion. There is, nevertheless, a direct relation between the malignancy of the tumor and the amount of the fluid found (Terillon). As a general statement, ascites is infrequent in uterine tumors (Cruveilhier) but when observed is most common in adenomyoma (Freund). It is more often seen in papillomatous cysts; in glandular cysts whose

NICHOLS' TABLE.

	"ZUCKERGUSS-LEBER"; CHRONIC PERIHEPATITIS.	ATROPHIC CIRRHOSIS OF LIVER.	CHRONIC TUBERCULOUS PERITONITIS.	CARCINOMA OF PERITONEUM.
Age.	Occurs about middle life or later.	Oftenest about middle life.	Commonest between ages of 20 and 40.	Occurs late in life.
Sex.	Both sexes equally liable.	More frequent in males.	Predominates in females.	More frequent in females.
Previous history.	Often a history of acute pericarditis or perihepatitis.	History of alcoholism, syphilis or digestive disturbances.	Often a chronic cough, diarrhœa, or genital tuberculosis.	In some cases a history of cancer of stomach or ovaries.
Alcoholism.	No influence.	Frequently present.	Unimportant.	Unimportant.
Syphilis.	No influence.	Occasionally.	Unimportant.	Unimportant.
Heredity.	No influence.	Unimportant.	May be family taint.	Unimportant.
Incidence.	Acute becoming chronic or insidious from first.	Insidious.	Onset may be acute or insidious.	Insidious.
Chronicity.	Cases last for 2–10–16 years.	May last for years.	Prolonged.	Fairly rapid course.
Fever.	Generally absent except during exacerbation or some complication.	May be afebrile; when present is slight.	Usually slight; often absent.	Rarely absent; due to complication.
Pain.	Indefinite and trifling.	Trifling.	Apt to be troublesome.	Variable.
Digestive disturbance.	Trifling or none.	Constant; dyspepsia, nausea, vomiting, gastric hemorrhage, melæna.	Fairly common.	Often marked.
Ascites.	Constant and extreme; nutrition long maintained—Rumpf's case tapped 301 times; may resemble peritonitis, albumin 3 per cent., fibrin.	Constant.	Never extreme, may be absent, may be hemorrhagic.	Moderate grade; may be hemorrhagic or pseudochylous.
Anasarca.	Constant but slight.	Relatively slight.	Trifling.	Slight.
Jaundice.	Absent in pure cases.	Occurs in 27 per cent. of cases.	May occur exceptionally.	Common when liver is enlarged.
Liver.	Not cirrhotic; at first enlarged, then small; smooth.	Cirrhotic; at first enlarged, then small and warty.	Often enlarged.	May be enlarged, with nodules.
Spleen.	Gradual enlargement.	Gradual enlargement.	Nothing special.	Nothing special.
Omentum.	Thickened and contracted.	Normal.	Often matted up.	Often matted up.

walls rupture by fatty degeneration and pour out their contents into the abdominal cavity; serum is secreted by the peritoneal surface because of the irritation, even though the ruptured cyst be very small (Quénu). The fluid in cysts contains more solids (50 to 60 pro mille) than does the ascites of renal disease (25 pro mille, Méhu). Ascites is most often observed in solid ovarian tumors.

Treatment.—The treatment of ascites varies with its cause, for which reference should be made to valvular heart disease, atrophic cirrhosis, tuberculous peritonitis and nephritis.

Paracentesis in peritoneal carcinoma and chylous ascites (*v.i.*) hastens the fatal issue. Hale White asserts that cases of cirrhosis sur-

vive but few punctures, while cases of perihepatitis may be punctured more than 200 times. Lécanu reports a case of ascites in which 886 tappings were performed in fifteen years. In puncture, surgical antisepsis is most necessary, for reduced subjects are easily infected. The bladder should be emptied by catheterization. The trochar should be introduced in the *linea alba* to avoid injury to the blood-vessels. It should be pushed carefully and is felt to penetrate the parietal peritoneum by the sense of something giving away. A dull-pointed probe should be sterilized, with which to push back the omentum if it should prolapse against the canula. The fluid should be evacuated slowly to obviate collapse which may result from dilatation of the abdominal veins, if they are suddenly relieved of the pressure of the effusion. A cat-o'-nine-tails bandage may be applied as the tapping progresses, to compress the abdominal contents.

Chylous and Adipose Ascites.—The peritoneal cavity, more frequently than any other serous sac, is the seat of those unusual exudates known as chylous, chyliform, lactiform or adipose ascites. The first authentic case is Poncy's (1699). During the last two centuries the literature gives about 200 cases. Seven have come under the author's observation.

Much confusion has arisen from inaccurate nomenclature. *Chylous ascites* properly designates an effusion of chyle. *Adipose ascites* contains a large percentage of fat with no chylous admixture. Some authors use the terms "adipose," and "chyliform" interchangeably. Chyliform ascites is a chyle-like fluid in which lymph or chyle is mixed with exudates or transudates.

1. Chylous Ascites.—Straus' case is the clearest example, in which typical chyle was extravasated through two fistulæ. Ingested butter was recognized in the fluid withdrawn by paracentesis. Senator's latest case gave similar results with the administration of olive oil. Chylous ascites contains sugar, which is said to be diagnostic when diabetes is excluded. Sugar when primarily present may disappear later (Whitla). Its presence is very rare and very suggestive; it has been detected not more than two dozen times. Of late many clinicians discount the importance of the presence of sugar as a diagnostic test; Bock found sugar (0.04 to 0.07 per cent.) in all cases of hydrops, and Eichhorst in ten out of seventeen cases of pleural exudate. A small amount of fat (0.9 per cent.) indicates chyle. Chylous ascites is rich in solids, mineral salts and albumin. Albumin and fat occur in small punctiform granules, susceptible of chemical differentiation. The escape of chyle may occur through a visible rupture or by transudation through the walls of the chyle vessels; among the most frequent causes of lymph or chyle obstruction are compression of the thoracic duct or lymph system by glands, neoplasms, peritonitis, occlusion of left subclavian vein, pressure efforts, lifting or coughing, filarial disease or occlusion of the thoracic duct. Rupture may occur in any part of the lymphatic system, in the thoracic duct, receptaculum, lacteal vessels, lymph glands and chylous cysts. Chyle

may extravasate into a serous effusion by mural alteration of the lymphatic vessels.

2. Adipose or Chyliform Ascites.—Adipose ascites is characterized by the absence of sugar and a higher percentage of fat. The degree of opacity is no index of the amount of fat, because turbidity may be due to emulsionized albumin. Fat is found in most cases; its greatest amount is 5.25 per cent. (Schmidt), and in one of the author's cases 6.5 per cent. The granules of albumin and fat are much coarser in adipose than in chylous ascites. Hydropic and fatty carcinoma cells may, if numerous, cause a creamy layer. Red blood disks may occur, also fibrin, casein, mucin, sodium alkali albuminate, peptone, lecithin, cholesterin, fibrinogen, etc. Among the numerous etiological conditions are tuberculosis of the peritoneum and glands; carcinoma of the glands, peritoneum and lymph vessels; chronic peritonitis; liver cirrhosis, heart disease and sarcoma of the omentum or mesentery.

Diagnosis.—A diagnosis has never been made prior to puncture, except in Morton's early case. The local signs and symptoms do not differ from those of serous ascites. It is probable that some cases diagnosed as the vulgar ascites, healing without treatment, are instances of chylous hydrops. It is not easy to differentiate between chylous and adipose effusions, even at necropsy.

Prognosis.—About 90 per cent. of both types die. Continuous chylous fistulæ are always fatal. The immediate prognosis is seemingly more favorable in the adipose form.

Treatment.—Surgical interference is chiefly indicated in tuberculous peritonitis. Paracentesis should be avoided as much as possible for it is weakening and precipitates the fatal issue.

SECTION V.

DISEASES OF THE KIDNEY.

ACUTE NEPHRITIS.

Etiology.—(*a*) Acute nephritis may result from *infections.* The *scarlatinal* nephritis (*q.v.*) is the most frequent type and develops during the third week after desquamation. It occurs in diphtheria, at the height of the disease; it is found in a large percentage of cases of acute enterocolitis in children; it may result from malaria, variola, sepsis, ulcerative endocarditis or pneumonia; there is scarcely an infection in which acute nephritis may not occasionally develop, as typhoid, typhus, measles, tonsillitis or epidemic meningitis; it is rare in German measles, varicella or purpura. It may develop in tuberculosis and syphilis. Infections operate (i) more often by their toxins than (ii) by the causal microbe, though the typhoid bacillus, pneumococcus, streptococcus and others, have been found in the kidneys and urine; (iii) in some instances (nephritis hæmoglobinurica) hæmolysins apparently cause acute nephritis. (*b*) *Toxic substances,* taken internally or applied to the skin, may induce acute nephritis, as turpentine, salicylic compounds, carbolic acid, potassium chlorate, cantharides and kindred substances. (*c*) Exposure to *cold and dampness* (with alcoholism) is frequently the apparent cause; possibly it lessens the tissue resistance or acts as a hæmolysin, as in the hæmoglobinuric type. (*d*) *Skin diseases,* burns, eczema and psoriasis, may lessen the skin functions or produce hæmolysins. (*e*) *Pregnancy* (*v. i.*) may result in nephritis. (*f*) The cause is often *obscure;* chronic Bright's disease, cardiac affections and the dyscrasiæ predispose to acute nephritis. (*g*) Acute nephritis may occur independently of any known cause; this primary form may be epidemic.

Pathology.—*Gross changes* may not be apparent in mild cases, though in some the kidneys are swollen from marked interstitial exudation and are dark and heavy. In other cases the kidney is yellow-white and perhaps mottled from small hemorrhages. The œdematous capsule strips readily and the cut section drips blood. On section the cortex is swollen and granular, and its striations are blurred and pale in contrast with the dusky medulla; the glomeruli are frequently visible as red or later as yellow dots. Without the microscope, lesser degrees of acute nephritis may be confused with fatty or parenchymatous degeneration. *Microscopic changes* are usually diffuse. (a) *Glomerular* changes. Glomerulonephritis, espe-

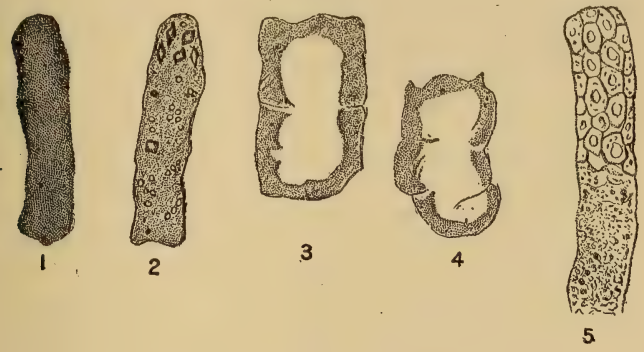
cially described by Klebs, is caused by toxins reaching the tufts through the blood current. It is best exemplified by the scarlatinal form (*q.v.*). The glomerular capillaries show infiltration with leukocytes, an albuminous or cellular exudate escapes into the capsule and the epithelium of Bowman's capsule degenerates, desquamates and later proliferates; in some cases red disks escape with the serum and leukocytes. (*b*) *Tubular* changes. Cloudy swelling, hyaline and fatty degeneration occurring especially in the cortex may be difficult to differentiate from febrile and other degenerations, though interstitial exudation occurs in every case of nephritis. In severe cases some cells necrose entirely and desquamate; the diphtheritic form is the best type. The swelling around the convoluted tubules is one cause of the kidney enlargement. (*c*) *Vascular* and *interstitial* changes are essential to the pathological diagnosis and are more focal than diffuse. Serum exudes into the connective tissue, tufts and tubules and coagulates in them; it is probably the cause of the casts; leukocytes escape into the parenchyma and interstitium and in some cases red cells also (hemorrhagic nephritis). Councilman describes Unna's plasma cells, which he thinks are conveyed to the kidney from the spleen and marrow.

Symptoms.—The *onset* may be *sudden,* especially in cases following exposure. In other cases it develops *gradually* in the course or convalescence of the causal infection, as scarlatina, diphtheria, etc. In many cases it may be unrecognized unless frequent analyses of the urine are made or until uræmic symptoms develop.

1. Urinary Findings.—(*a*) The *urine is decreased* to ten or twelve ounces or in severe cases is *suppressed;* this is due to glomerulitis or stoppage of the tubules by coagulated albumin and desquamated epithelium; the decrease usually runs parallel with the intensity of inflammation and hydrops. During convalescence the urine becomes abundant and clear. (*b*) Its *color* may range from that of febrile urine to a *dark* smoky tinge; it is seldom red or actually bloody; in the hæmoglobinuric form it is chocolate-colored. (*c*) The specific gravity averages 1,025 to 1,030 and (*d*) the reaction is *acid.* (*e*) *Albuminuria* is almost invariable; the albumin amounts to ⅓ to 1 per cent. and one to two drams (5 to 8 gm.) daily. There is serum albumin, considerable quantities of globulin and if there are many cells, as in tubular nephritis, nucleo-albumin. The author has seen six fatal cases of acute diffuse nephritis, confirmed by autopsy, in which there was no albuminuria. Similar cases are reported by Quirolo, Henoch, Senator, Fermi, Sanné and others. (*f*) *Urea* is *decreased* even to ⅙ its normal quantity, as are also the *chlorides* and phosphates; the uric acid remains normal and the xanthin (alloxur) bases may be increased; for the molecular constitution of the blood and cryoscopy, see page 749. (*g*) The *sediment* in mild acute tubular nephritis consists of many *epithelial cells,* some red and *white corpuscles* and hyaline *casts* with crystals of oxalate of lime and uric acid and sometimes hæmoglobin granules, which may be also

found as free granules. In diffuse glomerular nephritis there are almost always *red cells,* mono- and polynuclear leukocytes, epithelium, fatty, hyaline and granular casts, granules of fat and often of hæmoglobin, uric acid and calcium oxalate crystals. Senator

FIG. 45.



Casts. 1–4, amyloid; 5, cast, epithelial in its upper, and granular in its lower, part.

maintains that the leukocytes from the kidney are mononuclear. Waxy and epithelial casts are found only in the severest types. Blood and casts may appear before and may also outlast the albuminuria. The significance of casts (cylindruria) will be considered under chronic parenchymatous nephritis.

2. HYDROPS (ANASARCA).—This is often the first symptom to attract attention. (*a*) It occurs *about the eyes* early in the morning

FIG. 46.

Urinary casts. 1, hyaline, with fat droplets and cells; 2, hyaline, with leukocytes; 3, cast with fat droplets.

and later in the day in other parts, as the limbs and external genitalia. As the process advances it may become general. (*b*) It changes its location readily and with *anæmia* gives a rather *characteristic facies.* (*c*) Anasarca occurs most commonly in cases due to *scarlatina and exposure,* less commonly in pregnancy, malaria, alcoholism and skin

diseases and seldom in sepsis, pneumonia, typhoid or diphtheria. (*d*) It is caused by *inflammation* of the *skin vessels,* which is identical with the inflammation in the glomerular vessels and is nearly always associated with glomerulitis. (*e*) Identical changes may occur in the *serous sacs* (pleura, pericardium or, far less often, peritoneum, meninges and joints), *mucous membranes* (glottis or intestinal tract) or viscera (pulmonary or cerebral œdema). (*f*) It parallels the intensity of the nephritis and the decrease of urine.

3. Anæmia.—Anæmia is often an early symptom and with the dropsy is suggestive of acute nephritis. The red cells and hæmoglobin are reduced, the specific gravity of the serum is lowered and the freezing point is raised (*v.* Chronic Nephritis).

4. Cardio-Vascular Symptoms.—The right heart may become dilated. Hypertrophy of the left ventricle is very uncommon, though observed by Riegel and Leube within ten days of the beginning of the disease. The pulse is usually slow and tense; the sphygmogram shows increase of the tidal and decrease of the dicrotic wave and the sphygmomanometer shows the blood tension increased even to 180 or 200 mm.; these phenomena are probably due to vascular contraction induced by metabolic products retained in the blood. The second aortic tone may become accentuated and, if heart fatigue develops, the gallop-rhythm is heard.

5. Temperature.—Fever and rigors are exceptional.

6. Pain.—Occasionally there may be pain and tenderness in the back, frequent and painful urination, emaciation and epistaxis. Constipation is the rule. The skin is generally dry.

Diagnosis.—The urinary findings are characteristic; *albuminuria* (*q. v.*) may result from other causes, as fever, amyloidosis or stasis; casts, leukocytes, epithelia and even red cells, may also occur in chronic nephritis, but the history and evolution determine the diagnosis (*v. i.* Differential Table of Diseases of the Kidney); cases without urinary findings are extremely rare. The greatest danger lies in the diagnosis of an acute nephritis when the condition is only an *acute exacerbation of a chronic process;* fully half of the so-called idiopathic acute cases which the author has seen belong to this category. In *renal hæmaturia* the urine contains no casts (save perhaps red cell casts), few leukocytes and practically no epithelia; there is no anasarca; the urine is clear at one time and is laden with blood at another. *Anasarca* (*v. s.*) occurs in certain types only and also in various cachectic states.

Diagnosis of Types.—(*a*) The *kidney of pregnancy* usually develops in the last half of gestation and infrequently before the third month. Its pathogenesis is vigorously disputed, though toxæmia due to the double task imposed upon the maternal kidneys seems the most probable cause. It has been suggested that decreased antitoxic activity of the thyroid gland is a causal factor. The placenta is thought by some to elaborate toxins (syncytiolysins). The urine is decreased, pale and of low specific gravity; it contains much albumin and not

much sediment, though red and white cells, lymphocytes, fatty epithelia and sometimes casts are found. The attendant anasarca, like that of stasis, develops from below upward. When retinitis develops its prognosis is more favorable than in other nephritides. The convulsive seizures (eclampsia) are generally uræmic, though Ingerslev collected 106 cases in which there was neither clinical albuminuria nor renal alteration at necropsy. Fehling remarks on the slight anatomical changes in the kidney and on the fact that but 5 per cent. of pregnant patients having an old nephritis develop eclampsia. The exact explanation of the convulsions remains obscure, though it seems that the brain centres during gestation are unduly susceptible to irritation. The embolism of placental cells in the brain or lungs (Schmorl) is considered by some rather as a result than a cause of eclampsia. Zweifel maintains that the oxidation of the albumins is decreased, the nitrogen output is decreased, the ammonia (as in diabetic coma) is increased and that sarcolactic acid is found in the blood and urine. The maternal mortality is 25 to 30 per cent. and that of the child over 50 per cent. Very few cases develop chronic nephritis. (*b*) The *cholera nephritis* is parenchymatous and chiefly tubular. Various factors are operative, as toxæmia, arterial anæmia, venous hyperæmia and heart weakness. By some it is considered renal ischæmia rather than nephritis. The urea, chlorides, calcium and magnesium salts are decreased and much indican, aromatic sulphates, acetone and ammonia are found. The anuria may last three or even fifteen days. (*c*) *Hæmoglobinuric nephritis* is probably due to hæmolysins, which disorganize the blood and irritate the kidneys by the hæmoglobin thus set free. It is found in burns, acute infections (typhoid, pernicious malaria, scarlatina, yellow fever, Winckel's disease of the new-born), exposure, hæmoglobinæmia and various drug poisonings, notably that of chlorate of potash. Hæmoglobinuria, red disks in the urine, casts, leukocytes, epithelial cells and abundant albuminuria are indicative of hæmoglobinuric nephritis. (*d*) *Acute recurrent* (chiefly hemorrhagic) nephritis is uncommon.

Prognosis.—The prognosis of acute nephritis is always dubious. The following factors are important: (*a*) Recovery is usual in *light forms* not involving the glomeruli. Atypical forms are generally favorable. (*b*) The prognosis varies according to the *causal affection;* the scarlatinal type has a mortality of 33 per cent.; the virulence of the epidemic is an important factor, as are also heart complications. (*c*) *Complications* may affect the prognosis, as (i) intercurrent *inflammations,* such as pneumonia, pleuritis, pericarditis or erysipelas; (ii) *œdema* of the pharynx, larynx, lungs or serous sacs; and (iii) *uræmia,* which will be fully covered under chronic interstitial nephritis; uræmia is more often an adult than an infantile and a late rather than an early complication; headache, delirium, dyspnœa, convulsions, coma and vomiting are among its salient signs.

Course.—The course is seldom fatal within a few days. In general there are more recoveries than deaths. The average duration is less

than two weeks, though one or two years may elapse before recovery. Convalescence is usually complete, though gradual transition into chronic nephritis sometimes results.

Treatment.—Most of the points mentioned will be considered more fully in the treatment of chronic nephritis. (*a*) *Prophylaxis* is of limited value. Drugs which irritate the kidneys should be carefully avoided in acute infections or in pregnancy. In fevers, with or without albuminuria, water should be given freely by mouth or by enema; hydrotherapy is prophylactic, save in scarlatina when all prevention is seemingly futile and cold baths are contra-indicated. Induction of labor is indicated in severe cases in pregnancy. (*b*) *Diet.* Milk is diuretic, non-irritant and contains little or no extractives or salt. It should be given as in typhoid (*q. v.*). Thoroughly cooked carbohydrates, as gruels, are well combined with it. Broths, meats and beef-tea are to be avoided. Alcohol is contra-indicated, save as exceptionally necessitated by the causal disease. (*c*) *Absolute rest in bed* is imperative; rest with the body covered protects the skin and spares the kidneys from the products of the tissue waste; the patient should lie between flannel blankets and should be dressed in flannel night-gowns. (*d*) *No drug* modifies the nephritis; tannin and ergotin (of each five grains three times daily) seem to restrict renal hemorrhage. Salt should be withheld, because it increases albuminuria and œdema (see TREATMENT OF CHRONIC NEPHRITIS). (*e*) The best *diuretics* are *water and milk,* which flush out detritus from the kidneys. The alkaline charged waters seem to search out waste substances in the tissues and facilitate their removal by the kidney; excessive quantities are often given; potassium citrate and sodium benzoate are said to have the same effect; the citrate may be given in doses of half a dram with sweetened weak lemonade. Sweet spirits of nitre 3ss to 3j, q. i. d., is diuretic and also dilates the bloodvessels. All stronger diuretics are to be avoided. (*f*) *Skin. Warm baths* are diuretic and stimulate some vicarious elimination through the skin. Careful covering and rest in bed keep the skin relaxed and not only materially aid its functions but also relieve renal hyperæmia. (*g*) Management of the *bowels* is as important as that of the skin. *Saline catharsis* lowers arterial tension and aids compensatory elimination. As salts often derange the stomach, an ounce should be given by rectum with two ounces of glycerine and one of water. One-half to one dram of compound jalap powder or $\frac{1}{10}$ grain of elaterin is indicated when uræmia is imminent. (*h*) *Treatment of uræmia.* For suppression of urine a full warm bath lasting fifteen to twenty minutes should be given; a hot pack should be applied followed by wrapping in blankets and covering heavily with a rubber sheet; or the alcohol sweat or hot-air sweat may be given as described under TREATMENT OF CHRONIC NEPHRITIS. Cups applied to the back may divert blood away from the kidneys, because the vessels of the loin anastomose with those of the kidney. Pilocarpinæ hydrochloridum, gr. $\frac{1}{5}$ in adults and one-half as much in children, causes profuse sweating and

salivation but the author abandoned its use after two deaths from sudden pulmonary œdema in patients with apparently strong hearts; it should never be employed when the heart is weak. Active, rapid catharsis is effected by giving two drops of croton oil on the tongue; this may be repeated in two hours if necessary. If uræmic symptoms (in an œdematous patient) appear slowly, an incision two inches long should be made over the lower tibiæ down to the bone, thereby directly draining away the serum with its retained toxins; this is better than the use of small punctures or Southey's capillary tubes, which are attended by all the dangers of puncture-wounds. If uræmia develops suddenly the legs should be incised and a pint or more of blood should be withdrawn from the veins of the arm; whether venesection relieves the circulation mechanically or removes toxins is still a matter of theory, but relief is often obtained. It should be followed by subcutaneous normal salt infusions—or plain water without the salt —which stimulate the heart and kidneys and lower the molecular concentration of the blood. Pleural or ascitic hydrops should be aspirated. The heart must be stimulated by strychnine and digitalis as in cardiac insufficiency, though hypodermics are better than oral administration. A hypodermic of morphine is beneficial and harmless when there is much dyspnœa and restlessness. Chloral and bromides modify sudden convulsions (*v.* Chronic Nephritis). Œdema of the lungs (*q. v.*), vomiting and diarrhœa are caused by several factors, *viz.*, cardiac stasis, uræmia, œdema of the alimentary mucosa and the vicarious elimination of urea, which breaks up in the digestive tract into irritating ammonium; vicarious vomiting and diarrhœa should not be checked at once; if persistent, the vomiting is treated as outlined in chronic gastritis (*q. v.*) or typhoid; gastric lavage is beneficial and dilute hydrochloric acid, ♏x to xv, neutralizes the ammonium. (*i*) *In convalescence,* iron and other mild tonics should be given, irritating foods and beverages interdicted, exercise carefully restricted and cold and exposure avoided by sending the patient, if possible, to a warm climate.

CHRONIC NEPHRITIS.

Our knowledge of chronic Bright's disease dates from Richard Bright's communications in 1827. Aetius, Avicenna, Cotugno (1770) and other writers mentioned the association of hydrops and albuminuria, but to Bright is due the credit of correlating renal disease, albuminuria and dropsy; he also noted the etiological importance of alcohol and cold, as well as the cardiac hypertrophy, uræmia, brain hemorrhage, coma, convulsions, blindness and the tendency to serositis.

The following classification of nephritis covers typical cases, though *combined* or *mixed types* are equally frequent. As a cause of death chronic nephritis ranks sixth, after pneumonia, tuberculosis, heart disease, endocarditis and unknown causes.

I. Chronic Parenchymatous Nephritis.—Definition.—Chronic Bright's disease, with special involvement of the parenchyma (though the interstitial tissue is always affected), with much albuminuria and with abundant formed elements in the urinary sediment. The affection constitutes 3 per cent. of all diseases.

Etiology.—It may result from (*a*) *infections,* as tuberculosis (which causes 25 per cent.), malaria and syphilis; (*b*) from exposure to *cold and dampness;* (*c*) from toxic factors, as alcohol and lead and less frequently mercury or arsenic; (*d*) from *cardiac disease,* which accounts for 14 per cent. of nephritis. (*e*) Exhausting *discharges,* suppuration and ulceration may cause it. (*f*) *Acute nephritis* seldom becomes chronic, though it is a somewhat predisposing factor. (*g*) *Age and sex.* Most cases occur between twenty and fifty years of age; it is rare in children. Males are affected more often than females.

Pathology.—*Three types* of the disease exist, besides the mixed forms.

1. The "Large White Kidney" or "Inflamed Fatty Kidney."—This is never decreased in size and may weigh 10 to 12 ounces (300 to 360 gm.) and measure two or three times the normal size.

Gross Characters.—The capsule is loose and non-adherent; the surface is smooth, yellow-gray, with prominent stellate veins and sometimes dots of hemorrhage. On section the cortex is broader than normal, the striations are poorly marked and when cut streaks of fat may show; the glomeruli and convoluted tubules are yellow and contrast with the gray bands of newly formed connective tissue. The medulla shows fewer changes and is red, in sharp contrast with the light cortex.

Microscopic Characters.—The most essential changes are hyaline and fatty degeneration and atrophy, chiefly in the convoluted tubules, in which lie desquamated epithelia and coagulated albumin. The glomeruli are always diseased, but to a varying degree; they are enlarged, their vessels show hyaline and fatty degeneration and nuclear multiplication; albumin is found in their capsules and the epithelium is swollen, fatty or even necrotic. The connective tissue is always increased, which histologically separates chronic nephritis from acute nephritis and mere degeneration. These changes are never equally diffuse.

2. The "Large Red" or "Variegated Kidney."—*Gross Characters.*—It is also large, but is firmer than the kidney just described or that of acute inflammation, for it has more connective tissue, which may cause some tearing and loss of the cortex when the capsule is stripped off. The surface may be perfectly smooth. The cortex is swollen, indistinctly striated, dotted yellow from fatty glomerular change or red from minute ecchymoses and sometimes streaked yellow and red from similar tubular changes.

Minute Characters.—These are essentially those of the large white kidney, with the exception that fatty degeneration is less marked and connective tissue alteration and hemorrhages are more conspicuous.

3. The Secondary Contracted Kidney.—This form may develop after a year or two from the above forms. Some consider it a primary independent form.

Gross Characters.—It is seldom decreased in size and may be as large as the "large white" or "large red kidney"; the name refers rather to its connective tissue than to any visible renal shrinkage. The capsule is adherent in places and, when stripped, tears off small areas of the renal substance. The surface is rough and shows in some places reddish depressed areas (fibrous contraction or granulations and parenchymatous atrophy) and in others yellow areas (which are more normal, though fatty). Section shows decrease in the cortex corresponding to the reddish foci. It differs from the genuine contracted kidney (primary interstitial nephritis) in the following respects: it is secondary, much more rapid in development, contains fewer cysts, is more yellow and its red granulations and yellow prominences are larger.

Minute Characters.—Epithelial changes resemble those of the two forms described. The membrana propria of the tubules thickens, adheres and obliterates many tubules. The glomeruli collapse, thicken and shrink to small dark-gray granular bodies, which sometimes calcify. The retracted areas, capsular adhesions and cortex shrinkage correspond to connective tissue contraction.

Symptoms.—The *onset* is in most cases *gradual* and *insidious,* the earliest symptoms being loss of strength, pallor, emaciation, simple decline of health, indigestion, anorexia, headache, shortness of breath on exertion or chronic bronchitis. Sometimes there is tenderness over the kidneys.

1. Urinary Findings.—Except in the secondary contracted kidney (*a*) the *urine is decreased* to seven ounces or a quart (200 to 1,000 c.c.); (*b*) the color is *dark* yellow-red, opaque or rarely smoky; the urates are held in suspension by the albumin; sometimes its surface is shimmering from fatty cells and casts; (*c*) the specific gravity is 1,020 to 1,040 and (*d*) the reaction is acid; (*e*) *albumin* is almost never absent, is *more abundant in the day time* than at night and amounts to 1 to 3 per cent. and one-half to one ounce (15 to 30 gm.) daily; it coats the bubbles formed by agitation of the specimen and preserves them for a long time. (*f*) The absolute amount of *solids* is generally *decreased;* for example, the urea (which normally constitutes 90 per cent. of the nitrogen output and amounts to about an ounce (35 gms.) daily), kreatin and sodium chloride are decreased and the freezing point of the urine is lowered (*v. i.*). (*g*) The *sediment* is *abundant* and hyaline, granular, fatty and epithelial *casts abound* (first noted by Simon and Nasse); coarsely granular and waxy casts appear late in the disease; the granular type is due chiefly to fat particles, which may darken the casts. They have been long regarded as significant of nephritis. Key, Nothnagel, Rosenstein and others afterwards observed their occurrence in the urine of normal individuals and of late there has been a tendency to under-

estimate their importance. According to Senator, casts originate in the kidney chiefly from epithelial cells and always indicate some renal disease. They may be dissolved in the bladder by the pepsin contained in the urine (Sehrwald). Leukocytes and fatty epithelia and, in some forms of nephritis, red cells occur in considerable amounts. In Ackermann's case no casts were found in the urine, but were found in masses in the renal pelvis at necropsy.

In the secondary contracted kidney the urinary findings are the same except that the urine amounts to three pints or two quarts (1,500 to 2,000 c.c.), is clearer, is somewhat lower in specific gravity and contains somewhat less albumin and casts.

2. Œdema.—Œdema is very frequent. Its absence argues for little glomerular involvement. It commences *about the eyes, changes* its location readily and is at first evanescent, but soon *becomes general, obstinate* and *ominous.* It is more pronounced in the "large white kidney" than in other types and increases as the urine decreases. The puffy eyes, pale swollen cheeks, dull expression, distended abdomen and shapeless wrists and ankles often suggest the diagnosis. The *serous* and *mucous membranes* are similarly involved, though to a lesser degree, and also as possible terminal events, pulmonary or cerebral œdema may develop. Œdema is caused (*a*) by hypalbuminosis and hydræmia of the blood (Bright's theory) or (*b*) by alterations in the vessel walls, *i. e.,* abnormal permeability, a theory advanced by Cohnheim and Lichtheim.

3. Anæmia.—Anæmia distinguishes chronic parenchymatous nephritis from the genuine interstitial type. There is usually retention of urea, uric acid and chlorides; sometimes the blood is cloudy.

4. Cardiovascular Findings.—The pulse is more often small, soft, rapid and associated with a weak apex, indicating dilatation, than tense and associated with a loud second aortic tone and strong apex, indicating hypertrophy. Secondary contraction and mixed nephritis are likely to develop findings similar to those of the genuine interstitial nephritis; Senator finds hypertrophy with dilatation (eccentric hypertrophy) in parenchymatous and simple or concentric hypertrophy in interstitial nephritis. Myocardial degeneration is common.

Complications and Causes of Death.—(*a*) *Marasmus* results from the anæmia, indigestion, diarrhœa, œdema of the alimentary mucosa or its irritation by decomposed urea. (*b*) Intercurrent *infections* may result, as inflammation of the pleuræ or pericardium, cellulitis and especially pneumonia; many pneumonia deaths really result from nephritis; in one instance the writer saw acute necrosis and sloughing of the scrotum, after which the patient improved remarkably. Other complications are (*c*) *œdema* of the lungs, larynx, serous sacs and extremities, impeding respiration and obstructing the arterial flow in the peripheral vessels; (*d*) *hemorrhages* (in 8 per cent.) in the brain or other organs; (*e*) *retinitis* (in 18 per cent.); (*f*) *uræmia,* with its numerous nervous, circulatory and digestive

symptoms (*v.* page 750); (*g*) *acute exacerbations,* particularly in the chronic hemorrhagic type; and (*h*) *cardiac insufficiency.*

Diagnosis.—The diagnosis rests upon three cardinal symptoms: (*a*) the *urinary,* which are characteristic when twenty-four-hour specimens are carefully and repeatedly examined in every case coming under our care; (*b*) the *anasarca,* which is usually present, though sometimes absent in secondary contracted kidney and in Wagner's hemorrhagic type; and (*c*) the *anæmia.* (For *differentiation,* see pages 764 and 765.)

Prognosis.—Though some cases suggesting an arrest of the process are found at autopsy and some cases recover after a year or two, the clinical course is usually progressive and fatal. Dropsy usually causes death in a few months to a year, though the author has seen five typical cases recover after two and a half years; these cases did not result from acute infections like the cases of recovery reported by Senator and Rosenstein. The outlook is better in children than in adults. The "large, white kidney" causes death in one-half to one year, the "large, variegated kidney" in one and a half to three years and the secondary contracted kidney in one and a half to three years. The treatment is considered on page 754.

II. Chronic Interstitial Nephritis.—Definition.—A primary, interstitial, contracting sclerosis of the kidney, characterized clinically by abundant urine of low specific gravity, with small amounts of albumin and few cellular elements; and by marked cardiac hypertrophy, arteriosclerosis, retinitis and uræmia.

This genuine contracted kidney must not be confused with secondary contracted kidney, arteriosclerotic contraction, embolic contraction or the contraction resulting from ascending infection.

Frequency.—In ten years Eichhorst treated 31,562 cases, of which 1.4 per cent. had contracted kidneys; he finds this form six and a half times as frequently as the parenchymatous form, though with us the latter is at least equally frequent.

Etiology.—The etiology is essentially that of *arteriosclerosis,* with which contracted kidney has a three-fold relation: (*a*) arteriosclerosis may cause the arteriosclerotic kidney; (*b*) contracted kidney may cause arteriosclerosis; or (*c*) both may result from a common cause. The noxa or toxin reaches the kidney through the blood stream.

The disease may result from (*a*) chronic *alcoholism* combined with exposure or over-eating; (*b*) chronic *lead-poisoning,* proven a cause by clinical and experimental evidence (Lancereaux and Ollivier); (*c*) *gout* (Todd), caused by uric or oxalic acid and the alloxur bodies; (*d*) *syphilis;* (*e*) *diabetes* (Senator), caused by sugar, acetone or oxybutyric acid; the diabetes lessens or ceases when induration develops; (*f*) *age;* it begins between the years of *thirty and fifty* and is clinically manifest after fifty years of age. Sawyer collected 24 cases in children, 13 of whom were under ten years. (*g*) *Sex.* The disease is two to three times as frequent in *men* as in women. In the in-

fantile form most cases occur in girls. (*h*) Other *infrequent causes* are acute nephritis, the kidney of pregnancy, hereditary tendency, as in Dickinson's series through four generations, and endocarditis.

Pathology.—It is undecided whether the process is inflammatory or atrophic; Weigert believes that cellular degeneration occurs first and induration follows.

Gross Pathology.—(*a*) The kidneys are *contracted* even to one-third their original size and may weigh but 0.6 ounces (21 gm.); contraction is rarely symmetrical in both. (*b*) Their *consistence* is *increased.* (*c*) The *capsule* is *thick,* tendinous, wrinkled, vascular and *adherent* and strips off particles of cortex when removed. (*d*) The *surface is uneven;* there are small reddish or sometimes paler prominences, measuring 0.5 to 5 mm. and representing the more normal tissue; the paler retracted areas are fibrous retraction. (*e*) Small *cysts* are frequently found, usually with clear contents; they vary in number and their size ranges from that of a pin-point to a cherry; they apparently result from glomerular and tubular constriction by connective tissue (retention cysts). (*f*) On section the *cortex is shrunken,* even to 1 mm., irregular and gray from fibrillar connective tissue. The medulla is less changed, though also shrunken; pale fibrous lines alternate with red dilated vessels; uric acid or calcium salts are deposited. (*g*) The pelvis is large and much fat surrounds the kidney.

Minute Pathology.—The changes are largely cortical, focal, asymmetrical and interstitial. (*a*) The *glomeruli* early contain round cells, nuclear increase, desquamated cells and albumin; later they *atrophy* from degeneration of the afferent artery; multiplication of cells is noted between the loops and hyaline change occurs in the glomerular vessels; in the extreme stage the glomeruli become *hyaline and fibrous;* some tufts remain normal and others may visibly hypertrophy, Sabourin's compensatory adenomata. (*b*) The *epithelium* is always changed, though much less than in parenchymatous forms; in the sunken areas many tubules disappear and degenerated cells are seen in the prominent surface granulations; in the medulla the collecting tubules are larger, even cystic, and are filled with colloid material. (*c*) The *connective tissue* is increased everywhere, but more in the cortex than in the medulla; round cell infiltration occurs early and induration late; the vessels are sclerotic in all their coats, but especially in the intima, and many writers consider that the vascular change is the earliest and causative alteration.

Symptoms.—*Long latency* for years, *insidious onset* and *protracted compensation* by *heart hypertrophy* are characteristic of this type. Many cases are first discovered during an acute infection, as pneumonia, on examination for life insurance or at the coroner's autopsy after accidents. It may appear to be a physiological, intermittent or cyclic albuminuria. Polyuria, cardiac symptoms, retinitis, indigestion or depreciation of health may be the first symptoms. The œdema, pronounced urinary findings (in a single specimen) and

anæmia of chronic parenchymatous nephritis are seldom conspicuous and almost never early symptoms in this type. The condition may be first declared by heart incompetence, uræmic coma or convulsions.

1. Urinary Findings.—Urinary findings are generally the earliest signs. Twenty-four-hour specimens should be examined repeatedly in doubtful cases (with a fresh specimen for microscopic examination). (*a*) The *amount,* at first normal, is gradually increased (*polyuria*); this is a frequent cause for consulting the physician. Nagel found polyuria in only 47 per cent. The urine is also passed frequently (*pollakiuria*), especially at night; this is always suspicious when the prostate is normal. In well-developed cases it totals two to three quarts (2 to 3,000 c.c.), rarely more, though 8 to 10 quarts (8 to 10,000 c.c.) were passed in a case of Lecorché and Talamon and 12 quarts in Bartel's case. (*b*) It is *pale* and (*c*) *weakly acid.* (*d*) The specific gravity averages 1,010 (1,005 to 1,012). (*e*) A *permanent foam* due to the albumin is frequent. (*f*) *Albumin* is present *in most cases,* but is *not abundant;* the merest traces are found up to 8 grains (0.5 gm.) pro mille, and a daily total of one-half to one dram (2 to 5, rarely 10 gm.). It is often absent in single specimens, especially in the morning, whence a twenty-four-hour specimen should always be examined. Delicate tests are often necessary (*v.* Albuminuria). It may appear only after eating, exercise, excitement or fatigue, whence its greater frequency toward night; it may increase after dyspepsia or bronchitis. Senator's statement that its alleged absence is due to an arteriosclerotic kidney and not to interstitial nephritis is not invariably correct, according to personal experience. Albuminuria may be absent for weeks or months. (*g*) *Solids are decreased, i. e.,* the urea, uric acid, ammonium, chlorides and phosphates. The determination of the freezing point, *cryoscopy,* first employed by Korányi to estimate the amount of soluble molecules in the blood and urine, is not of great practical value in medical cases. (*h*) The *rapidity of elimination* is decreased. Methylene blue was employed by Achard and Castaigne (1897) to test this point. One cubic centimeter of a 5 per cent. aqueous solution is injected subcutaneously; normally it should stain the urine in one-half to one hour, reach a maximum in three to four hours and cease in thirty-six to seventy-two hours; elimination may be nearly normal or even more rapid in chronic parenchymatous, but it is much slower in interstitial nephritis (Bard). The permeability of the kidneys may also be tested by a hypodermic injection of one cubic centimeter of a one-half per cent. solution of phloridzin (Achard), which produces glycosuria in normal kidneys; phloridzin glycosuria is reduced or absent in interstitial nephritis. (*i*) *The sediment is scant,* absent or can be obtained only by the centrifuge. Hyaline, granular and epithelial casts are found in small numbers when the light is moderated by the diaphragm; the granular form is found in some cases only after repeated search; the granular casts are often wide, originating in the dilated tubules of the medulla. A few epithelia, leukocytes and crystals of uric acid and oxalate of lime are found. Red cells are infrequent,

The urinary findings are somewhat modified by intercurrent uræmia, fever and heart failure, all of which concentrate the urine, raise its specific gravity and deepen its color.

2. Cardiovascular Symptoms.—Cardiovascular symptoms were noted by Bright, but their significance was first established by Traube. They appear in 50 to 90 per cent. of cases and later than the urinary findings. *Cardiac hypertrophy* is more quickly detected clinically than pathologically, especially in the young, because the urine is increased early before the physical signs of hypertrophy prevail and again because concentric hypertrophy is present long before dilatation appears. Emphysema may obscure the precordial evidences of hypertrophy, such as the *heaving apex beat* and double impulse at the apex, and one must therefore depend on the evidences presented by the *tense pulse,* the hardness of the arteries, the sphygmographic tracings with a square apex, the loud ringing second aortic tone or other manifestations of increased arterial tension, as epistaxis, vertigo, headache, tinnitus, palpitation or cardiac asthma. The systolic blood pressure in interstitial nephritis may amount to 200 mm. or more. High tension is one of the most constant and early symptoms; its early occurrence leads some writers to incorrectly regard contracted kidney rather as a disease of the arteries than of the kidneys. The cause of the hypertrophy is not definitely known; theories of mechanical obstruction to the blood-flow by the renal contraction and of vasomotor constriction excited by retained waste substances have been advanced. Loeb's explanation is plausible; the increased blood pressure is of a regulatory nature and is produced by changes in the glomeruli; whether reflex or chemical agencies induce the increased pressure is not known. Though hypertrophy should be looked for in interstitial rather than in parenchymatous nephritis, the two types often mingle, with the urinary findings of the "large white kidney" and the cardio-vascular findings of a renal contraction. In the last stage of contracted kidney the hypertrophied heart weakens, the pulse tension falls, the apex becomes weaker, a systolic murmur appears from myocardial insufficiency and Potain's gallop-rhythm, a sign of heart fatigue, is heard.

3. Retinitis.—Retinitis may be the first declaration of the disease and may come under the observation of the ophthalmologist. It occurs more frequently than in any other renal lesion, 22.4 per cent. (Grönauw) to 28 per cent. (Eates). Its various forms are stellate white areas of fatty and fibrous tissue, a white zone surrounding the disk, degeneration of the disk, retina or of both together, "flame-like" hemorrhages and the choked disk whose large veins are compressed where the arteries cross them. (See Plate VIII, Fig. 8.) The retinitis albuminurica is not specific, identical findings occurring in other conditions.

4. Uræmia.—Uræmia is the most dreaded and frequent complication; its nature and immediate cause are unknown; it can only be stated that it is an auto-intoxication, due to disturbance of renal

function, probably with retention within the blood of nitrogenous substances. Further explanations are wholly theoretical, such as decreased alkalinity of the blood (acidosis); failure of the normal "internal secretion" of the kidney; the toxic action of abnormal products of metabolism or breaking up of the proteids, resulting from destruction of the renal tissue (nephrolysins, Ascoli); it is not due to retention of urea alone nor to mechanical conditions alone, as œdema or anæmia of the brain. It occurs most often in chronic interstitial, next in acute, then in chronic parenchymatous nephritis and is much less common in other diseases of the urinary tract, as pyelonephritis, etc.

Uræmic *symptoms* may be acute or chronic, but will be considered as nervous, digestive, respiratory and cardiac.

Nervous Symptoms.—(*a*) Acute *convulsions* (eclampsia) are due to irritation of the cortex by toxins; they closely resemble the epileptic attack. There are usually prodromes, as pain or pressure in the head, migraine, neuralgia, insomnia, vertigo, visual disturbance, tinnitus, nausea, vomiting or dyspnœa; there is usually no "epileptic cry." In less than a day clonic or tonic convulsions appear, usually generalized and involving especially the flexor or extensor muscles of the arms and legs; the abdomen is retracted; there are dyspnœa, cyanosis and involuntary evacuations; the reflexes, especially the patellars, are increased; the pupils are wide and without reaction; the pulse is full and slow before the attack but becomes rapid during the convulsions. The skin is covered with sweat and the temperature is usually elevated. The convulsions cease after fifteen to thirty minutes and coma intervenes, during which the convulsions may recur. Death is the usual outcome, but recovery is possible, as in one of the author's cases in which the anterior third of the tongue was bitten off. (*b*) *Other motor manifestations.* The general statement regarding uræmic nervous phenomena is as follows: *paralysis* affects especially the sensorium, to a lesser degree the special senses and rarely motility; *irritation* seldom affects the sensorium (*e. g.,* produces delirium), it acts principally upon the motor paths (convulsions) and very seldom upon the special senses. Uræmic convulsions are usually general but may resemble focal symptoms, such as *convulsions of only half the body, e. g.,* of one-half the tongue, of the left arm and leg or deviation of the head to the left. They may rarely occur with convergent strabismus, facial spasm, Jacksonian epilepsy, nystagmus, tremor, localized trembling and twitchings, muscular cramps, grinding of the teeth, tetanic convulsions, trismus, convulsions on one side and contractures on the other, rigidity of the neck or opisthotonos. In seven cases seen by the author the symptoms were those of an acute focal brain lesion; in two, treated by lumbar puncture, relief was immediate.

Paralyses in uræmia are infrequent, are chiefly transitory hemiplegias and are due to *cerebral œdema,* capillary hemorrhage, softening, apoplexy or cortical overstimulation by toxins. Hemiplegia may occur with aphasia, amnesia, deafness, crossed oculomotor paraly-

sis, glossoplegia or conjugate deviation of the eyes. The author has reported five cases in which uræmia resembled meningitis very closely. Other cases have been suggestive of brain tumor. (*c*) *Acute coma* often occurs without antecedent eclampsia, being preceded by headache, stupor or apathy; coma may last for days or even months. (*d*) *Psychical symptoms.* One of the most common chronic symptoms is a *characteristic restlessness and anxiety.* Obstinate *headache* of a dull, throbbing nature is common: it often begins in the early morning; it may be occipital or hemicranic. *Insomnia* is common, though during the day the patient is often sleepy. Delirium and mania are more frequent in chronic nephritis in adults than in juvenile cases; melancholia and delusional insanity occur; these mental states may assume medico-legal importance as to the testamentary capacity of the patient. (*e*) *Special senses.* Sudden, complete and bilateral blindness is cortical in origin; this *amaurosis* may be an initial symptom; there are usually no retinal findings and the condition lasts but a few hours, though in exceptional cases œdema of the disk (Schmidt) or even choked disk is found. The pupils are wide and reactionless in acute eclampsia, but are small and mobile in chronic uræmia. There may be sudden or gradual uræmic deafness and tinnitus aurium.

Digestive Symptoms.—These rank second to the nervous symptoms in frequency. The breath is often offensive and sometimes has an odor of urine, which Senator refers to trimethylamine; he considers it of prognostic value, for it often precedes eclampsia. The tongue is foul, the mouth is inflamed and the saliva is frequently increased. *Nausea and vomiting* are partly central and partly local from decomposed urea, eliminated vicariously into the stomach; the same may be said of *hiccough* and *diarrhœa;* the HCl is decreased or absent. All these symptoms may be very acute. Diarrhœa may be serous or dysenteric from the Bac. dysenteriæ (Flexner). Mathieu and Roux in a recent study of 23 cases of "uræmic ulcers," described by Treitz (1859), found them chiefly in cases of advanced nephritis in subjects under twenty years old; they rarely occur in the stomach; diarrhœa is common, but bowel hemorrhage and constipation are rare. They are seemingly produced by necrosis due to toxæmia. Healing is very rare and the prognosis is almost always unfavorable.

Respiratory and Cardiac Symptoms.—Dyspnœa is very common. It is sometimes toxic and central, occurring in close parallelism with cerebral manifestations. In other cases asthma uræmicum develops when the mind is absolutely clear and is then correctly referred to the heart. *Prodromal slowing* of the heart to 60 to 40 beats is frequent in incipient uræmia. The dyspnœa may be continuous or paroxysmal and is often nocturnal. Cheyne-Stokes' breathing may develop in the day time or during sleep and is an unfavorable sign, though recovery is possible. Obstinate hoarseness and a rare form of laryngeal spasm are recorded.

5. Other Complications and Symptoms.—(*a*) Various *hemorrhages* occur; in the brain in 12 per cent. of cases; also in the dura,

skin, nose, bronchi, stomach, bowels, uterus, conjunctiva or retina. The author has observed 9 cases of profuse gastro-intestinal hemorrhages in uræmic subjects. (*b*) *Inflammations* may develop. Chronic bronchitis always suggests kidney disease. Pericarditis, pleuritis and pneumonia especially, may develop as terminal infections. Many deaths from pneumonia are due to nephritis, which was previously unnoticed. Laryngitis and acute (inflammatory) œdema glottidis may occur. (*c*) *Œdema* is a late symptom and results not from the nephritis, unless it is of the mixed type, but from failing of the heart. Ascites also is nearly always cirrhotic or cardiac. (*d*) The *skin* is usually dry and harsh and is sometimes covered with urea crystals—the "urea frost" or uridrosis. Itching, erythema, urticaria, eczema and numbness or "dead fingers," unusual susceptibility to cold (cryæsthesia), neuritis or rheumatoid pains are probably toxæmic.

Diagnosis.—There are *three cardinal findings, cardiovascular, urinary* and *retinal;* as Senator says, he who invariably examines the urine and heart in every instance rarely fails to diagnosticate nephritis correctly.

Cardiac Symptoms.—The very interdependence of cardiac and renal changes may prove confusing; thus primary cardiac disease may cause renal congestion, embolism or even acute or chronic nephritis; again alcohol or syphilis may be a common cause of arteriosclerosis, myocarditis and nephritis; and finally, the heart lesion may be secondary to renal disease. Renal stasis is considered in the differential table given below. In terminal cardiac weakness caused by contracted kidney, the hydrops, dyspnœa, rales or systolic murmur may suggest myocarditis with renal stasis; gallop-rhythm is more common in the heart of renal disease than in primary cardiac affections.

The Urinary Findings.—In *diabetes mellitus* there is an increased flow of urine, in which the high specific gravity, glycosuria and less prominent heart findings are distinctive; in *diabetes insipidus* the absence of albumin, casts and cardiac changes are characteristic; and *pyelitis* is characterized by albumin and pyuria but not by casts or cardiac changes. In the *arteriosclerotic kidney* arterial and cardiac antedate the urinary changes (the converse is true in interstitial nephritis), the specific gravity is higher, albumin less in amount and in frequency of occurrence and the course is much more rapid. For differentiation from other renal diseases see page 764.

Diagnosis of Uræmia.—Skull-fracture, miliary tuberculosis, leptomeningitis, cerebral abscess, sepsis and other diseases may be diagnosticated uræmia simply because nephritis is also present. Senator has said that without a previous history we can often only diagnosticate a renal disease and suspect the presence of other (latent) affections. The danger of mistaking uræmic symptoms, as hemicrania, nervous symptoms, dysentery, etc., for independent disease is greater in direct ratio to the difficulty of diagnosis of the nephritis itself and many authors have remarked on the lack of correspondence between

the clinical and pathological findings in nephritis. According to Leube, uræmic intoxication is assumed to be the cause of nervous symptoms only when other causes may be excluded, because in the course of renal disease organic nervous lesions, as hemorrhage and meningitis, may simulate uræmia. Though the urine is usually decreased before an attack of uræmia, Liebermeister instances a case in which, immediately before a uræmic seizure, both urine and urea were increased two-fold. Biermer reports an anuria which lasted two hundred and twenty-two hours before uræmia developed.

Apoplexy is probably present when pressure symptoms, as vomiting or slow tense pulse, complete hemiplegia or conjugate deviation exist; it is rarely completely simulated by uræmia. Meningitis, insolation and alcohol or opium coma are rarely simulated; in very exceptional instances focal symptoms and fever may cause diagnostic error. The author has seen a few cases in which uræmia simulated typhoid or sepsis by the constant high temperature and two cases with no bacteriological evidence of terminal infection.

Prognosis.—Because the onset is insidious it is often detected only when the patient suffers from cardiac insufficiency, beginning uræmia, apoplexy or intercurrent inflammations; for this reason this incurable malady is seldom treated in its early stages. The clinical course usually runs five to ten years after albuminuria is found; a course of twenty years is reported by Senator and Aufrecht, of twenty-three years by Oppolzer and of thirty by Rosenstein. Much depends on the docility and financial ability of the patient. Marked arteriosclerosis hastens the fatal issue. *Retinitis* is an ominous symptom; in Belt's 419 cases 72 per cent. died in one and 90 per cent. in two years. Retinitis increases the gravity of the outlook four-fold. Staub's and Grüning's figures are almost identical. Webster, Wert, Hare, Mix and the author have seen cases which lived from four to ten years after retinitis was found. Uræmia, pneumonia, apoplexy and other complications are decidedly unfavorable.

Treatment of Chronic Nephritis, Parenchymatous and Interstitial.—Both forms are considered together, because they often blend (mixed or diffuse nephritis).

1. Prophylaxis.—Prevention and treatment of the cause have a very limited practical application. Irritating foods, excessive eating, all forms of intemperance, over-work, neglect of infections and dyspepsia come under this head, but are usually treated too late.

2. Rest, Exercise.—The kidneys should be relieved by rest in the horizontal posture, because exertion and even the erect position, which *per se* may cause albuminuria, only increase preëxisting albuminuria. If in severe cases exercise seems advisable it should be given passively with the patient in the recumbent posture. The rest should be absolute as long as the heart is weak, the urine scanty or hydrops conspicuous. The importance of rest in bed was recognized by Bright and Bartels. When there is moderate renal involvement or in the

intervals of improvement relative restriction of exercise should be enforced. Rest in bed lessens the amount of waste products to be eliminated by the kidneys and so protects the skin and dilates its vessels that the cutaneous functions become more active and relieve the vascular hypertension, just as a patient with a cold should remain in bed in order to increase the activity of the skin and kidneys. The author treated two severe cases of parenchymatous nephritis by rest in bed for a year and absolute recovery resulted. It is a question if lying covered in bed is not one of the reasons why nephritics pass more urine at night than in the day time. Fatigue, dampness and cold must be avoided; these indications are best carried out by rest in bed during the time of severe symptoms or of acute exacerbations and by relative rest and warm clothing in cases of moderate severity. Practitioners do not sufficiently insist on psychical rest; in men leading a strenuous life moderate nephritis has often decreased and sometimes disappeared when nervous tension was relieved; too often these subjects take physical exercise or courses of training after they are already exhausted by head-work; in any event exercise should supplant work and not be taken after hard nervous strain.

3. Springs, Climate.—"Cures" at water resorts have the general advantage that the importance of diet and hygiene is impressed on the patient; the psychical or suggestive element is also an important therapeutic agent. Diffuse nephritis (with considerable parenchymatous and interstitial change) is most benefited. Only patients without œdema or serious heart involvement should be sent to such places as Poland Springs, Bedford Springs, Saratoga, Vichy or Neuheim. A stay in the California, Mississippi or North Carolina climate during the winter, at least, will materially benefit.

4. Diet.—The diet must be non-irritative, must not especially tax the kidneys in its elimination, must not burden the circulation and must maintain nutrition. Disease of the glomeruli alone may be compensated by vicarious excretion of water through the lungs, skin and intestines, but disease of the tubules whose function is elimination of the end-products of albuminoid metabolism, cannot well be compensated by other structures, whence the indication for *limitation* of *albuminous foods* and substitution by fats and carbohydrates. The small amount of albumin lost through the kidneys can be offset by a glass or two of milk daily; in this respect nephritis differs from diabetes in which the system is irritated by unappropriated sugar and loses a vast number of units of heat and energy each day. Albuminous food is restricted only to spare irritation of the excreting renal cells and no absolute rule can be laid down for all cases. Chittenden has recently shown that we eat twice as much meat as we need. It is an obvious mistake to gauge the severity of a nephritis by the degree of albuminuria or to estimate improvement by the influence of diet upon the albuminuria alone. Fats as butter, olive and cod-liver oils and cream, are very important, because they supply energy and heat units in place of the restricted albumin. As in diabetes the re-

striction of diet must not merely concern renal irritation, but most of all, the maintenance of nutrition, especially when the patient suffers from parenchymatous nephritis, the very form in which restriction of diet is theoretically most needed; anæmia and malnutrition are the most distinguishing features of the parenchymatous as contrasted with the interstitial type of nephritis. The patient is considered more than the disease or the type of the disease.

The *milk diet* is free of extractives and salt, easy of digestion and diuretic. It has its restrictions; it is bulky, over four quarts being necessary to maintain nutrition and it therefore may cause dyspepsia; it lacks iron, but this can be easily compensated; four quarts contain an excess of about 60 per cent. of proteid; it contains too much water, thereby possibly overloading the blood and straining the heart; and its phosphates tax the kidneys. One to one and a half quarts of milk with a pint of cream, fats and carbohydrates (baby foods, sago, rice or potatoes) will meet the needs of metabolism; and sometimes over half of the phosphates of the milk can be precipitated and kept in the intestine and away from the blood and kidneys by giving calcium carbonate gr. v to x with the milk (von Noorden). It should be taken slowly, in sips, as it is a food, not a beverage. Milk was one of the chief articles of diet of the older masters of medicine; a very short time ago we heard that it contained an excess of water; now we are told it is excellent because it contains so little salt. Part-truths are misleading.

In regard to a *meat diet,* probably the present reaction in favor of the meat diet is extreme, as Strubell's experiments demonstrate that meat promotes uræmia. The average daily amount is three ounces (100 gm.). There is absolutely no difference between red and white meats as to extractives or purin bodies. Some meat is preferable to overloading the stomach and bowels with a bulky vegetable diet, save when uræmia is imminent.

Raw *eggs* increase (perhaps induce) albuminuria; not more than two should be given daily. *Substances excreted with difficulty* include the following, according to von Noorden: urea (in meat), creatinin (in meat broths, extractives and to a less extent in eggs), urinary pigments (in hæmoglobin), hippuric acid (in cranberries, fruits with pits, prunes, plums and gages), phosphates (in milk) and inorganic sulphates (in meat); uric acid and alloxur bodies, contrary to general opinion, are, according to von Noorden, eliminated readily (they are found in glandular organs,—such as the thymus, sweetbreads, liver and kidney,—in broths and coffee). The *chlorides* are especially avoided in parenchymatous nephritis with œdema. As pointed out by Widal, Lemerre, Javal, Mercklen and Courmont in 1903, administration of salt (which is retained in the blood) increases the body-weight, induces œdema and aggravates albuminuria, while "dechloridation" of the food reduces weight, œdema and albuminuria. Probably the entire truth is not yet known; Rumpf found cases with œdema, uræmia and retinitis in which the chlorides of the blood were

decreased. Widal's dechloridation diet is ½ pound meat, 1 pound potatoes, 3 ounces sugar, 3 ounces unsalted butter and 2½ quarts milk. *Irritants,* as cheese, relishes, horseradish, onions, rhubarb, asparagus, spices, sauces, broths, beef-tea and alcoholics, should be absolutely avoided; tea and coffee must be limited because they may aggravate or produce nephritis (Penzoldt, Hubach).

5. Water.—In regard to water, the same extremes of opinion are noted; fluids were once excessively restricted and later undue quantities were recommended; von Noorden states that flooding the vascular system with water overtaxes the heart; "water can only be excreted by the kidney if the blood pressure is increased; many patients having weak hearts give a history of excessive water drinking; in such cases improvement is frequent if the ingestion of water is limited; about three pints should be the average amount." Nevertheless relief of high arterial pressure and minor uræmic symptoms may follow copious drinking of water.

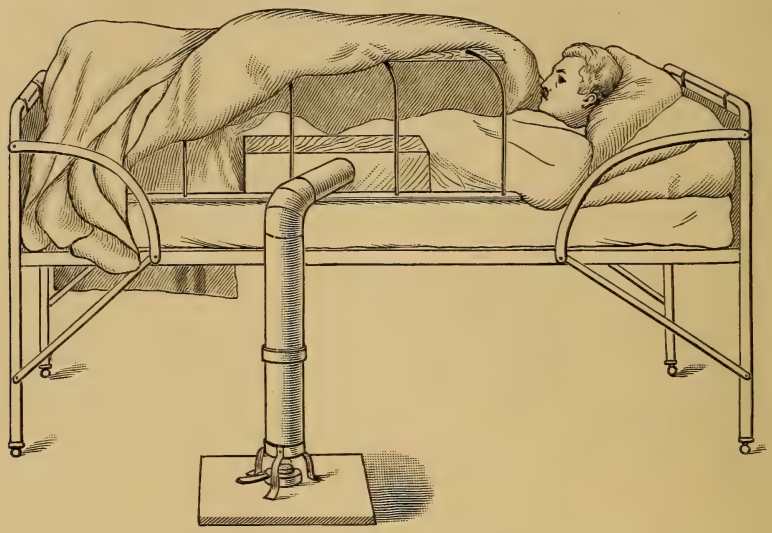
The following therapeutic measures are but symptomatic:—

6. Diuretics.—Diuretin is said to produce no renal hyperæmia but to act directly on the epithelium and, according to Grünwald, to sometimes reduce the albuminuria; it may sometimes be employed with success (*v.* Treatment of Valvular Disease). The use of diuretics is indicated by increase of hydrops, scanty urine and imminent uræmia. The refrigerants (cream of tartar, 3j) or potassium citrate (gr. xx to xxx) are the only safe diuretics; they are thought to abstract water from the tissues and facilitate the excretion of urea, uric acid and similar products; they are apparently most active in the effervescent form. Basham's mixture (liq. ferri et ammonii acetatis) as a tonic and diuretic should be given in doses of 1 or 2 drams. Diuretics which act through the circulation will be considered below.

7. Hydrops.—Hydrops necessitates treatment because it prevents movement, threatens suffocation by pulmonary or laryngeal œdema, increases the danger of secondary infection, as erysipelas or cellulitis, embarrasses the circulation by pressure on the afferent capillaries and efferent lymphatics and venules, interferes with digestion and increases the danger of uræmia. (*a*) Though *cathartics* may eliminate fluids as well as solids, drastics must be given with due care for the digestion and nutrition,—pulv. jalapæ co. 3ss to 3j at night; vegetable are better than saline cathartics. (*b*) *Sweats* are generally given, possibly less so of late. Though the functional relation between the skin and kidneys cannot be overestimated, profuse sweating eliminates fluid rather than solids. Leube holds that sweats concentrate the blood and predispose to uræmia; he states that when a patient sweats one quart (1,000 c.c.) only 7 to 15 grains (0.5 to 1 gm.) of urea are eliminated. The author has more often seen aggravation of symptoms or a fatal issue result from sweating than relief and hence prefers the old-fashioned full warm bath, recommended by Osborne and Liebermeister, to the more active methods; an ice-bag is placed on the head to prevent brain congestion. Commencing with water at

body temperature this is raised five or six degrees. After twenty or thirty minutes the patient is removed and wrapped—without drying—in blankets. A hot-air bath may be given by placing a lamp under one end of a stove-pipe which runs by an elbow into a tent built over the patient, as illustrated in the following cut. In the

FIG. 47.



Sweat bath employed in nephritis.

alcohol sweat, hot bricks protected by flannel are placed around the heavily covered patient; equal parts of alcohol and water are poured on the bricks and the patient is covered again; the heat produces a steam vapor which is well tolerated unless the heart is weak, when fatal collapse may ensue. It is often unsafe to give pilocarpine (*v.* ACUTE NEPHRITIS) because it may produce vomiting, diarrhœa, syncope and pulmonary œdema. (*c*) Mechanical relief by means of *incision* is superior to all other measures which must first bring the fluid with all its urea and extractives into the circulation, with the danger of inducing uræmia. If the kidneys and other emunctories cannot relieve the blood, how can they remove the additional refuse in the anasarcous fluid, which comes to the blood from the tissues? The author makes deep incisions, three inches long, over the lower leg (not over the malleoli where infection from the feet is likely) whenever the hydrops is at all extensive. In testing the fluid evacuated there has frequently been 0.1 per cent. urea which may serve as a measure for other retained products of kindred nature; when quarts seep out during the first twenty-four hours one is well justified in incising the legs, despite the slight danger of occasional infection. Sweats and drugs never achieve equal results. Free incision is obviously less dangerous than punctures or Southey's capillary tubes.

8. The Circulation.—Treatment often resolves itself into the care and stimulation of the heart. Mental excitement, nervous strain, physical exertion, constipation, tobacco, coffee, alcohol and exposure to heat must be avoided. The more the heart hypertrophies the better is the outlook, although ultimate cardiac dilatation is the inevitable fate of every hypertrophy. In the uræmic seizures, with precisely the same symptoms in other respects, the heart may vary; it may beat strongly or very weakly. (*a*) *With cardiac over-action* observed early in uræmia, the angiospasm or the high tension must be corrected by *vasodilators.* Iodides are beneficial when there is no urgency in doses of grs. v to x t. i. d.; iodism, especially salivation, may result from renal retention. When extreme tension and imminent uræmia exist, tr. veratri viridi ♏v to x every half hour for three or four doses may be given; the author prefers spiritus glycerylis nitratis ♏ij until results are produced; he believes that the vasodilators are often given to excess, as hypertrophy and increased arterial tension are less pathological than compensatory conditions. *Bleeding* may so relieve the intracranial blood pressure as to avert for a while the uræmia, but too often it does not; salines may be given afterwards, either subcutaneously or by rectum; they "lavage the blood" and stimulate the kidneys if enough sound tissue be left. Venesection lessens the molecular concentration of the blood (*i. e.,* its toxicity) and relieves the laboring heart; infusions act in the same way; an excess of salt may be given in the infusions (*v. s.*) and when they are repeated the salt should be left out, for it may produce œdema of the lungs. Bouchard states that the abstraction of 32 gm. of blood removes more toxins than 280 gm. of fluid fæces or 100 liters of sweat. Increased intracranial tension may be relieved by *lumbar puncture;* the normal pressure of 120 mm. (as indicated by the manometer) is often increased, sometimes to five or six hundred mm.; the author has seen marked, sometimes protracted relief in medical and eclamptic cases; the headache, vomiting, convulsions and coma are often decreased or entirely relieved. (*b*) When the *heart is weak,* rapid, irregular or galloping, cardiac stimulation by digitalis and strychnine is indicated. Infusion of digitalis by mouth may cause vomiting and ♏ij to v of the fluidextract may then be given hypodermatically or by rectum. Its combination with squills or calomel is inadvisable; the latter may cause the most intense salivation; in one case necrosis of the entire tongue occurred. Cardiac unrest, palpitation or dyspnœa should be relieved by a hypodermic of morphine, by application of *ice to the heart* or by champagne, nitroglycerine, bromides and valerian. Pilocarpin and sweats are distinctly contra-indicated, because they over-tax or fatally depress the heart. Von Ziemssen gave champagne and digitalis by mouth as long as the patient could swallow and then digitalis by rectum and camphor subcutaneously.

9. Vomiting.—Vomiting and diarrhœa are as often vicarious as toxæmic and should not be checked at once. Gastric lavage and colonic flushings are beneficial.

10. Convulsions.—For convulsions, hypodermics of morphine should be given (Loomis and Mackenzie); the author never saw harm result from its use, though medical opinion is generally against it. Chloral (gr. xv every hour for not more than four doses) aids the morphine; bromides generally act too slowly.

11. Operation.—Surgical treatment is still *sub judice*. It is certain that many cases of "decapsulated" kidney were not genuine nephritis, but rather albuminuria from floating kidney or ascending infection. Again, it is still a question whether chronic nephritis is merely a renal disease or perhaps a result of a general blood condition; this idea, advanced by Semmola, is not yet disproven.

PASSIVE CONGESTION.—INFARCTION.

I. Passive Congestion.—Venous hyperæmia is an important renal lesion compared with arterial hyperæmia, of which latter little is definitely known, though it occurs in renal inflammation, left ventricle hypertrophy, diabetes, perhaps also in nervous lesions and after the administration of diuretics.

Etiology.—(*a*) Among the chief causes of *general stasis* are cardiac insufficiency from valvular disease, myocarditis and pulmonary emphysema. (*b*) *Local stasis* has scarcely any part in the renal pathology (Cohnheim). Berkmann (1859) described renal stasis and thrombosis of the renal veins occurring in the new-born or in nurslings after severe diarrhœa; when unilateral there is renal pain, great albuminuria, some hæmaturia, swelling of the kidney and decreased urine; most cases of bilateral thrombosis die from anuria and uræmia. Thrombosis of the cava and pressure by large tumors may cause passive congestion in the renal veins.

Pathology.—Anatomical changes result from slowing of the arterial flow, lessened arterial pressure and to a less degree from venous hyperæmia, which changes alter the integrity of the secreting renal structures, notably the glomeruli. In experiments, arterial insufficiency causes initial glomerular alteration, while venous obstruction causes primary congestion in the medullary portion.

Early Changes.—The kidney is swollen, dark red and firm and its stellate veins are prominent. On section it expands beyond its close-fitting capsule, which is readily stripped off. The organ is very vascular and may drip blood. The glomeruli are seen as dark red points and the medullary bloodvessels are clearly defined. Microscopically the conspicuous changes are hyperæmia, occasionally with punctate hemorrhages, fatty and cloudy degeneration of the epithelium of the tufts and convoluted tubules, escape of albumin into them, forming hyaline casts in the tubules and finally some thickening of the vessels.

Late Changes.—Prolonged stasis leads to greater atrophy of the secreting structures and to increase of connective tissue; this "cyanotic induration" has been rather overdrawn in clinical descriptions, but may result from extreme and protracted stasis. The kidney be-

comes pale and firm, the cortex shrinks, the capsule may become somewhat adherent and connective tissue develops in the medulla. Microscopically, fibrillar connective tissue, thickening of Bowman's capsule and epithelial degeneration are found.

Symptoms.—Aside from the causal heart or other lesion and the signs of stasis in other parts the urinary findings are of chief interest. The urine is decreased and its amount follows directly the heart's activity. It is acid, dark, concentrated (specific gravity 1,025 to 1,030) and it has a lateritious sediment of urates and often of uric acid crystals. Albumin and globulin are present, usually in moderate amounts. Microscopically a few leukocytes, epithelial cells and hyaline casts are found; granular casts are seldom found and should arouse suspicion of nephritis; red cells almost always indicate nephritis or infarction rather than congestion.

Diagnosis.—The urinary findings are significant in association with the causal lesion; œdema begins in the legs; the nutmeg liver, dyspnœa, cyanosis and pulmonary stasis are present. The percentage of albumin runs parallel with the strength of the heart, increasing with its failure or decreasing if digitalis or strychnine improve its action. If cardiants increase the urine and if abundant albumin and casts still remain there is probably coincident nephritis. (See page 764.)

The *treatment* is that of chronic valvular disease (*q. v.*).

II. Infarction—Embolism.—Though infarction may rarely occur from local disease of the renal arteries, acute or chronic valvular disease of the left heart is its predisposing cause. Sixty-six to 75 per cent. of all embolisms are renal. Stoppage of a renal terminal artery causes the well-known wedge-shaped anæmic infarct in twenty-four to thirty-six hours; the local ischæmia causes fatty degeneration, necrosis and a finely granular transudation into the connective tissue and glomeruli. After a while fibrous substitution occurs with ultimate shrinkage and deformity. After repeated infarctions the irregular "embolic contracted kidney" is seen. As in other infarctions, the process may be innocent (mechanical) or less commonly suppurative.

Symptoms.—In most cases there are no symptoms. Exceptionally a diagnosis is possible, based on (1) the valvular lesion; (2) sudden onset with fever, chills and vomiting; (3) lumbar pain and tenderness and (4) hæmaturia and albuminuria.

There is no *treatment.*

AMYLOID DEGENERATION OF THE KIDNEY.

Etiology.—The etiology of amyloid kidney is that of amyloid liver (*q. v.*); its three principal causes are *ulcerative tuberculosis, protracted suppuration* and *inveterate syphilis.* The most plausible theory is that toxins produce, either in the blood or in the organ involved, an albumin which is deposited in and about the bloodvessels. Amyloid kidney was first described by Rokitansky (1842) and its symptomatology was developed by Wilkes, Todd and Traube.

Pathology.—Amyloidosis seldom occurs in the kidneys without associated changes in the liver and spleen. To the naked eye the kidneys generally appear *enlarged,* weighing together even as much as 26 ounces (870 gm., Johnson); when amyloid infiltration is slight, there may be no alteration in size or weight; in some cases the kidneys are large from coincident chronic parenchymatous or small from interstitial nephritis. The capsule strips readily, showing a pale, smooth surface. On section the *cortex is enlarged,* glistening, waxy and pale and its *edge translucent;* the *glomeruli stand out* as *glistening points* and become brown when treated with Lugol's solution. Microscopically the amyloid substance can be seen clearly; it is so named because of its starch-like reaction, though chemically it is an albuminoid. It is either deposited from the blood in the small vessels or more probably formed *in situ,* whence it is found especially in the glomeruli, then in their afferent vessels and in the straight vessels of the medulla. The renal interstitium is involved secondarily and the epithelial cells never suffer primarily, though the *membrana propria* may be affected and the cells may suffer secondary compression, degeneration and atrophy. As the changes indicate, amyloid disease cannot be classified with Bright's disease, though their association is not uncommon (*v. i.*).

Symptoms.—Its symptoms are variable. (*a*) *Urinary symptoms* are exceptionally absent (Rosenstein and Litten). The urine is generally *limpid and increased,* but may be decreased when amyloid degeneration is associated with parenchymatous or greatly increased when associated with interstitial nephritis; some writers place undue importance upon variability in its amount. The specific gravity is 1,010 to 1,015 or higher in syphilitic cases. *Albuminuria* is *usual,* being absent only in those exceptional cases which invade the vasa recta and evade the glomerular capillaries; albuminuria results from increased permeability of the glomerular vessels and may amount to 1 or 2 per cent.; Senator has remarked on the frequently large amount of globulin in the urine. There is little change in the solids; indicanuria is not infrequent. Microscopically hyaline casts may be present and perhaps a few leukocytes and epithelia, but the sediment is scanty in most cases; the so-called waxy or amyloid casts found in severe nephritides have no relation to amyloidosis of the kidney. (*b*) Cardiac hypertrophy, uræmia and retinitis do not occur in renal amyloidosis, except when it is associated with nephritis. (*c*) Amyloid degeneration of both the *spleen and liver* and sometimes the alimentary tract is associated in 66 per cent. of cases; these and the causal disease are necessary for diagnosis. (*d*) *Œdema* and *anæmia,* which is almost cachexia, are usual in diffuse amyloidosis. Hydrops of the serous sacs, especially *ascites,* is less common than œdema and usually occurs as a late or terminal event. *Differentiation* (*v.* p. 764).

Prognosis.—Amyloid disease is incurable, though light grades may recede. Amyloid of the kidneys in most cases runs its course in a year or less, particularly when associated with parenchymatous ne-

phritis. Cases may endure for several, even fifteen years, but these are generally complicated by contracted kidney.

Treatment.—Treatment consists chiefly of prophylaxis; with modern surgical treatment amyloid disease is less common than formerly. Early treatment of tuberculosis and syphilis decreases the percentage of amyloidosis; for the developed disease there is no treatment.

MALFORMATIONS OF THE KIDNEY.

I. Malformations and Structural Anomalies of the Kidney.—To malformations and structural anomalies in general but little clinical importance is attached. Complete absence or rudimentary development of both kidneys occurs only in unviable infants.

One kidney, usually the left, may be *absent or rudimentary,* whereon the normal kidney vicariously hypertrophies; the enlargement may be mistaken for malignant disease, whence its removal is fatal. Before operation cystoscopy is imperative in doubtful cases to determine the presence of two ureters or a preliminary incision should be made to ascertain that there are two kidneys. Two ureters may come from one kidney. Ballowitz collected 184 cases in which one kidney was lacking; 61 per cent. occurred in males and in 63 per cent. the left kidney was lacking. According to Gérard, unilateral absence of one kidney is usually associated with unilateral malformation of the internal genitalia, but this is not true in unilateral renal atrophy or in cases where the two kidneys fuse.

Double kidney and deformation from lacing or scoliosis, are of pathological interest only; 15 cases of *supernumerary* kidneys are recorded (Graser) and in two cases there were four kidneys.

The "*horse-shoe*" kidney (*ren arcuatus*) due to the fusion of both kidneys, is fairly common. Errors in diagnosis are common, especially if the kidney is dislocated and are inevitable if the renal pelvis is inflamed or dilated. The *ren arcuatus* has been mistaken for a tumor and extirpated, obviously with fatal results.

II. Movable Kidney.—*Congenital dislocation* occurs twice as frequently in males as in females and is usually left-sided, in which respects it differs from the acquired form. It frequently coincides with congenital anomalies of the genitalia and intestines and is of more anatomical than clinical interest. The kidneys may lie one upon the other (dystopia renum cruciata), of which Chatelin could collect but 15 cases. The kidney may lapse into the pelvis. The ureters are sometimes quite short.

Acquired dislocation, described by Rayer nearly a century ago, is much more common and important. It is called movable or floating kidney, ren mobilis s. migrans, ectopia renis acquisita or nephroptosis.

Etiology.—(*a*) *Sex;* 88 per cent. occur in *women,* as shown in Kuttner's collection of 667 cases; Dietl claimed that women constituted 99 per cent. of the cases. (*b*) *Age.* Most cases occur between the twentieth and fiftieth years. Comby reports 18 and Abt 5 cases

DIFFERENTIAL TABLE FOR THE DIAGNOSIS OF THE DIFFUSE DISEASES OF THE KIDNEYS. (MODIFIED FROM LEUBE.)

		Passive Congestion.	Acute Nephritis.	Chronic Nephritis.			Amyloid Kidney.
				Chronic Parenchymatous Nephritis.	Secondary Contracted Kidney.	Genuine Chronic Interstitial Nephritis.	
Etiology.		General stasis (heart, liver, kidney) or local stasis (thrombosis renal veins, cava, etc.).	1. Cold, exposure. 2. Poisons, *e. g.*, cantharides. 3. Acute infections, scarlatina, sepsis, pneumonia.	1. From acute nephritis. 2. Cold and moisture. 3. Toxic. 4. Toxæmic, phthisis, malaria. 5. Heart disease.	Secondary contraction may follow, but even then the kidney remains large.	Gout, alcoholism, diabetes, plumbism, syphilis, arteriosclerosis.	1. Protracted suppuration. 2. Old tuberculosis. 3. Inveterate syphilis.
Pathology.		Kidney, larger (may shrink), cyanotic, fatty, cloudy change, infarcts.	Some swelling, exudation of cells and serum; glomerular, epithelial and interstitial changes.	1. Large white kidney. 2. Large red (variegated) kidney. Fatty changes in tubules. Some connective change, especially in 2.	Contractions less than in genuine interstitial form, less cysts, larger prominences, larger granulations, and more yellow, and smooth.	Very small, atrophied, indurated kidney with fibrous hyperplasia, epithelial atrophy, adherent capsule, small granulations, etc.	Large, homogeneous waxy, vascular infiltration.
Urine	*Quantity.*	Scanty; varying with heart's activity, whence prognosis from amount, and from action of digitalis.	Decreased (glomerular changes), suppression even.	Decreased (tubular and glomerular changes); 200 to 1,000 c.c.	Somewhat increased, 1,500 to 2,000 c.c.; at least more than normal.	Very abundant, polyuria, pollakiuria, usually 2–3,000 c.c. (rarely 4–5,000).	Normal or increased; variable.
	Color.	Dark red.	Turbid (urates or blood) — pale red to dark red.	Opaque; "meat water" appearance; urates suspended in albuminous urine.	Moderately clear.	Pale, clear.	Clear yellow.
	Sp. Gr.	High; 1,025–30.	High; 1,025–30–40.	Normal or somewhat higher, 1,020.	Somewhat under normal.	Low, 1,010 being the average.	Normal or less.
	Albumin.	Due to arterial anæmia, moderate, 0.5 per cent., varying with condition of heart.	Much; ¼ to 1 per cent.	Much; 1½ to 3 per cent. (very rarely absent).	Considerable.	0.5 per mille, slight, intermittent, latent or long absent.	Usually abundant.
	Sediment.	Moderate, urates, hyaline casts, few red cells.	Great; much urates, red and white cells, blood, epithelial, waxy casts.	Great; red and white cells; casts, all kinds, granular and fatty especially; fatty cells.	Moderate—many casts of all types.	Scanty; few hyaline (granular) casts.	Usually none, unless co-incident nephritis.

DIFFERENTIAL TABLE FOR THE DIAGNOSIS OF THE DIFFUSE DISEASES OF THE KIDNEYS.—*Continued.*

Urine Continued	*Solids.*	Practically normal, urea decreased, chlorides normal.	Decreased chlorides, urea, phosphates.	Decreased.	Markedly decreased.	Decidedly decreased urea, ammonia, uric acid, chlorides, phosphates.	Normal.
	Blood.	Absent, save for stray red cells; blood from infarct.	Much.	Usually some; more in large red kidney (chronic hemorrhagic nephritis).	Little or none.	Absent.	None.
Heart Hypertrophy.		Changes depending on valvular lesion, etc., present.	None as rule (very rarely early hypertrophy).	Occasional, eccentric hypertrophy; especially in diffuse nephritis.	Usually present.	Almost invariable (concentric hypertrophy).	None in pure cases.
Hydrops.		From stasis; stationary, mechanical, in feet first then abdomen, pleuræ, etc.	Usually marked, rarely absent; mostly in scarlatinal and exposure nephritis; from vessel changes.	Usually extreme and obstinate (in skin, serous cavities and viscera); marked vascular change; somewhat less in large red kidney.	Moderate; less than in large white kidney but more than in true contracted kidney.	Absent, unless in stage of cardiac incompetence.	Marked, from cachexia or vessel infiltration.
Anæmia.		Often present when is usually associated with cyanosis.	May be some.	*Very* frequent; a cardinal symptom with the urinary findings and hydrops.	Moderate.	Usually none (except in young subjects).	Marked.
Uræmia.		Absent.	Frequent; especially when due to cold and scarlatina.	Moderately frequent.	Frequent.	Most frequent.	Never.
Visceral Symptoms.		Lung congestion, stasis liver, infarcts spleen, etc., dyspnœa.	Pulse somewhat tense; often latent; fever due to other causes.	Anæmia; retinitis; bronchitis. Fairly frequent.	Retinitis, bronchitis, etc.	Retinitis and arteriosclerosis more than in any other forms.	No retinitis. Amyloid degeneration of liver, spleen and alimentary tract.
Course.		Depends on age and extent of lesion; may be long.	Average duration 1½ weeks; usually wholly disappears if patient lives; sometimes becomes chronic (scarlatina).	½ to 1½ years (may be longer in chronic hemorrhagic nephritis).	Slower than in large white kidney, but faster than in the genuine contracted kidney; 1½ to 3 years.	Much longer than in other nephritides, 5 to 10 years.	Depends on causal disease.
Cause of Death.		Cardiac dilatation, embolism, coronary disease.	Death from causal disease; uræmia, hydrops (glottis), œdema of lungs, inflammation (pericardium, pleura, lung, etc.).	Uræmia, œdema glottis, hemorrhages, marasmus, sepsis, acute exacerbations, but mostly (visceral) inflammations.	Uræmia, cardiac insufficiency, myocarditis, hemorrhage (cerebral), retinitis, inflammations (pleural, pericardial, pneumonic, cutaneous) very common.		Causal disease, hydrops, cachexia.

in children. (*c*) The *actual cause* is disputed. The kidney is supported by the fat around it, the peritoneum in front of it and by its own bloodvessels. Among the alleged causes are: atrophy of the perirenal fat; the presence of a *mesonephron* (Gérard) surrounding the kidney to a variable extent; *weakening of the abdominal walls* by pregnancy and other causes; alterations in the intra-abdominal pressure by *malpositions of the uterus, e. g.,* prolapse (Landau); pressure exerted by *corsets* on the liver (Cruveilhier); *congenital laxness of the supporting structures,* in connection with which Stiller claims that the tenth rib is usually "floating" like the eleventh and twelfth ribs (costa fluctuans decima); tumors of the kidney, which drag it downward; and trauma or sudden efforts. Several of these causes are also thought to produce enteroptosis, in connection with which floating kidney is very frequently observed.

Symptoms.—Symptoms are absent in many, probably in most cases; they may date from the time when the physician incautiously remarks upon the presence of "a floating kidney." There are *two main symptoms,* the subjective pain and the objective-tumor.

1. Pain.—Pain may be merely a dragging sensation or may amount to severe renal colic, which is said to result from stretching of the renal nerves; it may be lumbar, abdominal or may shoot toward the heart, thigh or sciatic region; it may occasion intercostal neuralgia, even of the opposite side of the body. Pain may be constant or intermittent, mild or severe or like a crisis (*v. i.*).

2. Objective Kidney Findings.—On bimanual examination with one hand over the kidney in front and the other pushing it up from behind, the findings vary; (*a*) frequently the *kidney* is not painful or tender, but *palpable,* which condition has no significance, especially in women; (*b*) the kidney may be *movable,* descending on deep inspiration, maybe to the navel, where it can be retained by pressure during expiration; movable kidney is found in 20 per cent. of women (Tuffier); (*c*) the kidney may be *floating* (the most extreme type), in which it may fall or wander to the lower abdomen or pelvis. In 76 *per cent. of cases the right kidney* is involved, in 11 per cent. the left and in 13 per cent. both kidneys (Kuttner); the right kidney has longer vessels, but the left renal vein is more firmly connected with the suprarenal vein and the pancreas. When the kidney is grasped a sickening sensation sometimes results as when the testicle is compressed. The *tumor is smooth* and is usually recognized by its shape. In rare cases the pulsating renal vessels are felt (v. Frerichs and Eichhorst). If not clearly felt when the patient is lying on the back, palpation is made in the standing posture or with the patient lying on the left side. Little confidence can be placed in percussion along the spine, though occasionally tympany can be found over the normally dull area along the spine occupied by the kidney; the renal dulness merges with that of the liver and spleen above and extends outward 2.5 inches on the left and 3.5 inches on the right side. In thin subjects the sinking in of the back and the tumor in front may some-

times be clearly seen. Rarely the kidney may be found in a hernial sac.

3. Other Symptoms and Complications.—(*a*) The same train of *neurasthenic,* hysterical or hypochondriacal symptoms are present as in enteroptosis; Tuffier believes that these symptoms are causal rather than sequential. Nervous dyspepsia is particularly common. (*b*) Dietl (1864) described attacks of sudden severe pain and collapse known as *"Dietl's crises";* they are marked by fever, chill, vomiting, collapse, severe pain, sometimes with swelling and tenderness of the kidney, scanty urine containing blood, pus, uric acid and oxalates and in some instances peritonitic manifestations; Dietl's theory that the kidney became "incarcerated" between the connective tissue and peritoneum is now abandoned; Gilewski maintains that the ureter is twisted, causing acute renal retention and hydronephrosis and in one of Sutton's cases the ureter was found twisted three times. Landau holds that the renal vessels suffer torsion and Mosler explains the crises by acute nephritis, ureteral stoppage or peritonitis. (*c*) *Dilatation of the stomach* is neither causal nor sequential. It is only a coördinate and not at all a common coincidence. (*d*) *Dysmenorrhœa,* constipation and enteroptosis are common complications. Colitis mucosa is reported by Morris. Compression or actual stenosis of the intestine, icterus, abdominal aneurysm by tugging through the renal vessels upon the aorta, compression of the cava inferior and adhesions to the gall-bladder are rare (and questionable) sequences. Albuminuria may occur periodically, on palpation of the kidney or when the patient stands (orthostatic type).

Diagnosis.—The diagnosis is rarely difficult. The finding on bimanual examination of the sensitive kidney-shaped movable tumor is more important than the inconstant subjective or ambiguous neurasthenic symptoms. Severe pain may be confused with a tabetic crisis, appendicitis or the passage of gall- or kidney-stones. The kidney may possibly be mistaken for some other movable tumor, as omental, gastric, intestinal and gall-bladder tumors or corset liver. Morris reported cases in which an enlarged gall-bladder has been mistaken for movable kidney; both tumors are movable, may induce icterus or dyspeptic disturbances and both may be attended by colic and a dull (or resonant) note over the tumor. Doubtful cases are determined by repeated examinations, perhaps under anæsthesia. Particular reserve is necessary in the diagnosis of left-sided floating kidney.

Prognosis.—Death is an extremely rare occurrence, though it may result from "incarceration" and peritonitis.

Treatment.—The patient should never be told of a movable or floating kidney, because the symptoms, chiefly the neurasthenic, may date from the diagnosis. (*a*) A *rest cure* may develop adipose tissue and relieve the nervous symptoms. (*b*) A well-fitted *bandage,* with padding to prevent renal descent, is excellent in many cases. (*c*) In obstinate and extreme cases, *nephrorrhaphy* or nephropexy, as

recommended by Hahn, is indicated. According to Czerny and Giess, 82 to 85 per cent. are cured. Tuffier gives results of 163 cases as follows: absolute recovery, 53 per cent.; improvement, 30 per cent.; temporary improvement, 5 per cent.; and failure, 12 per cent. Frank gives the results of Hahn's clinic as 75 per cent. complete recovery, 15 per cent. decidedly improved and 10 per cent. failures; the mortality was under 1 per cent. Nephrectomy is seldom indicated. (*d*) The crises are treated by morphine, reposition and later by operation.

ALBUMINURIA AND OTHER ANOMALIES OF RENAL SECRETION.

I. Albuminuria.—Albuminuria, discovered by Cotugno in 1770, results (*a*) from leakage of albumin through the glomeruli and tubules (*albuminuria vera, renalis*) or (*b*) from blood, pus, semen or fluid of tumors (*albuminuria spuria*). The genuine form will be first considered and the spurious or accidental form later under pyuria and hæmaturia.

The most common albumin is serum albumin (or simply albumin) with which serum globulin is frequently associated.

Etiology.—(*a*) Its incidence in the *diffuse renal diseases,* the nephritides, amyloidosis and passive congestion has already been discussed. (*b*) *Blood alterations* are frequent causes; (i) blood diseases proper, as anæmia, leukæmia, pseudoleukæmia, pernicious anæmia, scurvy or purpura in which the poverty of the blood or toxins induce renal degeneration; (ii) toxæmic states, as the acute and chronic infections, in which "*febrile albuminuria*" (Gerhardt) evidences the renal degeneration; this condition borders very closely on or merges into mild cases of acute nephritis; (iii) diabetes, icterus and poisoning by lead, mercury, ether, etc. (*c*) *Nervous maladies* possibly act by lowering the local blood pressure, as epilepsy, exophthalmic goitre, migraine and the psychoses. (*d*) Affections of the *digestive tract,* as intestinal incarceration, hepatic diseases and lead colic may produce albuminuria. (*e*) In the *new-born* it was first noted by Virchow. It is almost regular in the first week or ten days of life (Ruge, Martin) and occurs with hyaline casts, urates and epithelium. It is hardly explained by change of metabolism or of circulation or by uric acid infarcts. Ribbert holds that it is due to imperfect development of the glomeruli. (*f*) *Pregnancy;* it occurs in 40 per cent. of cases in the last few months of gestation (from increased tax on the kidneys and from pressure) and in the same percentage after delivery (from effort and strain). (*g*) Is there a "*physiological albuminuria*"? Various types are described, as Pavy's cyclical albuminuria, which appears and disappears; intermittent albuminuria; adolescent albuminuria, in young anæmic subjects, often with vasomotor disturbances; orthostatic albuminuria, also termed postural or orthotic, which appears only when the subject stands. Some bring under this caption albuminuria after heavy eating, severe exer-

cise, coitus, mental strain or cold baths. Senator, Bradford and Grainger Stewart, whose names alone carry weight, insist that there is a physiological albuminuria, in which signs of nephritis, as casts or cardiovascular changes, are absent. The author regards every case of albuminuria as pathological; its cause may be an insidiously developing nephritis, a receding nephritis, stasis or maybe "constitutional," as Martius calls it, but it is always a sign of glomerular or tubular lesion (see PROGNOSIS). The amount of albumin in this type rarely exceeds 0.5 per mille (Senator).

Detection.—The urine must be clear and acid; if turbid it should be filtered and if it then remains turbid it should be cleared by shaking with magnesia; it must be acidulated, if necessary, with acetic acid; if very concentrated, dilution with distilled water is advisable.

1. BOILING TEST.—A test-tube, two-thirds filled with urine, is held by its lower part and the upper layers are boiled. Albumin produces clouding, which may also result from phosphates and carbonates (dissolving when nitric acid is added), balsams (dissolving in alcohol) and, it is said, urotropin and formaldehyde. Nitric acid should not be added before boiling, for a soluble acid-albumin may result.

2. NITRIC ACID OR HELLER'S TEST.—Urine, somewhat diluted, is placed in a test-tube or conical glass and nitric acid is run under it by a pipette. Albumin, globulin, mucin and albumose are precipitated at once or after a few minutes at the contact point; albumose dissolves by heating, the others do not. These tests are very satisfactory.

3. FERROCYANIDE TEST.—To a test-tube half full of urine 10 drops of acetic acid are added, which may precipitate uric acid, urates, oleoresins or nucleo-albumin (mucin). These are filtered out and one dram (5 c.c.) of a 10 per cent. solution of potassium ferrocyanide is added. A precipitate indicates albumin, globulin or albumose, which latter alone dissolves on heating.

4. ACETIC ACID AND SALT TEST.—Adding to the urine *acetic acid* to an acid reaction and then an equal volume of *saturated sodium chloride* solution precipitates albumin and albumose, but the latter dissolves on heating. The above tests detect one part in 10,000.

5. SPIEGLER'S TEST.—This should be used if more delicate tests seem necessary. Nucleo-albumin (mucin) is removed by adding 15 drops of dilute acetic acid and filtering. To the urine is added a quantity of Spiegler's reagent (bichloride mercury 8, tartaric acid 4, glycerine 20 and distilled water 200) when albumin or albumose develops at the contact zone. One part in 350,000 may be detected. Jolles' modification: bichloride 10, succinic acid 20, sodium chloride 20 and 100 parts of distilled water.

Globulin occurs chiefly in renal congestion, acute and chronic nephritis and amyloid degeneration. It dissolves in an excess of acetic acid. It is precipitated by boiling. Flocculi of globulin are thrown down, when 2 drops of dilute acetic acid are added to 1.6 ounces of filtered urine diluted 10 times with distilled water.

Nucleo-albumin (formerly called *mucin*) occurs in acute desquamative nephritis, icterus, poisonings causing necrosis of the renal cells and regularly in catarrh of the urinary passages. It is precipitated by boiling and by acetic acid and unlike globulin, does not dissolve in an excess of acetic acid. Shreds of mucus, voided with colic, have been observed by von Jaksch and Baumüller; the condition seems analogous to colica mucosa and spasmodic asthma.

Peptone or albumose is of little clinical importance; the terms include albumins which do not coagulate by heat. When the urine is boiled the albumin is precipitated and filtered out. Albumose in the filtrate causes clouding and precipitation on cooling. What Kühne once called peptone is now termed albumose. (*a*) *Bence Jones' albumose* is rather a peculiar albumin than an albumose; described by him in 1848, albumosuria has also been called Kahler's disease. Muffat (1905) collected 38 cases. Its significance is uncertain; it has been found in osteomalacia and in multiple myeloma. It is precipitated by cold nitric acid, dissolves on heating and reappears on cooling. (*b*) Albumose may be found in various acute infections (meningitis. rheumatism, pneumonia), septicæmia, peritonitis, empyæma, etc. Albumin in the kidney, bladder or urinal may be converted by pepsin into albumose.

Diagnosis.—The diagnosis of albuminuria is easily determined by the tests enumerated; blood or pus may give albumin reactions (accidental or spurious albuminuria, *v.* hæmaturia and pyuria).

Prognosis and Treatment.—These are etiological. The outlook is not favorable, though not necessarily bad. In seemingly "physiological" cases, the condition of the heart must be watched and casts carefully looked for. In youth and adolescence albuminuria may be "physiological" but in middle and later life it is usually nephritic. Shattuck and Osler, however, hold that "albuminuria is more common apart from Bright's disease than is commonly supposed, that it increases with advancing years and that a small amount of albumin with finely granular casts of small diameter in those over fifty years of age is of small importance." Life insurance companies are suspicious of "physiological" albuminuria, for figures show that it entails double the average death-rate. Albuminuria is said to result solely from high arterial tension, but the author regards these cases as incipient arteriosclerosis or contracted kidney. Hawkins reported a case in which albuminuria lasted, without any unhappy symptoms, for forty-three years after Bright had given a fatal prognosis.

II. Hæmaturia.—Etiology.—Like albuminuria, blood in the urine (hæmaturia) is only a symptom and is observed in many diverse conditions. (*a*) It is *most frequently renal.* (i) *General conditions* may cause it, as intoxications (by quinine, carbolic acid, turpentine or salicylic acid), acute infections (of which malaria is the most common, acute exanthemata, particularly the hemorrhagic types) or the blood diseases (purpura, scurvy, hæmophilia, leukæmia and the anæmias). (ii) Other causes are *local renal conditions,* as trauma,

vascular maladies (thrombosis of the renal veins, infarct, aneurysm, varicose veins, arteriosclerosis and rarely passive congestion); inflammation (acute, chronic, hemorrhagic and suppurative nephritis); rarely amyloidosis or parasites (distoma, filaria, nephrophages sanguinarius); tumors; tuberculosis; calculous ulceration and pyelitis; sometimes floating and cystic kidney, especially after bimanual examination. (iii) Hæmaturia may be *"idiopathic" or "essential."* In some very rare cases it occurs in "apparently healthy kidneys," to which phrase there has been a just objection. This condition has been called renal epistaxis (Gull), angioneurotic hæmaturia (Klemperer) and renal purpura (Senator). M. L. Harris collected 18 cases in which simple nephrotomy gave relief. In Eshner's series of 48 cases of unilateral renal hæmaturia nothing was found at operation in 33 per cent., floating kidney existed in 12.5 per cent., renal pelvic changes in 19 per cent. and diffuse renal disease in 24 per cent. (*b*) It may occur from *lesions in the lower urinary tract.* The chief types of these lesions are stone in the ureter or bladder; ruptured varices anywhere in the urinary tract; stasis from liver cirrhosis or thrombosis of the iliac veins; inflammation (pyelitis, ureteritis, cystitis, prostatitis or urethritis, sometimes called "Russian gonorrhœa"); and tuberculous, malignant or other ulcerations.

Symptoms and Diagnosis.—Hemorrhage may be macro- or microscopic. The urine may be clear or turbid, red or smoke-colored. It is usually acid, but may be alkaline when there is copious vesical hemorrhage. The color is generally brighter when the urine is alkaline. Confusion with concentrated, icteric, carbolic, fuchsin, hæmatoxylin and senna urines is possible. The presence of blood is positively determined (*a*) by the *microscope;* the red cells remain normal for a long time in acid urine, but may become pale in dilute and mulberry-form in concentrated urine; (*b*) by the *spectroscope;* if no red cells are present with positive spectroscopic findings it indicates hæmoglobinuria (*v. i.*); and (*c*) by *chemical tests,* as Heller's (boiling with caustic potash, which throws down a rusty phosphate and hæmatin precipitate), the Almén guaiac test (acidulating the urine and adding old turpentine and fresh guaiac) which gives a blue reaction (also produced by pus) and Teichmann's test (treating the sediment with sodium chloride and glacial acetic acid, slightly heating and cooling, when hæmin crystals are deposited.

Diagnosis of the origin and cause of hæmaturia is often most difficult. (*a*) In cases of *renal* origin, the blood is intimately mixed with the urine, as much with that first voided as with the last. The color is often that of water in which raw meat has soaked ("beef water" color). Clots are infrequent in renal hæmaturia, save from trauma, tumors, aneurysm or varicosities. The red cells are seldom seen in rouleaux-form and, according to Gumprecht, are fragmented from action of the renal cells on the red disks; this does not occur in vesical hemorrhage. If there is some other renal lesion, as nephritis, there is more albumin than the red cells would account for, and albumin

is present after careful filtering; according to Goldberg, if there is albumin with a red cell count, by the hæmocytometer, of less than 3,000 per c.mm., the albuminuria is renal. Blood casts, granular casts and renal epithelium are indicative of renal origin. (*b*) Hæmaturia originating in the *pelvis and ureter* is more often marked by clots and possibly by caudate epithelium. (*c*) Blood from the *bladder* is more apt to clot and the last part voided is more bloody than the first. Other symptoms, as tenesmus, alkaline urine reaction and perhaps pieces of tumor, very often indicate a vesical origin. (*d*) *Urethral* hemorrhage stains most deeply the first urine voided. It must always be borne in mind that the presence of blood may be accidental, from menstrual or other contamination.

Treatment.—The treatment is etiological and can be but briefly considered here. Rest and sometimes morphine are indicated. Aside from the local treatment of vesical disease, urethritis and calculous pyelitis, tannic acid, ergotin, thyroid extract and calcium chloride āā gr. v. t. i. d. may check bleeding. Aromatic sulphuric acid ♏x, well diluted, is also given at three-hour intervals. Adrenalin solution 1 to 1,000 parts, given by the catheter, may stop vesical hemorrhage. In essential (idiopathic) renal hæmaturia, simple incision of the kidney is frequently efficacious.

III. Hæmoglobinuria.—This is hæmoglobin in the urine without hæmaturia. Though first described by Dressler (1854), most of the early accounts are English, from Harley, Dickinson, Gull and Pavy. There are two main groups, the toxic and the paroxysmal.

Etiology.—The *toxic form* may follow transfusion of blood, injections of glycerin, poisons, organic and inorganic, as potassium chlorate, phosphorus, mercury, quinine (in malarial cases only), carbolic acid, carbon monoxide; it may also result from burns, severe icterus, pernicious anæmia, scarlatina, acute nephritis, Winckel's disease in the new-born and æstivo-autumnal malaria. The *paroxysmal form,* of which Gilman Thompson recently collected 206 cases, occurs largely in England, France and Germany; 96 per cent. are males, chiefly between *thirty and forty years* of age. *Cold* seems to be the most important factor; exercise, excesses or menstruation may precipitate an attack. It is observed in horses after cold and exertion; hæmoglobinæmia and hæmoglobinuria appear, paresis of the hind legs develops and death results from disturbed circulation and respiration. In cattle and sheep epidemics occur in which Babes, Krogius and others have found a hæmatococcus. In the majority of human cases, hæmoglobinuria is preceded by hæmoglobinæmia, though genuine renal hæmoglobinuria (without hæmoglobinæmia) seems to occur in some cases of nephritis and from certain intestinal nematodes.

Symptoms.—Symptoms occur only after destruction of at least one-sixth of the blood. After a chill, fever, lumbar pain which radiates to the legs, cyanosis, oppression and gastric symptoms, the urinary findings appear; the urine is dark and contains clumps of blood pigment, casts and epithelium stained with hæmoglobin or methæmo-

globin, albumin and sometimes oxalate of lime crystals and bile pigments. In most cases the hæmoglobin is already altered to methæmoglobin, as shown by the spectroscope. There may be slight jaundice, œdema or urticaria. The blood shows increased coagulability and diminution in the number and redness of the red cells, which do not form in rouleaux. There may be swelling of the liver and spleen from hæmoglobin deposits in them. The paroxysm lasts one-half to several hours and in the interval the general health is fairly good. The course is most chronic. No death is known from the paroxysm itself and very rarely from the disease.

Treatment.—Prophylaxis in regard to colds and exertion may modify the attacks. Chvostek was able to abort attacks by giving nitrite of amyl inhalations. Warmth may modify the seizure. Calcium chloride and thyroid extract have been recommended.

IV. Pyuria.—Etiology.—(*a*) Pus may originate from *pyelitis* or pyelonephritis. It may occur intermittently, as in pyonephrosis or continuously, as in calculous or tuberculous inflammation. The urine is usually acid in tubercle and colon infections; it is acid in proteus and in some infections ascending from the bladder; the staphylococcus may render the urine less acid than normal or alkaline (*v. i.* pyelitis). (*b*) Pyuria may result from *cystitis;* the urine may be acid or alkaline and contains ropy mucus and triple phosphates. Pus is seen especially in the last urine voided. (*c*) *Urethral* pyuria, usually gonorrhœal, is greatest in the urine first evacuated. (*d*) *Accidental* pyuria occurs from leucorrhœal contamination and is easily excluded by catheterization. Under this heading may be brought pyuria from rupture into the urinary tract of appendicular, pelvic or psoas abscess; much pus is voided at first, but it gradually lessens and disappears.

Symptoms.—The urine in pyuria is yellow-white, turbid and has a ropy sediment becoming gelatinous when caustic potash is added. The *reaction* is often ammoniacal, due to germs decomposing the urea, as the proteus and staphylococci. Gas may be voided with the urine (pneumaturia); this occurs particularly in enterovesical fistula and in diabetic cystitis. Alkalinity was formerly thought characteristic of cystitic, and acidity of pyelitic pyuria, but this distinction has been dropped. *Albumin* is found, the amount of which in simple pyuria corresponds with the number of pus cells. Albuminuria may be absent if there are not over 6–800 pus cells to the c.mm. It is less or may disappear when the urine is filtered. The question often arises whether the albuminuria is simply accidental or is combined with renal albuminuria, for instance, whether cystitis is coincident with contracted kidney. Besides consideration of the amount of urine, its specific gravity, cardiovascular findings and retinitis, besides washing out the bladder and ureteral catheterization, a study of the characteristics of the pus cells and their number is of great value. Senator has shown that leukocytes coming from the renal parenchyma in nephritis are mononuclear and those from suppuration are poly-

morphonuclear. Goldberg found that when (*a*) there is more than 1 part per mille of albumin in the clear urine, as determined by Esbach's method, with (*b*) 50,000 pus cells per c.mm., as determined by the hæmocytometer, the albumin is partly renal. De Lint altered Goldberg's method by gravimetric determination of the albumin; when there is one part per mille of albumin and less than 7,000 pus cells the albuminuria is (*a*) renal and (*b*) it results from the pus alone when there are more than 40,000 pus cells; no conclusion can be drawn if the pus cells number between 7,000 and 40,000 per c.mm.

Treatment.—The treatment is that of the fundamental tuberculosis, calculus, pyelitis, cystitis, etc. Phenylis salicylas and hexamethylenamina āā gr. x t. i. d. are excellent antiseptics for the urinary tract.

V. Chyluria.—The endemic or *tropical form* is found in Florida and South Carolina, Brazil, the Antilles, Zanzibar, India, Egypt and Japan. It is due to the filaria, first recognized by Bancroft and Leurs in the scrotum, by Wucherer in the urine and by Lewis in the blood. It enters the body by the drinking water in which mosquitoes have on their death liberated filaria embryos. The filaria plug the lymph vessels, which dilate and rupture in the kidneys, as proven by Mackenzie's autopsy. They are found in the blood at night (see Animal Parasites). The *non-tropical forms* are not understood and have rarely occurred in Europeans who have never been in the tropics; the rare autopsies have shown no marked renal alteration (Roberts).

The *urine* resembles chyle, is weakly acid or neutral in reaction, may present a *fatty, creamy surface* and contains 2 to 10 per cent. albumin (serum albumin, globulin or albumose), cholesterin, lecithin, fat and stearic and palmitic acids. Its specific gravity is normal. Sugar is seldom found. Microscopic findings are fat globules, leukocytes, sometimes red disks and the filaria parasites, usually in the fibrin clots, which sometimes occasion dysuria. Casts are never found. Chyluria is often intermittent; it occurs especially at night and may be initiated by exercise and changes of posture. In some cases there is considerable free fat in the blood (*lipæmia*). Its course is chronic, covering months to years. Recovery is the rule, though death may occur from anæmia and marasmus. *Treatment* is unsatisfactory. Potassium picronitrate gr. ⅙ t. i. d. is said to accelerate recovery. Prophylaxis regarding drinking water must be considered in the tropics.

VI. Lipuria.—Lipuria or adiposuria is symptomatic of (a) *lipæmia,* or fat in the blood. The clearest instance is after bone fractures, contusions of the liver or after delivery in which fat enters the blood from the pelvic cellular tissue. Fatty foods, as cod-liver oil; acute yellow atrophy; diabetes; diseases of the pancreas and various poisonings and tumors are possible causes. (*b*) It is also symptomatic of *renal lesions* (from fatty epithelial cells, degenerated tumors or fatty pus cells).

Under the *microscope* small fat globules are seen which stain black with osmic acid or red with the alcanna test. The fat may be saponi-

fied by alkalies, emulsified by albumins or dissolved by alcohol, ether, xylol or chloroform. Lipuria is often simulated by bacteria or phosphates or by oil from catheters, suppositories or greasy urinals.

VII. Phosphaturia.—Phosphoric acid is chiefly excreted in combination, mostly with sodium and potassium, forming the alkaline phosphates and with calcium and magnesium, forming the earthy phosphates. The sodium exceeds the potassium and the calcium is in excess of the magnesium salts. Phosphoric acid is chiefly derived from the food, but some is derived from decomposition of lecithin, nuclein and protagon. It averages thirty to forty-five grains daily. Amounts over one dram or alteration of the normal relation of the phosphorus to the nitrogen (17 or 20 to 100) are pathological. An excessive deposit which occurs in dyspepsia is not synonymous with an actual increase. Phosphaturia may be observed in neurasthenia, pulmonary tuberculosis, diabetes (largely from the diet), acute yellow atrophy, leukæmia and in anæmia. As much as two drams daily have been reported by Teissier and Ralfe, who termed the condition *diabetes phosphaticus.* Earthy phosphates may cloud the last part of the urine, which is mistaken by the laity for "spermatorrhœa." The addition of an acid rapidly clears the urine. The *diet,* recommended for this condition, consists of meat, fish, bread, potatoes and apples. Coffee, nux vomica, arsenic, cod-liver oil and ammonium benzoate (gr. x to xxx t. i. d.) are also given.

VIII. Lithuria.—No subject has occasioned more clinical absurdities than uric acid. It normally bears to urea a ratio of 1 to 70. Futcher has found a ratio in gout of 1 to 300 or 500 (*v.* Gout). It is deposited in larger amounts than normal when the urine is very acid and the mineral salts or pigments are decreased. An increased precipitation is far more common than an absolutely increased excretion of uric acid. The sediment is clearly seen as red granules resembling red pepper. Uric acid combines chiefly with ammonium and sodium and less with potassium, lithium and calcium, which form *urates* (*sedimentum lateritium*) of the pink or "brick-dust" color so frequently seen in febrile urine and urine of high specific gravity or of excessive acidity. They are soluble when heated and precipitate on cooling.

IX. Oxaluria.—Oxalates first detected by Donne (1838) in the normal urine amount to less than 10 mg. daily. Quantities larger than this daily are pathological. Helen Baldwin decided that they are not formed during normal metabolism and are taken in with the food. The actual conditions of oxaluria are in doubt. It has been observed in gout, emphysema, dyspepsias (especially in hyperacidity and increased intestinal putrefaction), neurasthenia, icterus and diabetes. Oxalates occur as octahedral and dumb-bell crystals, which, according to Klemperer, occur in 50 per cent. of renal calculi. Cantani maintains that oxaluria is associated with *nervous depression, irritability,* hypochondriasis, *lumbar pain,* sweats and nervous *dyspepsia,* though the consensus of opinion is against any definite clinical

picture or diathesis. Klemperer advised a diet of meat, fat and vegetables (except spinach and cabbage); he excludes milk and eggs and administers magnesium sulphate ʒss once daily. Nitromuriatic acid ♏ij with tincture of nux vomica ♏x is an old and excellent empirical treatment.

X. Indicanuria.—Potassium indoxylsulphate is present in small amounts in normal urine. It is colorless as a rule, but may be voided blue or violet in alkaline urine. When acid or some oxidizing substance is added it decomposes and frees indigo. The steps in its production are as follows: the action of the pancreatic juice on albumins produces indol, which is absorbed, oxidized in the tissues to indoxyl and unites with potassium sulphate to form potassium indoxylsulphate. Its absence in some pancreatic diseases has been discussed. It has been rarely found in urinary calculi. It is increased in cachexiæ in which albuminous substances are decomposed, in intestinal obstruction, particularly of the small bowel and in carcinoma, suppuration and peritonitis. Boiling the urine and adding nitric acid, as in the ring test for albumin, gives a blue ring; adding half a dram (2 c.c.) of chloroform and shaking brings down the violet or blue-stained chloroform.

XI. Other Anomalies.—*Alkaptonuria* was discovered by Bädecker. It is a crystalline, aromatic body, probably derived from proteid decomposition, whose chief clinical significance is that (*a*) it reduces Fehling's solution, thereby simulating glycosuria, though it is negative to polarization and fermentation tests; and (*b*) it imparts to the urine a dark brown color, usually apparent only after it has stood or after caustic potash is added (alkaptonuria by derivation means urine which "absorbs" an "alkali" freely). Adding a drop of a very dilute solution of ferric chloride causes a dull green color which at once disappears. It is said to be homogentisic or uroleucinic acid. Sometimes it occurs as a familial affection. Garrod collected 47 cases, 73 per cent. of which were males; in 32 congenital cases, 19 of them occurred in 7 families. Virchow (1866) described a case of *ochronosis,* a discoloration of the cartilages and ligaments. Osler described 2 cases associated with alkaptonuria, making in all 11 cases in the literature; it may occur with pigmentation of the face, chronic arthritis and deformation of the lumbar vertebræ. Chronic phenol poisoning is a possible cause.

Hydrochinon, from carbolic acid poisoning, may cause a somewhat similar discoloration.

Hæmatoporphyrinuria, discovered by Hoppe-Seyler, imparts a dark color to the urine. It occurs in pulmonary tuberculosis, rheumatism, intestinal hemorrhage, sulphonal- and lead-poisoning, etc. Hæmatoporphyrin resembles hæmatin, save that it contains no iron.

Acetonuria is of clinical importance, chiefly in diabetes (*q. v.*).

For fuller details the reader may consult works on clinical diagnosis, such as the recent treatises by Simon, Emerson and others.

PYELITIS—PYELONEPHRITIS—SUPPURATIVE NEPHRITIS.

I. Pyelitis.—Pyelitis and pyelonephritis are inflammations of the pelvis, and pelvis and kidney, respectively. They were first described by Rayer.

Etiology.—Pyelitis is invariably *mycotic.* The kidneys usually eliminate bacteria without injury to their structure, but when the general vitality or the local resistance of the kidneys is lowered inflammation of the pelvis or kidney may result. Various bacteria may be found, including the tubercle bacillus (*v. i.,* SUPPURATIVE NEPHRITIS). (*a*) *Foreign bodies,* as large, hard or sharp calculi, less often cancer or tubercle, and still more rarely parasites, promote infection. (*b*) *Ascending infections* from the lower urinary tract are peculiarly dangerous when associated with urinary obstruction, as urethral stricture or hypertrophied prostate. When the bacteria which produce cystitis cause decomposition of urea, the ammonia evolved acts as an additional eroding factor. According to Rosving, certain streptococci act in this way, but not the tubercle or colon organisms. Infection usually ascends from cystitis along the ureters, producing ureteritis, then pyelitis and perhaps a pyelonephritis, but it may reach the kidney along the lymph vessels, the microbes entering through erosions in the vesical mucosa. James Goodhart has shown that 50 per cent. of cases with obstruction to the flow of urine die of renal suppuration. (*c*) Infection may be *hæmatogenous,* as in typhoid, the exanthemata or intestinal catarrhs of infancy. (*d*) Less common factors include *traumatism,* extension by *contiguity* from neighboring inflammations, as pyelonephritis (pyelonephritis is usually an ascending process, in which the kidney is secondarily involved, and only rarely is it a process descending from the kidney to the pelvis), toxic factors, acrid, irritating diuretics, such as turpentine, and hemorrhagic measles or scurvy; cold, venous stasis and diabetes are somewhat predisposing factors; arteriosclerosis induces the form of pyelitis described by Ollivier, attended by hemorrhage and the formation of fibrin coagula. Pyelitis is most prevalent in women.

Pathology.—Pyelitis may be acute or chronic; solitary or combined with cystitis; catarrhal, suppurative or ulcerative; unilateral or less often bilateral. Acute catarrhal forms present redness, œdema, desquamation, formation of mucopus and minute ecchymoses. Chronic forms show grayish to brown pigmentation and thickening. In any form croupous or diphtheritic inflammation or even gangrene may develop. Urates and triple phosphates may precipitate. Small retention cysts may form in the pelvis or ureter; the small lymph nodes may enlarge and in rare cases the epithelium may hypertrophy, causing cholesteatoma (*v. i.,*-issues).

Symptoms.—(*a*) There are no certain symptoms in the rare cases in which a suppurative nephritis descends to the pelvis. (*b*) In the ascending type early vesical symptoms antedate and often obscure the pyelitis. (*c*) The symptoms are most clear in primary pyelitis from calculus or from acute hæmatogenous infection.

Pyuria is the chief symptom. The amount of pus is usually proportionate to the albumin (*v.* PYURIA). Its amount varies, as well as its constancy; when the ureter is obstructed, pyuria is suspended and when the obstruction is removed the pus escapes in increased amounts. *Polyuria* and a low specific gravity are usual in chronic cases; the writer has observed a case in which 6 quarts were voided, simulating diabetes insipidus; polyuria results from decreased absorption of water from the medullary portion or from compensatory hypertrophy of the sound kidney. In acute cases the urine is decreased. *Other urinary findings.* The urine is *usually acid* but quite often alkaline from coincident or causal cystitis or from bacteria decomposing the urea. It is often *cloudy.* It is true that large, caudate, club-like epithelial cells, often overlapping each other like shingles, are present in moderately severe pyelitis, but they may occur, though less frequently, in ureteritis and cystitis. When pyelitis extends to the papillæ or higher (pyelonephritis), cylindrical plugs of pus cells, epithelium, but chiefly of cocci, are seen (Fischl). Red cells (calculus), triple phosphate or uric acid crystals, fibrin (croupous or diphtheritic pyelitis) and various bacteria (usually the colon bacillus, perhaps the tubercle bacillus) may occur. *Pain* over the kidney is usually dull, but in acute cases may suggest stone. There is a desire —probably reflex—to urinate frequently; this symptom may lead to an erroneous diagnosis of cystitis. *Fever and rigors* are common, especially in severe calculous, colon bacillus or tuberculous types or those invading the kidney substance. Temperature is sometimes sudden and high ("irritative") and at other times is intermittent from septic absorption.

Complications.—(*a*) There may be extension to the renal substance (*pyelonephritis*) and it may be reduced to a pulpy, inspissated and even calcified mass. (*b*) Prolonged inflammation may lead to fibrosis of the kidney (Ackermann, Albarran), known as the "*ascending contracted kidney.*" (*c*) Plugging of the ureter may occasion distention of the pelvis by retained urine (*hydronephrosis*) or pus (*pyonephrosis*), both of which conditions will be described below. (*d*) *Anuria,* described by Morgagni and later by Israel, is occasioned by reflex suppression of the sound kidney or by extensive lesions in both kidneys. (*e*) *Amyloidosis* may develop. (*f*) *Intoxication* may occur by bacterial toxins and renal inadequacy. The condition resembles uræmia in some respects, but œdema, retinitis and convulsions are absent. Senator described a dyspnœa like that of diabetic coma. Treitz and v. Jaksch believe that intoxication results from absorption of ammonia (*ammoniæmia*) from decomposed urea. (*g*) Perforation outward causes the *paranephritic abscess* (*v. i.*). In some cases an interesting condition called "spinal paraplegia" has been described, probably resulting from an ascending neuritis.

Prognosis.—The prognosis is influenced by the basic simple, tuberculous or calculous infection and by the complications as described. In acute cases the outlook is generally good. Chronic cases without much necrosis may last many years and may even recover.

Diagnosis.—Much depends on careful analysis of pyuria (*q. v.*). *Cystitis* may be confused with as well as combined with pyelitis. Their differentiation is often difficult from examination of the urine alone; the albumin and pus correspond in both and possibly there is more albumin in pyelitis; no absolute reliance can be placed on the reaction, acidity or alkalinity occurring in either condition, though pyelitic urine is more often acid. The tailed, clubbed or shingle-like epithelia are more common in pyelitis, though not pathognomonic. Von Bergmann's method consisted of thorough vesical irrigation and then gentle compression of the suspected kidney to force into the bladder and thence into the catheter, pus or cells; pain over the kidney extending downward is significant; the only certain method is cystoscopy and ureteral catheterization. In *nephritis* the urine contains more albumin than is explained by the pus, though the amount may be slight in interstitial nephritis; the leukocytes are mononuclear and the presence of casts and cardiovascular changes are usually decisive.

Treatment.—*Prophylaxis* involves care in the use of catheters or irritating diuretics, the expectant treatment of gonorrhœa, etc. *Water, milk* and mineral waters should be used freely to flush out the urinary tract. *Pain* is relieved by hot applications to the kidney; opium is seldom indicated. For *pyuria,* phenylis salicylas and hexamethylenamine āā gr. x p. c. are most efficient. Methylene blue gr. j to ij, t. i. d. may also be given, but digestive disturbances must be watched for. These remedies are superior to lead acetate, santal oil or turpentine. *Surgical interference* may be necessary when ureteral drainage is incomplete. Nephrotomy is the best operation, nephrectomy being seldom indicated.

II. Suppurative Nephritis.—Its *etiology* is partly that of pyelitis. The microörganisms encountered are the staphylo-, strepto- and pneumococcus, Bacillus coli, gonococcus, proteus, typhoid and tubercle bacilli and actinomyces. (*a*) The *embolic form* results from suppurative processes elsewhere and represents merely one phase of septicopyæmia of more pathological than clinical interest. The suppurative foci are chiefly *bilateral* and *cortical;* the glomeruli are filled with microörganisms and, as the foci enlarge, they show as yellowish areas through the cortex; some deposits may also occur in the medulla; the kidneys are enlarged and lax. Clinically, *pyuria,* hæmaturia and cylindruria are *seldom* seen and a diagnosis can seldom be made; anuria or oliguria may result from multiple glomerular involvement. (*b*) The *ascending type* is essentially a cystopyelonephritis, which has been already described, and forms the so-called "surgical kidney"; infection and urinary obstruction are its usual antecedents; pyuria, polyuria and albuminuria its usual symptoms; and suppuration in the medullary rays its usual anatomical evidence; very rarely necrotic renal tissue is evacuated. (*c*) Suppuration of the kidney may result from its *invasion by contiguity* from adjacent pus foci, as appendicular, peri-uterine or vertebral foci; much pus

may be voided at once with later gradual cessation of the pyuria.

The diagnosis is always uncertain, save when sequestra come away, the prognosis always ominous and the treatment always surgical.

PERINEPHRIC ABSCESS.

A distinction is sometimes made between perinephritis, inflammation of the fibrous capsule and paranephritis, inflammation (suppuration) of the fatty capsule and surrounding tissue.

Etiology.—(*a*) The most common cause is *suppurative* disease of the *kidney,* as pyelitis, pyelonephritis, pyonephrosis (*v. i.*) and tuberculosis, from which extension occurs by actual rupture or along the blood or lymph vessels. (*b*) *Extension by contiguity* may occur from vertebral caries, appendicular, pelvic or pancreatic suppuration, liver abscess, cholecystitis or even from the lung and pleura. (*c*) It may result from *trauma* plus infection. (*d*) The so-called *idiopathic cases* are probably *embolic.* Most cases occur in persons between thirty and forty years of age and 70 per cent. occur in males.

Symptoms.—(*a*) The *onset is acute,* with a rigor and sudden fever in the embolic (so-called primary) cases, but it is *insidious* in the secondary group. (*b*) *Fever* of a continuous or remittent type is usual, though absence of fever does not exclude suppuration. (*c*) *Pain and tenderness* are experienced over one kidney, somewhat more often over the right than the left. Sometimes, with coincident involvement of the kidney, pain shoots along the ureter to the pubes or thigh. To relieve tension the patient frequently lies on the back with the *knee flexed* and the thigh rotated outward. Though we are discussing suppuration about the kidney (paranephritis), pain may arise from simple inflammation of the fibrous capsule (perinephritis); this is relieved by simple nephrotomy. (*d*) *Local findings* may be marked or absent. In some patients there are lumbar *œdema,* redness and *swelling.* The physical signs anteriorly are seldom marked, as suppuration usually occurs behind the kidney. The surgeon finds at operation a diffuse phlegmon and less often diffuse serous infiltration or circumscribed abscess; the pus may be odorless; it may smell of urine when the abscess has arisen from perforation outward of a pyelitic lesion; a fæcal odor results from infection (without perforation) from the bowel or appendicular abscess. Necrotic fat and sometimes altered blood are voided. (*e*) The *urine is negative,* unless there are (i) a pyelitic origin and (ii) rupture of the abscess into the urinary tract. (*f*) The chief *complication* is *rupture,* which occurs in about a quarter of the cases; in 15 per cent. it occurs upward (Küster), into the lung and pleura and in 8 per cent. downward, chiefly into the intestine. Burrowing occurs, according to Gerota, along the ureter toward the pelvis, when the abscess is anterior to the renal fascia and toward the lumbar region or iliac fossa when the abscess is behind the fascia. *Amyloidosis* may result from neglected or obscure cases of paranephritis. Serous pleurisy, subcutaneous

emphysema, compression of the portal vein, icterus and septicæmia may result.

The *course* averages two to four weeks, perhaps longer.

Diagnosis.—Diagnosis depends on (*a*) local pain, (*b*) œdema or swelling, (*c*) fever and (*d*) exploratory aspiration. Lumbago causes no serious confusion. Other affections causing occasional errors are psoas abscess, appendicular abscess behind the kidney or rupture downward of empyema, though these are more often etiological factors than separate questions for differentiation.

Treatment.—The only treatment is incision and drainage. In the cases of Schede and Schmieden complete recovery occurred in 73 per cent.

HYDRONEPHROSIS.

Definition.—Hydronephrosis is a sac-like dilatation of the pelvis and calices, with accumulation there of a watery fluid; obstruction to the urinary flow is its cause and atrophy of the renal substance is its result. It was first described by Tulp (1674) and named by Rayer.

Etiology and Pathology.—There are two forms, the congenital and acquired. (*a*) The *congenital form* is most often due to anomalies in the ureters; these include atresia, stenosis, valve formations, twists, compression by peritonitic bands, vestiges of Müller's or Gärtner's ducts, abnormal bloodvessels, imperfect insertion into the bladder or insertion too high into the pelvis, in the lower part of which the urine accumulates. Of the latter form the author has seen two cases in young men and J. B. Murphy has observed four others. When there are double ureters, one may end blindly in the bladder wall and dilating may compress its fellow of the same side. In some few cases no obstruction is apparent. Hydronephrosis may occasion dystocia and is rather commonly associated with other congenital malformations; 65 per cent. of congenital cases are bilateral (Roberts). (*b*) The *acquired form* may be bilateral or unilateral, depending on its cause. This may, in rare cases, be in the kidney itself (Rayer, Chopart, Christian Fenger), producing partial hydronephrosis. Enumeration of all possible causes of obstruction is unnecessary; one or both ureters may be compressed by malignant pelvic growths, as uterine cancer, and this in part explains the large proportion (67 per cent., Newmann) of bilateral hydronephrosis which occurs in autopsy figures; in clinical cases 62 per cent. are unilateral (Roberts). Ureteral obstruction may arise from calculus, tumors, tuberculous detritus, ulceration, cicatrization, floating kidney or rarely clots from trauma or parasites. Large sacs are usually caused by affections of the ureter. Other possible types are obstruction from urethral stricture, enlarged prostate or more rarely phimosis and atresia of the hymen.

As Cohnheim contended, a slow development with intermissions is more potent in the production of large sacs than is acute obstruction, for slow compression permits more continued renal secretion,

The *results* of *urinary accumulation* are (*a*) distention of the pelvis, (*b*) flattening of the papillæ, (*c*) atrophy of the renal tissue, though some normal tissue usually remains and (*d*) the formation of a fibrous sac.

Symptoms.—The chief finding is a *fluctuating renal tumor.* (*a*) It is located at first in the *renal region* but later assumes an abdominal importance. (*b*) Its *size varies,* being largest when the ureter is obstructed and in slowly developing cases becoming as large as the head and even simulating ascites; Peter Frank mentions a case in which it contained 60 quarts. (*c*) It may in the same case vary from time to time; this is known as *intermittent hydronephrosis,* and its chief causes are valve formation, stenosis or abnormal insertion of the ureter into the renal pelvis. The amount of urine is (i) normal from hypertrophy of the sound kidney, (ii) decreased in bilateral disease or from increased pressure in the diseased kidney, reflexly lessening the secretion from the sound kidney (Israel); or (iii) when the sac evacuates, suddenly very abundant and then the tumor disappears; this point is often volunteered in the patient's history. (*d*) Its *form,* on bimanual palpation, is *irregularly oval;* in moderately severe cases the kidney itself may be palpated, as well as its irregularly protuberant calices. (*e*) Sometimes the dilated ureter can also be felt. (*f*) The *ballottement rénal* is a repercussion shock obtained by tapping the tumor with one hand while the other is spread broadly over it. (*g*) Small tumors are *covered by the colon,* which imparts a modified tympanitic note on percussion. Distention of the colon by air or gas brings out tympany over the tumor. Very large sacs push the colon aside. The *ren saccatus* may develop in a floating or horseshoe kidney. (*h*) Its *contents* are albumin, paralbumin, mucus, urea and other urinary constituents, which are usually absorbed only after a long time. Diagnostic puncture is not always safe. (*i*) Sometimes it is *tender* from tension of the capsule; pain may arise from torsion of the ureter. The author has seen hæmaturia and renal colic occur in hydronephrosis due to an insertion of the ureter high up in the renal pelvis. (*j*) *Complications* include rupture, infection (pyonephrosis) or anuria and uræmia if both kidneys are implicated.

Diagnosis.—Diagnosis is quite easy (*a*) by recognizing the cause, if possible, as stone or tumor; (*b*) by careful bimanual palpation and (*c*) by cystoscopic proof that urine does not flow from one ureter. *Differentiation* concerns especially—

1. The Exclusion of Other Renal Tumors.—(*a*) *Pyonephrosis,* a dilatation of the pelvis and calices by purulent fluid (instead of water-like fluid as in hydronephrosis), is caused by obstruction plus infection, especially from pyelitis with obstruction. It occasions the same kind of tumor as to location and physical examination, but presents some other features; it is generally smaller and more irregular than hydronephrosis; its walls are thicker and more tender, for infection is superimposed on obstruction; there is more

renal necrosis; systemic septic symptoms prevail; its contents are pus, pulpy detritus, triple phosphates, bacteria, disorganized blood, fat, cholesterin, fœtid ammonium compounds and even gas. Puncture is always dangerous, in doubtful cases more so than operation. Its prognosis also is clearly more unfavorable. (*b*) *Cystic kidneys,* (*c*) malignant *renal growths* and (*d*) echinococcus will receive consideration below.

2. Other Abdominal Tumors.—(*a*) *Ovarian tumor* is connected with the uterus, grows upward and may be associated with changes in the mammary glands and genital functions. Ovarian tumor with a long pedicle may deceive, but it pushes the cæcum backward. The dulness is greater, though in large renal sacs the colon is pushed aside. The slight respiratory excursion and *ballottement* in hydronephrosis are of quite secondary diagnostic value. Confusion is likely when hydronephrosis develops in a floating kidney. Exploratory puncture is an unsafe procedure; when made, the fluid from ovarian cysts shows a high specific gravity (1,020); much paralbumin is present, also cylindrical epithelium, colloid or chocolate-colored material and of course no urea. Under narcosis the entire hand may be introduced into the rectum to determine the point of origin of the tumor, though this severe method is seldom necessary. (*b*) *Ascites and pregnancy* are readily differentiated. (*c*) *Encapsulated peritonitis* may be harder to differentiate, but the urinary conditions and sac-like distention are usually decisive. (*d*) *Splenic* and *hepatic* tumors are more flat on percussion, encroach more upon the lungs and are above or anterior to the gas-distended colon. In a renal growth a furrow and tympanitic loop of bowel may lie between it and the liver or spleen.

Prognosis.—The prognosis is generally unfavorable, as the cause is often inaccessible or the process well advanced. The prognosis is better in cases of renal stone than in cancer or in the generally inviable congenital forms. The course is often intermittent and protracted.

Treatment.—Treatment is surgical. Nephrotomy is indicated; nephrectomy is considered poor surgery since Ayrer and others have shown that some sound renal tissue nearly always remains. For the same reason ligature of the renal vessels should be avoided (Nicolai). The mortality resulting from nephrectomy for hydronephrosis is 5.7 per cent., for pyonephrosis 15.3 per cent. (Schmieden); Rosving reports 0 per cent. and 19 per cent. mortality in his cases of nephrotomy for hydro- and pyonephrosis, respectively; 34 per cent. of his cases recovered completely, 27 per cent. recovered though with fistula, and secondary operation was necessary in 20 per cent. Fenger advised conservative operations, especially in partial hydronephrosis. Reference should be made to surgical treatises for details of operations on the ureter, excision of the pelvic sac, etc. Caspar and Kümmel maintain that the phloridzin and cryoscopic tests are of great value in determining which side is involved, by testing the urine obtained from each ureter by ureteral catheterization; Israel

has a very different opinion, as has Rosving, who prefers to cut down on both kidneys before extirpating either one.

RENAL CALCULUS (NEPHROLITHIASIS).

Etiology.—Stone in the kidney or its pelvis results from deposition therein of the solid constituents of the urine. It is probable that stone formation is due to *bacteria*. The kidneys usually eliminate bacteria without changes to themselves, but sometimes bacteria, especially the colon and typhoid bacilli, form the nucleus of stones. Ebstein, Moritz, Mendelsohn and others have shown that an albuminous cement substance is found even in very slight urinary deposits or renal sand. Renal stone has been known since the time of Hippocrates.

The *predisposing factors* are obscure. (*a*) A *sedentary life* and over-indulgence in food and alcohol are apparently predisposing factors. Galen noted the coincidence of gout and renal calculus. Stones are said to be uncommon in very active persons. (*b*) Most cases occur between *thirty and sixty years* of age; in the extremes of life vesical calculus is more common. (*c*) Seventy-five per cent. of cases are in *males*. Stones are passed more easily and therefore are possibly less often detected in women. (*d*) Renal calculus occurs most frequently in Asia and there are certain foci, more of them in Europe than in America, where it is particularly frequent, as central Russia, Holland, Italy, Altenberg, Munich, Hungary, Scotland, west France and southeast England. (*e*) Other possible factors are heredity, which is especially a factor in formation of uric acid and cystin calculi; paraplegia (Müller reported 10 cases of spinal fracture with calculus formation) and foreign bodies in the renal pelvis, as blood clots from trauma and parasites, as the distoma. The older classification of primary and secondary calculus has been abandoned.

Chemistry and Pathology.—According to their *size,* these urinary deposits are spoken of as sand, gravel or stones; sand and gravel nearly always consist of uric acid or its salts. They may range from the size of a pea to that of a hazel-nut. The heaviest recorded stone weighed thirty-six ounces. Though most often round, oval or cylindrical, they may be dendritic or coral-shaped. Sometimes they are facetted. A number of them may exist, perhaps 15 or 20; in rare cases 1,000 to 2,000 may be found. (*a*) The stone caused by *uric acid* and urates (described first by Scheele, 1776) constitutes 66 per cent. of renal stones. It is hard, smooth or slightly uneven, sometimes facetted or lamellated and consists of uric acid, ammonium and sodium urates, urinary pigment and a small amount of xanthin. Its broken surface is amorphous. Its color is yellow, red or brown, depending on the amount of urinary pigment. It may contain some oxalates and phosphates. Under the microscope needles are seen. Uratic calculi turn carmine red on adding nitric acid and ammonia. They dissolve in caustic potash. They are deposited in acid urine, but the exact reason is not known; the normal sodium urate may lose

part or all of its sodium by action of the alkaline phosphates, so that free uric acid and the less soluble bi- and quadriurates are formed; Roberts and Klemperer consider a decrease of the urinary pigments of great importance; disturbed metabolism or indigestion may occasion uratic deposits, though little is known of the "uric diathesis." (*b*) *Oxalate stones* (Fouccoy and Vauquelin) are next in frequency; Klemperer holds that oxalates occur in even 50 per cent. of cases. They are very hard, rough, warty or prickly (the mulberry calculus); for this reason they cause more pain and hæmaturia than the uratic stones and are more often removed surgically. They are usually single and gray, but may be dark-colored from the bleeding they induce. Lamellation is never observed. Uric acid, calcium carbonate or xanthin may also enter into their composition. No effervescence occurs on adding acetic acid unless carbonates are also present. They dissolve in mineral acids and oxalate of lime crystals gradually precipitate on the addition of ammonia. Oxalates precipitate from acid urine. (*c*) *Phosphatic* calculi (Bergmann) are formed in alkaline or neutral urine. They are usually calcium salts, but if the urine is ammoniacal triple phosphates may be formed. Admixture of bacteria, calcium carbonate and xanthin may be noted; phosphatic stones are small, soft and clay-colored. They are infrequent and usually only form a coating to calculi of other composition. (*d*) *Other forms* are rare. Calculi of (i) *calcium carbonate* and more rarely ammonium or magnesium carbonate may occur in the aged, though rarely in man compared with their frequency in cattle; they are white and dissolve with effervescence in mineral acids. (ii) *Cystin* calculi (Wollaston) are smooth, yellow and waxy; they never exceed the size of a pea, are soluble in acids and alkalies and deposit hexagonal crystals when dissolved in acetic acid and ammonia. Only 60 cases are reported (Riegler, 1904). (iii) *Xanthin* stones (Marcet) occur almost exclusively in children; only 10 cases are recorded; they are smooth, yellow or brown, dissolve in nitric acid and on evaporation leave a residue which turns red on adding caustic potash. (iv) Only 3 cases of *indigo* stones are on record. (v) The fatty stones (*urostealiths*) described by Heller probably do not originate in the kidneys, but result from fatty substances deposited in the bladder; they are 85 per cent. fat and are soft, rubber-like and combustible.

Infarcts may be considered here. (*a*) *Uric acid* infarcts occur in gouty subjects and in 50 per cent. of the new-born, chiefly in the medullary collecting tubules; they are most common in the still-born or in weakly infants and are thought to result from destruction of the tissues or cells (nuclein). (*b*) *Lime infarcts* are chiefly phosphates and carbonates, sometimes oxalates; they are deposited principally in the pyramids and in the membrana propria when the renal cells are destroyed; they occur in the aged and in poisoning by mercury, arsenic, phosphorus or bismuth. (*c*) *Pigmentary infarcts* of hæmatoidin, bilirubin, hæmoglobin or methæmoglobin are uncommon.

Symptoms.—Stones may be first discovered accidentally at autopsy

or, very exceptionally, may pass without symptoms. Gravel or renal sand is more often latent than is stone.

1. Pain.—Pain is the most common symptom. It is of two kinds: (*a*) The *dull pain* in the loin, often associated with local tenderness over the kidney; Fenwick describes localized pain without any urinary changes, indicating that the stone lies in the medullary substance. Pain may be felt over the opposite kidney. (*b*) *The renal colic,* which arises from incarceration of the stone as it attempts to leave the pelvis, where it nearly always originates; it is often preceded by pain in the loin and may be initiated by exercise, as riding, or by indigestion; Eichhorst mentions a surgeon who had renal colic every time he drank white wine. The pain in renal colic begins over the kidney and *radiates down the ureter* to the testis, labia, bladder or urethra; the testicle is retracted (in the descent of the testis, its envelopes receive a twig from the first lumbar nerve). It may rarely radiate to the chest, heart, shoulder, epigastrium or to the opposite side. During the colic, the *kidney* is *sensitive;* when it is not so, Israel hesitates to diagnosticate stone. The *pain* is *cutting* and very *severe;* it often occasions reflex nausea and vomiting, sometimes chills, sweats and fever or in children convulsions and rarely collapse or even death in most exceptional cases. Vesical tenesmus is frequent. If the stone passes, the pain is greatest as it enters the bladder. Constipation of a spastic order is much more frequent than diarrhœa or involuntary movements. Some observers hold that renal colic occurs oftenest at night. It lasts, with intermissions, a few hours, rarely days; movement may excite recurrence. The patient lies on his back with the leg drawn up and the abdominal wall rigid. After the attack there may be dull pain (*v. s.*), which may be caused by large, numerous or sharp stones. Eichhorst records a case in which the sole symptom was the patient's attitude; he leaned far forward and depressed one shoulder to avoid the pain induced by the erect attitude.

2. Urine.—The urine is almost invariably altered. (*a*) *Hæmaturia* occurs independently of pain and colic, as well as after them. Absence of hæmaturia after a renal colic speaks strongly against a diagnosis of stone (Israel). It is often macroscopic and red disks are found with the microscope, except in the rare cases when stone is imbedded in the renal tissue or there is permanent occlusion of the ureter. At the time of the colic the scanty, bloody urine is frequently voided with considerable tenesmus. In exceptional cases early profuse hæmaturia may result from vascular erosion. Musser found hyaline casts in nearly every case. (*b*) *Anuria* (suppression of urine) is not common. It may occur (i) with stone in one ureter and disease or atrophy of the other kidney, (ii) with bilateral calculi (Albarran), (iii) with stone in one ureter and reflex suppression (Morgagni) of the other sound kidney through the splanchnic nerve or (iv) by urethral stoppage by a stone. Legeau (1891) collected 30 autopsied cases of calculous anuria; there was stone in one kidney and the opposite kidney (α) contained a calculus in 14 cases, (β)

was hypoplastic in 3, (γ) sclerotic in 6, (δ) its ureter was stenosed in 6 and (ε) in one case the opposite kidney was absolutely normal. The resulting symptoms resemble those of uræmia. However, in 41 cases collected by Herter convulsions were present in but 12 per cent., headache in 14 per cent. and vomiting in 29 per cent. Consciousness is usually preserved, the pupils are usually narrowed and the temperature is low. Roberts speaks of this condition as "latent uræmia"; 11 cases of anuria persisted over four days, 18 cases seven to fourteen days and seven over fourteen days. Russel records recovery after twenty-eight days of anuria. With its relief large quantities of urine are voided. (*c*) *Pyuria* results in protracted cases from pyelitis, which is usually secondary to the calculus; infrequently a renal intermittent *fever* is observed, resembling the hepatic intermittent fever of gall-stones. Pyelitis may be catarrhal, suppurative, ulcerative or perforative. (*d*) *Crystals* of uric acid and oxalates are considered especially important in the diagnosis by some writers, as Leube; Ulzmann regarded the pointed uric crystals as particularly suggestive of renal calculus. (*e*) *Stones in the urine* determine the diagnosis, though not frequently found.

3. The *X*-Rays.—Oxalate stones throw a dark shadow in every instance; uric acid stones seldom are visible. Opinions differ regarding the value of the *x*-rays. The difference may be wholly technical, as Leonard reports the unusual figures of but 3 per cent. of errors; he found calculi in the ureter twice as often as in the renal pelvis. Kümmel insists that 90 per cent. of stones cast a shadow.

4. Physical Findings.—The *physical findings* are generally less important than the urinary findings and the colic. Local tenderness has been mentioned (*v. s.,* Pain). Enlargement of the kidney (hydro- or pyonephrosis) is not common. Palpation of the stone in the ureter is rarely possible, either from without or by the rectum or vagina; once the writer felt a calculus at McBurney's point. Stones have rarely been felt to crepitate in the renal pelvis (Piorry). Cystoscopic examination and catheterization of the ureters are methods which can be employed only by the most expert and have some disadvantages. M. L. Harris' segregator may determine which kidney is affected.

Complications.—Ulcerative *pyelitis* may lead to perforation and paranephric abscess. Pyelonephritis may develop. Hydronephrosis results from mechanical occlusion of the ureter and pyonephrosis from obstruction with infection. Injury to one kidney may be compensated by hypertrophy of its fellow. Amyloid degeneration, renal tuberculosis or even cancer may develop. The ureter may rupture from ulceration or tension. Pregnant women may abort and Troja instances a case in which 14 abortions occurred after 14 attacks of colic.

Diagnosis.—The diagnosis is based on (*a*) the association of the pain and colic, (*b*) hæmaturia, (*c*) pyelitic pyuria, (*d*) *x*-ray findings and (*e*) less often, the anuria and swelling of the kidney. If the

results of the x-rays are negative stone is not excluded. A diagnosis of stone is found correct in only 60 per cent. of the cases operated on.

1. Pain and Colic.—Pain and colic may rarely occur in other renal conditions, as in renal embolism or passage of blood clots, parasites, echinococcus vesicles, malignant tissue or tuberculous detritus, renal aneurysm, capsular thickening and floating kidney. In one case the author made a diagnosis of calculus from pain, colic and hematuria; the operation by Murphy revealed partial hydronephrosis from high insertion of the ureter. Confusion may result from referred disease, the cases of tabes or angioneurotic edema, intestinal colic, gall-stones, appendicitis, diaphragmatic pleurisy or hysteria.

2. Hematuria.—Hematuria (*q. v.*), associated with pain, may cause errors in diagnosis, usually corrected only at operation; thus acute congestion and particularly capsular fibrosis may be found. Israel states that there is a unilateral nephritis *a* with colic, *b* with albumin and without casts, *c* with casts and without albumin, *d* with hemorrhage simulating tumor, but *a* the bleeding never causes the colic. In these doubtful cases exploratory incision is also indicated. The hematuria resulting from cancer and tuberculosis does not improve with rest in bed, while calculous hematuria usually does. Hematuria and renal colic may develop in chronic nephritis from acute renal congestion.

Prognosis.—The prognosis must be conservative, for the course is usually chronic and relapsing. The author knows of four physicians who passed calculi without symptoms afterward, but this is not the rule. Rupture of the ureter, collapse during the colic, pyelitis becoming pyelonephritis, anuria and uremia must be considered in the prognosis. Pyuria is more ominous than hematuria.

Treatment.—1. Prophylaxis.—Prophylaxis of renal calculus must be indefinite, because so little is known regarding its pathogenesis. It is usually stated that regulation of the diet concerns moderation in both proteids and carbohydrates; as in nephritis foods rich in nuclein are forbidden; vegetables may be allowed. Acid foods and alcohol should be interdicted, water given freely and exercise insisted upon. In uric acid calculi, the alkaline waters are valuable, as those of Karlsbad, Vichy, Marienbad, Saratoga, Bedford, Poland and Mountain Valley. Sodium phosphate gr. x or bicarbonate 3ss t. i. d. is indicated. Lithium carbonate, piperazin and glycerin possess no proven solvent powers. In localities where calculi develop very frequently the drinking water must be used with caution. Stones once formed cannot be dissolved by drugs.

If the oxalates are deposited, treatment is the same as in oxaluria (*q. v.*). In alkaline urines from which phosphates are precipitated the urine must be made acid by phosphoric salicylas, gr. x t. i. d.; acid waters, as Selters or Apollinaris, may be employed; meats should be restricted; hexamethylenamine gr. x t. i. d. is excellent in bacteriuria. Altogether, dietetic treatment, though important, has no great bearing in prophylaxis or treatment and the writer cannot agree with those who declare that these measures are generally efficacious.

2. Colic.—The colic should be medically treated like that of gall-stones (see page 707).

3. Surgical.—Surgical intervention is indicated by (*a*) anuria; (*b*) severe pyelitis; (*c*) septic or aseptic obstruction of the ureter; (*d*) exhausting hæmaturia and (*e*) repeated attacks of colic. As these conditions are present in most cases, successful treatment must be surgical.

The ideal operation, advocated by Morris (1880) is nephrolithotomy (nephrotomy), closing the renal wound if there is no infection; operation on the aseptic kidney entails a mortality of 3 per cent. and operation in the presence of infection 10 per cent. (Albarran); in these cases the pelvis should be drained (nephrostomy). Tenny (1904) collected 134 cases of ureteral calculus; 122 were operated on, with a mortality of 19 per cent. or of 47 per cent. if pyelitis was present; anuria is a serious complication, for 52 per cent. of the operated cases die; without pyelitis or anuria the fatalities were 6 per cent. Rosving has operated without a death. Medical treatment of anuria, with full doses of nitroglycerine, hot packs, cups to the lumbar region and subcutaneous saline transfusions for more than a day or two raises the mortality from operation; nephrostomy is indicated.

TUMORS OF THE KIDNEY.

I. Cancer.—Etiology.—The etiology is obscure. It constitutes but 2 per cent. of all cancers and but 1 per cent. of primary cancers in adults (Rubinstein); 62 per cent. occur in males and 32 per cent. in the first ten years of life. Its frequency in children was noted by Bright (though some reported cases were sarcomata) and it constitutes 52 per cent. of renal tumors in children (Hirschsprung). Weigert observed one case in a new-born child. Cancer in the renal pelvis may result from stone. In the Albarran and Imbert series of 380 renal tumors, cancer constituted 50 per cent.

Pathology.—The primary form is usually infiltrating and the secondary form consists of nodules. Medullary cancer is more common than scirrhus, colloid and other forms. It usually begins in the convoluted tubules of the cortex. Its growth is usually limited by the kidney's capsule. The kidney is nearly always enlarged, its average weight in children being 8½ pounds (Guillet) and in adults 10 to 15 pounds (or even 50). The opposite kidney and the sound parts of the affected viscera may show compensatory hypertrophy. Hemorrhages into the neoplastic tissue may occur. Local invasion of the renal veins, ureter, inferior cava, pancreas, adrenals or intestine and metastases (68 per cent.) in distant organs sometimes develop.

Symptoms.—Some, particularly secondary, cancers are latent.

1. Renal Tumor.—Renal tumor, the most common finding, occurs in 97 per cent. of cases. It begins in the region of the kidney, but, being resisted by the firm lumbar tissues, it grows forward. Bimanual palpation clearly demonstrates its origin in most cases. If

large, it may fall forward. It is tender and usually *firm,* though its degeneration may occasion slight fluctuation. The form of the kidney is usually preserved, though nodules may be palpated. It usually attains a large size (*v. s.*), compressing and dislocating the liver and spleen upward and crowding the abdominal viscera. Fürbringer reports a case in which the heart was pushed up to the clavicle. On tapping, a repercussion wave is sometimes noted (*ballottement rénal*). Percussion gives a dull note, except so far as the intestines lie over it, when mixed flatness and tympany are elicited. The *colon overlies the kidney,* running from left to right, from above downward; on the left side this relation is more clearly distinguished; on inflation of the colon with air or gas its tympanitic note in some degree obliterates the renal dulness; in a recent study by Kofmann, this condition was found in less than 50 per cent. of large renal tumors; in tumors on the right side the colon is often dislocated laterally or even downward. There is but little if any respiratory excursion. In rare instances the tumor may pulsate and produce by pressure on the aorta a systolic murmur, which, as in Richard Bright's case, may simulate aneurysm. If the tumor grows into the ureter hydronephrosis may augment the swelling.

2. Pain.—Pain is an early and frequent symptom, due to pressure exerted on the lumbar plexus. For this reason also there may be slight sensory disturbance in the legs. Pain is usually dull and only rarely is it lancinating or colic-like, due to passage of clots or bits of tumor (*v. i.*).

3. Hæmaturia.—Hæmaturia is an early symptom. According to Guillet it occurs in 50 per cent. and is the first symptom in 25 per cent. of cases; Israel holds that it occurs in 92 per cent. and is the first manifestation in 70 per cent. It is less frequent in children (16 per cent.) than in adults. Sometimes worm-like clots are passed, with great colic; they sometimes measure one-half to one inch; clots in a clear urine are almost pathognomonic (Israel). The cystoscope and ureteral catheterization prove that the hæmaturia is unilateral. The hemorrhage usually originates in the diseased kidney but may come from the opposite hyperæmic kidney. In other respects the urine is generally normal. It is usually held that albuminuria corresponds to the amount of blood, but Albarran asserts there is more albumin than the blood accounts for. Very rarely bits of cancer tissue pass. The urea is increased as in all cancers. Pyuria is no part of renal cancer.

4. Cachexia.—Cachexia develops in most cases.

Complications.—*Compression* symptoms include paraplegia dolorosa, constipation, intestinal obstruction, œdema from compression of the iliac veins and ascites; varicocele, first noted by Guyon, is said to seldom occur in other renal tumors and is due to late compression of the spermatic vein by the tumor or contiguous lymph nodes. Metastases, enlarged glands, rarely growth into the lumbar muscles or skin and perforation into the peritoneum or intestine with fatal

hemorrhage, uræmia, fever from cachexia or terminal infection may be observed. According to Israel, tumor of the renal pelvis causes greater hæmaturia and more hydronephrosis (therefore greater variations in the size of the tumor); it sometimes grows down the ureter and is seen with the cystoscope to project into the bladder; particles of tumor tissue and cells are more often obtained. Albarran and Imbert collected 65 cases of tumors in the pelvis and ureter; 27 were ureteral. Savory and Nash collected 60 cases of painful villous tumors in the pelvis.

Course and Prognosis.—Death is invariable without operation; it usually results from cachexia, in two years in adults and in eight months in children (Rohrer and Roberts). Guillet states that 6 cases have lived longer than ten years and 1 lived seventeen years.

Diagnosis.—The following are important: (*a*) renal tumor, (*b*) pain, (*c*) hæmaturia and (*d*) cachexia. Imbert speaks of the complete type (tumor and hæmaturia), the hæmaturic type (without tumor, 6 per cent.) and the tumor type (without hæmaturia, 23 per cent.).

Differentiation.—Differentiation is necessary, from (*a*) *liver tumor,* which encroaches much more upon the diaphragm and is characterized by its respiratory excursion, by the location of the tumor within the edge of the liver and by such complications as icterus and ascites. The colon lies below the liver and there are no urinary findings. Bimanual palpation clearly differentiates uncomplicated cases. Between a kidney tumor and the liver a furrow and loop of resonant intestine are frequently found. (*b*) A *splenic tumor* maintains the form of the spleen, lies above the colon (whose distention does not modify the splenic dulness), moves on inspiration, shows the characteristic notches and seldom causes confusion except when it so rotates that only its convexity can be felt. (*c*) An *ovarian* tumor projects upward from the pelvis, with which its connection can be demonstrated, save when it has a long pedicle. It is more central and sustains no characteristic relation with the colon. (*d*) *Retroperitoneal* growths are much more difficult to differentiate, especially enlarged lymph glands and lymphosarcoma. They are generally more central and less movable. Of perirenal retroperitoneal lipoma 50 cases are reported. (*e*) Other renal conditions must be differentiated. (i) *Cystic degeneration* (*v. i.*) is usually bilateral and runs a most chronic course. (ii) Sarcoma cannot be differentiated. (iii) Hydro- or pyonephrosis (*q. v.*), in which the tumor varies exceedingly and has characteristic urinary changes. (iv) Tuberculosis is distinguished by hectic fever, tuberculosis elsewhere, pyuria and finding of the tubercle bacilli in the urine.

II. Sarcoma.—Sixty-six per cent. occur under ten years of age; Walker in 145 cases found that most cases occurred in the first two years of life. Albarran and Imbert state that sarcoma constitutes 20 per cent. of renal growths. It is slightly more common in girls than in boys and in the left than in the right kidney. Cohnheim's

theory of embryonal inclusion is supported by the occasional presence of cartilage, bone and striated and unstriated muscle. Sarcoma is of the round, spindle or melanoid cell type, may contain fatty, vascular and even carcinomatous tissue and may weigh 6,000 gm. Secondary sarcomata may develop by metastasis from other organs or by invasion by contiguity.

Its *symptoms* are essentially those of carcinoma, from which an intravitam diagnosis is rarely possible, except when sarcomatous tissue is passed in the urine or excised at operation. Hæmaturia and cachexia are infrequent; age and glandular involvement are of no diagnostic value. A remarkable overgrowth of the pubic and axillary hair and pigmentation of the skin may be observed. Operated cases live on an average sixteen months and non-operated ones half as long (G. Walker), but death is frequently a direct result of the operation.

III. Hypernephroma.—This "struma lipomatodes aberrans renis" of Grawitz develops from aberrant (included) particles of adrenal tissue. Small tumors are not a rare autopsy finding. They constitute 20 per cent. of renal tumors and consist of a scanty but vascular connective tissue stroma, containing fatty cells like those of the adrenal bodies. Hemorrhage, cystic degeneration, a tendency to invade the veins and a large proportion of glycogen and perhaps cholesterin or lecithin are considered characteristic. Fatty cells may be found in the urine. Hæmaturia occurs in 80 per cent. of cases. Metastases may develop. Bone metastasis is the first symptom in 16 per cent. but may develop as a late symptom only. Fever is noted in 57 per cent.; it occurs in but 1 to 2 per cent. of other tumors of the kidney (Israel).

IV. Other Tumors.—Fibroma, lipoma, myxoma, angioma and rhabdomyoma are of little importance. Hæmaturia occurred in only one case, an angioma. Thirty-three cases of tumor of the capsule are on record.

Treatment of Renal Tumors.—This is symptomatic and surgical. The former concerns the pain, by anodynes, chiefly opiates. Surgical treatment alone is of value. The mortality resulting from total nephrectomy in Küster's figures was 24 per cent. Schmieden collected 329 cases; 33 per cent. died from the operation; the mortality in the first decade of renal surgery (dating from Gustav Simon, 1869) was 64, in the next 43, in the third 22 per cent. The mortality of nephrectomy for all causes is at present 17 per cent. and for tumor 22 per cent. (in children, 28 per cent.). Thirty-three recoveries after radical operation are on record, one patient being alive after eighteen years. Israel in 43 cases reported a mortality of 18.6 per cent., of which two-thirds died of heart paralysis; 19 per cent. enjoyed permanent recovery after three and a half to fourteen years. Schede in 18 operations obtained 22 per cent. permanent recoveries after four to nine years. Recurrence after two years is very exceptional. The extraperitoneal operation is three times as safe as the transperitoneal. Operations are usually performed too late; ad-

vanced cachexia, very large tumors, metastases and growth into the cava are contra-indications. The other kidney must be in good condition.

CYSTIC DEGENERATION.

Various renal cysts are observed. (*a*) Those which occur in chronic interstitial nephritis are the most frequent; they result from snaring off, by fibrous tissue, of the tubules and glomeruli. (*b*) Parasitic cysts (*v. i.*) are next in frequency. (*c*) But two dermoid cysts are on record. (*d*) Cysts of the fatty capsule, retroperitoneal cysts and degenerated lymph glands are very rare. (*e*) Complete cystic degeneration (*hydrops renum cysticus, degeneratio renum polycystica*) was first separated by Rayer from the above mentioned varieties and constitutes cystic degeneration, as it is generally understood.

Etiology.—Over 50 per cent. are observed in *men* between *forty and fifty years* of age. Sieber (1906) collected from the literature 32 cases of cystic kidneys in persons under and 211 in those over twenty years of age. Cystic kidneys are observed in later fœtal life, at birth and in the adult. It is often difficult to determine whether they are acquired or congenital, though the latter conception is most favored; the cysts are thought to remain latent until middle life.

Pathogenesis.—Three main theories obtain; (*a*) that they are retention cysts, (*b*) new formations, *i. e.*, neoplastic (cystadenomata), the most acceptable hypothesis, and (*c*) malformations.

Pathology.—Congenital cases are usually bilateral; Lejars found one unilateral case in 67 and Luzzatto 41 in 226 cases. The kidneys may contain innumerable small cysts, when the shape of the kidney is preserved, or it contains larger cysts, when its contour is more irregular. According to Engländer, only 23 unilocular cysts are on record. The size of the kidneys may be that of a child's head; they may weigh twenty-one pounds. On section the kidney is honeycombed. The cyst walls consist of fibrous tissue. The cysts usually contain thin, yellow, transparent fluid, less often milky, gelatinous or brownish contents (altered blood). They are uni- or multilocular, are lined with cells like the urinary tubules or with flat polygonal cells and contain albumin, oxalate of lime, leucin-like crystals, red disks, epithelial detritus, fat granules and rarely urea or uric acid. Between the cysts, vestiges of renal tubules or glomeruli are found, which here and there may be vicariously hypertrophied. Cysts may occur in the pelvis, ureter and liver (*v. i.*). In congenital cases other deformities are sometimes found, particularly in the genito-urinary tract, brain and fingers.

Symptoms.—In congenital forms the cystic kidneys may obstruct delivery; if the infant is born alive death from asphyxia is usual, for the diaphragm is crowded upward. In adult forms 90 per cent. of cases come to autopsy undiagnosticated or with the diagnosis of some other renal disease, as nephritis. The symptoms may be classified as follows: (*a*) *Renal cysts* are detected clinically in 29 per cent.

of cases only (Luzzatto) or in 12.4 per cent. (Sieber). Their general characteristics are those of solid kidney tumors, but they are almost always *bilateral* (*v. s.*). Fluctuation seldom occurs. In very few cases the individual cysts can be palpated; in two cases which the author demonstrated they could be clearly palpated. Echinococcus cysts, hydro- and pyonephrosis, sarcoma and cancer of the kidney are only distinguished by their unilateral occurrence; if bilateral, differentiation is almost impossible. (*b*) The *urine* is variable; it may be normal, increased or decreased. The urinary findings are typically like those of interstitial nephritis, *i. e.,* the urine is abundant, with few solids, low specific gravity and traces of albumin. Red disks are found in 20 per cent., sometimes in great numbers. In two of the author's cases many red cells appeared each time the kidneys were palpated. Leucin-like bodies may appear in the urine or in exploratory punctures. Beckmann first described these small colloid bodies ("rosettes") with structureless centres and one to five concentric rings and clearly radiating striations. (*c*) *Cardiovascular* changes (left ventricular hypertrophy and sclerosed arteries) are common and heighten the resemblance to contracted kidney. The changes were well marked in the three cases which the author was able to diagnosticate. (*d*) *Uræmia* is the common cause of death. The clinical course is very chronic and is broken by attacks of "periodic uræmia." The digestive disorders of uræmia are very frequent (see page 750). Lejars observed a case living fifteen years and Senator one living twenty years after the cysts were found.

Diagnosis.—The diagnosis can be made when there are (*a*) bilateral (perhaps palpably cystic) renal swellings; (*b*) the cardiovascular and urinary findings of contracted kidney, with hæmaturia and colloid "rosettes," obtained in the urine or by puncture; (*c*) uræmia and (*d*) perhaps renal pain and tenderness or congenital cysts in the liver (in 28 per cent., Lejars); in a case of Dr. W. F. Dickson renal cysts were found with cystic liver, atrophic heart and pain resembling appendicitis (see ECHINOCOCCUS OF THE LIVER, DIFFERENTIATION). Hanot describes cases resembling calculous colic.

Treatment.—Operation is permissible only in the exceptional, unilateral cases or when suppuration develops. About one-third of the operated cases die immediately and in few are any lasting results obtained. The treatment is that of chronic nephritis.

RENAL PARASITES.

I. Echinococcus Cysts.—Echinococcus cysts of the kidneys occur in 9 per cent. of echinococcus disease (Neisser's 900 cases). In 970 cases (Vegas and Cranwell) the liver was diseased in 64 per cent., the lungs in 7 per cent., the spleen in 3 per cent. and the kidneys in 2 per cent. Baradulin in 1906 collected 142 cases of renal echinococcus (see ANIMAL PARASITES).

Symptoms.—(*a*) A *renal tumor* is found in 50 per cent. when the

cyst attains large dimensions. It is easily confused with other cysts, hydronephrosis, cancer or tuberculosis without the exploratory puncture and the urinary findings. *Fluctuation* and the *hydatid thrill* are not often found (see LIVER ECHINOCOCCUS). The cyst begins in the cortex, usually in either pole of the kidney; it is unilateral in 96 per cent. of cases and is left-sided in 62 per cent. If the cyst calcifies it may be seen by the *x*-rays. The opposite kidney may hypertrophy. (*b*) Exploratory *aspiration* may show (i) fluid or pus containing urinary constituents, cholesterin, succinic acid, much sodium chloride and a reducing substance; (ii) vesicles, hooklets or particles of lamellated membrane. (*c*) The *urine* may contain vesicles, hooklets, membrane, pus from pyelitis and blood from rupture. As many as 100 vesicles have been passed. (*d*) Passage of vesicles is attended by severe *colic,* fever, chill and vomiting.

Complications.—Seventy-five per cent. *rupture,* chiefly into the renal pelvis; perforation may occur into the intestines, lumbar muscles, lung or pleura; the patient may feel the rupture. The toxic substances, liberated by rupture or puncture, may cause urticaria, severe heart symptoms or even fatal syncope. Adhesions to the liver, spleen and intestine may form. Suppuration or gangrene may develop (pyonephrosis or paranephritis). Obstruction of the ureter causes hydronephrosis; vesicles which plugged the lower end of the ureter have been seen with the cystoscope.

Prognosis and Treatment.—The course is very chronic, covering even twenty to thirty years. Suppuration may cause pyæmia. The cyst may shrink or calcify. Bilateral involvement makes the prognosis unfavorable. Spontaneous recovery by rupture into the urinary tract occurs in 32 per cent. (Béraud). Operation should be extraperitoneal. Nephrotomy is indicated, as there is seldom more than a single cyst. Nephrectomy entails a higher mortality (8 per cent.). In 30 operations (nephrotomy) by Schede there were no fatalities; 87 per cent. completely recovered and 10 per cent. recovered but had fistulæ.

Enstrongylus Gigas.—The enstrongylus gigas which occurs frequently in dogs or wolves, is very rare in man. It is a nematode whose ova appear in the urine. It causes pyelitis, hæmaturia, colic, tenesmus and sometimes chyluria.

Distoma Hæmatobium.—The distoma hæmatobium described by Bilharz (1851) in Egypt, causes endemic hæmaturia. It is a trematode and enters the alimentary tract by the drinking water. Reaching the veins, its ova obstruct them and cause pyelitis, hæmaturia, tenesmus and sometimes form the nucleus of renal stones. Salol is said to be beneficial.

Filaria.—The Filaria has been mentioned under CHYLURIA.

Tuberculosis and **syphilis** of the kidney (v. INFECTIONS).

SECTION VI.

DISEASES OF THE BLOOD.

Anæmia is a reduction in the blood as a whole or more often a reduction of its important constituents, as the red corpuscles, their number, hæmoglobin or albumin. Every anæmia has a cause, yet in our present ignorance we adhere to the older division of anæmia into primary and secondary forms. *Primary anæmia* includes chlorosis and pernicious progressive anæmia.

CHLOROSIS.

Definition.—Chlorosis is a disease of hæmogenesis, occurring exclusively in women, chiefly in the developmental years and in the decennium following, in which the cardinal symptom is anæmia; the red cells are reduced but the hæmoglobin is disproportionately reduced; chlorosis develops spontaneously, usually runs an uncomplicated course, tends to recur and responds to therapy by iron. Chlorosis is a disease, while anæmia is but a symptom.

Etiology.—Its etiology is uncertain. (*a*) *All cases occur in women,* especially in girls who menstruate before the pubes and mammæ develop (Niemeyer). It is more common in blondes than in brunettes. (*b*) Chlorosis occurs largely between the *fourteenth* and *seventeenth years.* It is rare after the twenty-fourth year (chlorosis tarda), except from relapses. (*c*) *Heredity* is a marked factor (Bramwell). Chlorosis, tuberculosis or hysteria is not infrequently associated in the family history. (*d*) *Malnutrition,* lack of hygiene, working in dark quarters, insufficient or excessive exercise, nostalgia, improper clothing, wearing of corsets and emotional or sexual factors are indirect causes. Various theories have been advanced as to the nature of the disease, as deficient development of the genitalia, hypoplasia of the vascular apparatus, adynamia of the blood-making organs, inability of the blood-making organs to respond to the bodily needs at puberty, insufficient absorption of iron, auto-intoxication from the internal genitalia at the time of puberty and intestinal auto-intoxication.

General Symptoms.—Among the early symptoms are *rapid tiring, dyspnœa* and *palpitation,* which are rarely absent and are most noticeable in the morning. *Pallor* is the earliest and most constant sign and affects the skin and mucosæ, the skin being of a *yellowish-green hue* (chlorosis, as named by Varandel in 1670). Sometimes the

color is maintained, the face being flushed, probably from peripheral vasomotor irritability (*chlorosis rubra*); hence the mucous membranes should be examined, even more than the skin. The eyes are often brilliant. Psychical changes are noted, as stupor and *lack* of *mental and muscular energy* (Sydenham and Morton). Associated symptoms are *headache,* which may be dull or sharp, sometimes migraine-like or increased at the time of menstruation, spots before the eyes, tinnitus, vertigo and syncope which occurs especially from standing, travel and exposure to the sun. *Menstrual disturbances* are frequent, the flow being often decreased in younger subjects. Digestion is very frequently disturbed and there may be *anorexia,* perversity of appetite, craving of sour articles, hunger between meals, pain in the stomach, eructations and gastralgia. *Constipation* is frequent. The hands are cold, the skin dry and the peripheral circulation sluggish. Circulatory symptoms appear, as the *bruit de diable* in the neck veins, *anæmic heart murmurs* and rapid pulse.

Special Symptoms.—1. The Blood.—The blood is fluid and its coagulability is increased. The red blood cells are decreased (*oligocythæmia*) and average *three to four millions* at the first examination; as few as one million red cells have been observed. In some rare cases they are not decreased (Duncan, Hayem, Laache). Formation of rouleaux is poorly marked. The red cells appear *pale* and their average *size is reduced.* Irregularity in contour (*poikilocytosis*) is not frequent; megalocytosis and microcytosis are rare; normo- and megaloblasts are found only in the severest cases; normoblasts are sometimes present in "blood crises." More suggestive than the red cell reduction (oligocythæmia) is the reduction of hæmoglobin (*oligochromæmia*), which is reduced more than the red cells (Duncan, 1867); the hæmaglobin averages 30 to 40 per cent. This *reduction in the color index to one-half* or less always suggests chlorosis, is most marked in the first seizure in young individuals and is less conspicuous in relapses or in older individuals. Some writers claim that the reduction in hæmoglobin is more apparent than real, because there is more rapid regeneration of the cells than of the hæmoglobin. The *white cells* are essentially *normal,* therein differing from many other forms of anæmia; occasionally lymphocytosis and eosinophilia occur. The iron and nitrogen of the blood and its specific gravity are reduced proportionately to the decrease in hæmoglobin.

2. The Vascular System.—(*a*) Rokitansky and Virchow observed hypoplasia of the aorta and vessels, which may co-exist with hypoplasia of the genitalia, muscles and bones, but cannot be regarded as causative. (*b*) *Palpitation* is almost constant on exertion; the apex-beat is strong to compensate for the poverty in hæmoglobin. (*c*) *Heart murmurs* occur in two-thirds of the cases; they are largely functional and may be caused by insufficient tension of the mitral valve, dilatation of the left ventricle causing relative mitral insufficiency, weakened heart muscle (producing a muscular murmur in place of the tone), tricuspid insufficiency or vibration of the pul-

monary artery. The murmurs are largely systolic; undoubted diastolic venous bruits have been heard. (*d*) *Dilatation of the heart* occurs from anæmia or myocardial degeneration, though according to Leube and Grunmach, the dilatation is not real but apparent, being due to retraction of the lungs away from the heart, because of insufficient lung expansion; this has been confirmed by the *x*-rays. (*e*) The *pulse* findings are not constant; the pulse may be rapid, throbbing or excitable. The arterial tonus is reduced, dicrotism and capillary pulsation are frequent and double tones have been noted in the crural vessels from decreased tension and diastolic recoil. (*f*) A venous *bruit de diable* in the neck occurs in over 50 per cent. of cases and more frequently than in other anæmias; it may be palpable and is heard more frequently over the right jugular vein, because it is more vertical than the left; it also is heard in other veins and in sound individuals. It is increased in the sitting posture by turning the head to one side and by deep inspiration. Its explanation is difficult; it is referred to change in the tonus of the vessel, waves in the veins above their valves and a change of the blood plasma. (*g*) Arterial murmurs may be heard, as over the subclavian artery; pulsation in the peripheral veins, angiospasm and erythromelalgia have been observed. (*h*) *Œdema* is seen in 12 per cent. of cases and is usually slight, occurring chiefly about the ankles and eyes and rarely in the serous cavities.

3. Respiratory Tract.—Dyspnœa is a common complaint, explained by Eichhorst as an hæmatogenous respiratory insufficiency. The respiration rate is 26 to 30. Sometimes a paroxysmal cough develops.

4. Digestive Tract.—The digestive tract is involved in one-third of the cases. There is sometimes *fœtor ex ore.* (*a*) *Pain in the stomach* is frequent; it may occur near the ensiform with tenderness, especially in nervous individuals. It sometimes results in vomiting; similar pains occur in the transverse colon. Sensitiveness at the tip of the twelfth costal cartilage is due to pressure from corsets. (*b*) *Dyspepsia* may be purely nervous. The stomach is exceptionally dilated and atonic, but as a rule no motor insufficiency occurs. (*c*) The hydrochloric acid is increased in 90 per cent. of cases, probably from nervous irritability. (*d*) *Constipation* is probably no more frequent than in the average case; intestinal fermentation may coexist, but it is not the cause of chlorosis.

5. Sexual Sphere.—The uterus infantilis and small ovaries are but coördinate with chlorosis. Menstruation is decreased or absent in 80 per cent.; menstrual disturbance is neither the cause nor result of chlorosis; both result from a common factor. Conception is unlikely to occur during chlorosis. Chlorotics are often first treated by the gynæcologist for leucorrhœa (in 22 per cent. of cases).

6. Special Senses.—The usual symptoms of anæmia, as spots before the eyes and weakness of accommodation, are observed. The retinal vessels may be transparent or pulsating; retinitis and neuro-

retinitis with degeneration and exudation, or "choked disk," occur in exceptional cases and simulate brain tumor. Retinal hemorrhage is rare.

7. Skin.—The skin is usually anæmic; angiospasm, coldness and paræsthesia occur. In chlorosis rubra the cheeks are flushed. Seborrhœa and acne are very common. Sweats, urticaria, eczema, purpura, chloasma and trophic disturbance of the hair and nails may occur.

8. Nervous System.—Chlorosis often brings out a latent *neurosis,*—especially hysteria,—and psychosis, the latter of which is frequently curable. Neuralgia is infrequent.

9. Metabolism.—Metabolism is not essentially disturbed. The body-weight is maintained, less from decreased oxygenation than from inactivity. Some cases are emaciated and occasionally an actual gain in weight is noted. The *urine* is clear, of low specific gravity, often alkaline with decrease of urea and uric acid and sometimes albuminuria. Leube found fever frequently in mild chlorosis. When present it is always well to differentiate "tuberculous pseudochlorosis."

Complications.—Chlorosis may induce *acute infections,* as typhoid, pneumonia, influenza and scarlatina, and may render their clinical course severe. Venous *thrombosis* complicates 2 per cent. of cases, occurring largely in the femoral veins, sometimes in the cerebral sinuses and axillary and other vessels. It is probably inflammatory. In Leichtenstern's series 56 per cent. involved the veins of the leg and 34 per cent. the cerebral sinuses. Pulmonary embolism occurred in 25 per cent. of 52 cases of thrombosis of the lower extremities (Welch). Virchow emphasized the frequency of septic endocarditis. Rheumatic endocarditis with organic valvular lesion is frequent, as also are ulcer of the stomach, tuberculosis and Basedow's disease. The *spleen* is enlarged in 16 per cent. (Leube). The thyroid gland is often moderately enlarged. *Hemorrhages* are not infrequent from the nose, stomach or intestine, although it is usually stated that they occur mostly in severe or pernicious anæmias.

Clinical Course and Prognosis.—Chlorosis develops rather suddenly and runs a relatively *short course* of two to four months. The prognosis is generally good. The tendency to *recurrence* is great, especially in unmarried or childless women; the blood may not return absolutely to normal, shown by persistent pallor or irregular menstruation. *Acute chlorosis* is observed especially in young girls after their first menstruation; it is attended by severe headache, nervous symptoms, great weakness and optic neuritis or "choked disk," which may be confused with organic disease, as brain tumor. The *periodic type,* according to von Noorden, occurs in the higher classes and in those who acquire the disease in their seventeenth to nineteenth year. The course may be *chronic;* the disease may last for years with chlorotic blood, menstrual, dyspeptic, muscular and nervous symptoms, with alternate exacerbation and amelioration, indicating inactivity or hypoplasia of the blood-making organs; this form

begins early in life in individuals with poor development of the bones, muscles, genitalia, mammæ and adipose tissue; marriage, pregnancy and iron often benefit these cases, but they may last until the menopause.

Diagnosis.—The diagnosis can be made by the entire group of symptoms, positive and negative, for no single symptom is pathognomonic. The diagnosis is based upon the *anæmia,* especially when there is disproportionate *reduction in the hæmoglobin,* when the color index is less than one and the white cells are normal; a diagnosis, however, cannot be made from the blood findings alone. (Rhomberg has described exceptional cases in which the blood was normal.) Chlorosis is *infrequent before the twelfth* and *after the twenty-fifth year,* unless the attack is a relapse. Its *rapid development* and *periodic manifestation* are important features. Successful *treatment* with iron is practically diagnostic. *Secondary anæmia* is most difficult to differentiate (see DIFFERENTIATION under PERNICIOUS ANÆMIA); it is usually attended by some leukocytosis. Anæmia from intestinal parasites, hemorrhage from latent ulceration of the stomach or bowel, renal disease, early pregnancy in unmarried girls, the anæmia of heart disease, incipient brain disease, rheumatism and lead and arsenic anæmias must be considered. *Splenic anæmia,* especially in children, requires differentiation; cases of chlorosis with enlarged spleen are regarded as a transitional form between chlorosis and splenic anæmia proper. *Malignant disease* usually causes little diagnostic difficulty after careful examination. There is sometimes difficulty in differentiation from incipient *tuberculosis.* Frequent physical examination and watching of the temperature usually establish the diagnosis.

Treatment.—1. PROPHYLAXIS.—Prophylaxis includes general hygiene and proper nourishment, in that development of adipose tissue to a reasonable extent is prophylactic. More albumin should be given to growing children than to adults, although fruit and vegetables, for their iron, and butter, cream and oils, rather than animal fats, should also be administered. Physical culture and trips to the country are valuable and constipation should be prevented. Education should not be hurried; all children at this age are confined too much to the school-room; they should be kept children as long as possible. In society and in the struggle for existence young girls too often overtax their strength.

2. SPECIFIC THERAPY.—Iron exists in the hæmoglobin of the blood cells as an oxygen carrier and is a food as well as a medicine; it is found in blood, meat, oatmeal, peas, spinach and beans.

Physiological Action of Iron.—(*a*) The tonus of the *nervous system* is increased, though an excess produces frontal headache from constipation; some persons exhibit a peculiar intolerance of iron, as shown by headache, vertigo and palpitation. (*b*) *Circulation;* locally astringent, it contracts the bloodvessels and increases the number of red disks and the amount of hæmoglobin. (*c*) Its action on the

gastro-intestinal tract is as follows: Iron stains the teeth because of the acid which it contains. Small doses stimulate digestion, improving, by their astringent action, the tone of the tissues. Hydrogen is generated and from it sulphureted hydrogen, which causes eructations. Small doses cause constipation but large doses relax the bowels; diarrhœa also sometimes follows small doses. Sulphide of iron is found in the dark stools. (*d*) The amount of urine is increased. Iron is found in the urine in very small quantities. The kidneys may be irritated by very large doses. (*e*) In regard to its absorption there is great conflict of opinion, but it is absorbed from the stomach and intestines as an albuminate; it reaches the blood and is deposited first in the spleen and then in the liver, whence it is then carried by the bloodvessels to the intestines for elimination. (*f*) Elimination occurs by the intestines, the kidneys and liver.

Administration.—It may be advantageously combined with quinine, particularly when the spleen is enlarged; it may also be given with oxygen, normal salt transfusions and massage; the patient should have rest and change of environment. Soluble preparations would seem to be better than insoluble ones, but Quevenne has shown that they are precipitated by the gastric juice. Headache is obviated by using laxatives, bromides or ferrous salts, which derange digestion less than ferric salts. Iron is said to be best absorbed when some gastric catarrh exists (Brunton), hence some practitioners give large doses at first to improve absorption. There are only 38 to 45 grains of iron in the body and the ordinary daily diet contains but one-sixth of a grain of iron, but the nucleo-albumin preparations, which are sufficient as food, are not sufficient as medication, because they do not stimulate the blood-making centres. The *rules for administration,* according to von Noorden, are: (*a*) The doses should not be too small (Niemeyer and Immermann). (*b*) Every physician has his own method, therefore no one form especially excels any other. (*c*) The administration should be regular, for irregularity is injurious. Statements of the patient that she has previously taken iron should not be regarded, for its administration may have been irregular. (*d*) Administration should continue for six weeks to three months. In the first week the dose should be gradually increased; for three weeks the maximum dose should be given and for the last two weeks it should be gradually decreased. (*e*) If iron is not beneficial it should not be repeated but other methods tried. (*f*) Relapses are less easy to treat than the first attack. (*g*) Idiosyncrasy, as gastro-intestinal disturbance, is only a relative contra-indication. The administration should then be subcutaneous. (*h*) At mineral springs hygiene is more important than iron; the water should be taken on an empty stomach, for the gas promotes absorption of iron. When bottled it is precipitated as a carbonate.

Iron may be given in the *following forms: Ferrum reductum* (reduced iron) which is iron gray (if it is black it is impure) and tasteless; it is least astringent, but may cause eructation; gr. j. (See

formula on page 417.) *Pilula ferri carbonatis,* a ferrous salt nearly free from astringency; grs. ij to v. *Ferri sulphas* (green vitriol), a ferrous salt which is very astringent, whence its use in chronic diarrhœa. It stimulates digestion. In large quantities it is toxic, causing vomiting or diarrhœa; gr. ij should be given with one dram of magnesium sulphate p. c. Ferri sulphas exsiccatus gr. ss to ij may also be given. Liquor ferri et ammonii acetatis (*Basham's mixture*) is a vascular tonic and diuretic; it is excellent for chlorosis in the obese; 3j to iv should be given after meals, well diluted. *Tinctura ferri chloridi* is the chloride of iron plus hydrochloric acid and alcohol, which develop hydrochloric ether. It is yellow-red, astringent, somewhat corrosive and diuretic, increasing the amount and frequency of micturition. It is used to best advantage when the tongue is white, flabby and indented. In weak, anæmic girls, with vomiting, neuralgia and epigastric pains, full doses should be given; when poorly tolerated, one drop in a full glass of water is more beneficial than chalybeate waters. *Syrupus ferri iodidi* is transparent and has a greenish color; iron and iodine given separately are usually more beneficial, but the syrup may be given in doses of 30 to 40 drops in adults and of 5 to 10 in children. *Ferri citras* in doses of gr. v, is efficacious in children and in persons having delicate stomachs. Ferri phosphas solubilis, gr. v, is almost free from astringency and taste. Most *organic preparations* are useless.

Arsenic may be given with the iron or in its place; it acts on the blood-making centres; it should be given as in pernicious anæmia (*q. v.*) uninterruptedly, with gradually increasing doses, sustained doses and then decreasing doses for a period of four to six weeks.

High altitude increases the number of red cells and the amount of hæmoglobin. Phlebotomy, sweating and organotherapy (bone marrow, splenic and ovarian extracts) have been suggested.

3. Dietetic Treatment.—Regular meals should be given at three-hour intervals. The amount of *albumin* should be increased, even in the obese. The heavy English breakfast with meat is the best one, because the albumins are absorbed more rapidly than fats and carbohydrates. In administering *milk* care should be observed lest the appetite be spoiled, lest too much fat be deposited and the stomach suffer atony. Equal parts of milk and cream should be given. Milk should be avoided in the obese. *Alcohol* is indicated in thin rather than in obese individuals. Patients should always rest before and after eating. The patient should retire early. In thin subjects the diet should be concentrated, consisting of albumin, vegetables, fats, alcohol and milk at breakfast and at bed time, but without increase of the carbohydrates. Iron produces better results in such cases after limiting the amount of water to about one quart. Sweats may produce the same result. When there is much gastric disturbance, eight meals may be given at two-hour intervals and fluid and solid meals should be alternated. Gastric disturbance disappears after administration of iron and increase in weight. Hydrochloric acid, strychnia and

belladonna are helpful. *Exercise* may be prescribed in convalescence, but during chlorosis the patient should rest in bed. Cool rubbings early in the morning are advantageous. Cold baths are avoided because they produce chills, insomnia and digestive disturbances. Marriage may help mild cases, but is injurious in the severe type.

PERNICIOUS ANÆMIA.

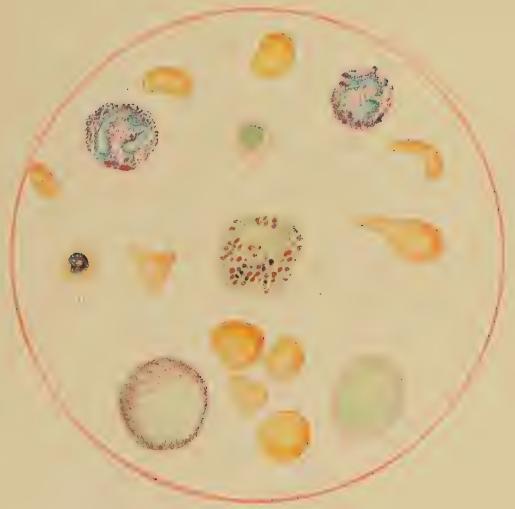
Definition.—A primary anæmia, in which are observed progressive diminution of the red cells, with changes in their form and color, and reduction in the hæmoglobin, without changes in the white cells; this form of anæmia is due to hæmolysis and to defective hæmogenesis; it is characterized by pallor, adynamia, œdema and cardiac symptoms and usually results in death from vomiting, diarrhœa, hemorrhages, fever and exhaustion. It was described by Andral (1826) and Channing of Boston (1832); Addison gave the best early description and Biermer later and more fully drew attention to the subject. It is sometimes called Addison's anæmia. Since Ehrlich drew the blood picture, little has been added to our knowledge of the disease.

Etiology.—The disease occurs in 0.2 per cent. of cases, especially in the *third and fourth decades* of life; six-sevenths of the cases occur after the thirty-fifth year (1,200 cases collected by Cabot, from whom the following figures are taken). Baginsky could collect but 16 cases in children. Sixty per cent. occur in males. The ultimate cause is to be sought in some *hæmolysin,* but its cause is unknown. Although called a primary or essential anæmia, some undoubted secondary cases may be included under this head, because the symptom complex is the same. Bothriocephalus and ankylostoma anæmias present identical blood findings, symptoms and pathology; not merely the parasite but also its toxins are causative. Pregnancy and the puerperium, in certain localities at least, are causative. Syphilis, poor hygiene, infectious diseases, as typhoid or malaria, and diarrhœa, vomiting and oral or gastro-intestinal sepsis have been considered factors.

Symptoms.—1. The Blood Changes (Sorenson, 1874).—The amount is decreased, several punctures often being necessary to obtain enough for the blood-count; it is often watery or dark or amber-colored in some instances. The specific gravity is low, even 1,027; the dry substance is reduced, even to 9 or 10 per cent. of the normal. There is an increase in the albumin in the individual red cell (hyperalbuminæmia rubra, von Jaksch). The serum does not contain free hæmoglobin, as has been asserted by Limbeck. If injected into normal individuals the serum has an hæmolytic action. In contrast to chlorosis and secondary anæmias, the *coagulability* of the blood is *decreased* and the blood may remain fluid for some time after death. Dropped on filter paper, there is a white zone outside the blood stain (Talquist). The *hæmoglobin* is decreased; it is rarely

PLATE XV

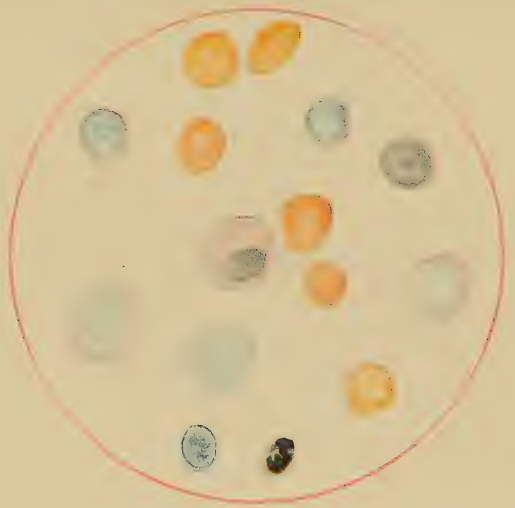
FIG. 1



Myelogenous Leukæmia.

1, myelocyte; 2, eosinophile myelocyte; 3, 3, eosinophile polymorphonuclears; 4, small, and 5, large lymphocyte; 6, nucleated red cell.

FIG. 2



Lymphæmia.

Large and small lymphocytes with various intensity in stain of nuclear chromatin.

FIG. 3

Pernicious Anæmia.

1, megalocyte; 2, microcyte; 3, 4, 5, malformed erythrocytes; 6, 6, normoblasts; 7, 7, 7, megaloblasts.

more than 50 per cent. of the normal and may be reduced to 10 per cent. In some instances severe symptoms may be present, with 65 to 70 per cent. hæmoglobin. In 90 per cent. of cases and in contrast to other anæmias, the *hæmoglobin per corpuscle* is proportionately *increased, i. e.,* the color-index is more than one (even 1.75). Laache's explanation is that certain red cells, especially giant forms, contain a *large compensatory individual amount* of hæmoglobin, which is also true of the microcytes, but less so of normal-sized red cells. Schmidt, Ponfick and others of the Dorpat school, maintain that the free hæmoglobin, acting on the white cells, produces a fibrin ferment, which causes fever, hemorrhage and capillary embolism. The *red blood corpuscles* are greatly reduced in number, the average being 1,200,000. (See Plate XV.) Patients often die when this figure is reached. Lower counts have been noted, as 143,000 (Quincke). The lowest count the writer has seen was 380,000. *Poikilocytosis* is marked, the irregularity in form and size of the corpuscles being great; great deformities, as Litten's horse-shoe or pessary-shaped corpuscles, are noted. In 90 per cent. the *red corpuscle* is *above the average size, i. e.,* 11, even 18μ instead of 5 to 8μ, the highest normal measurement. *Megalocytes* may be observed in number, with less marked or absent umbilication. Polychromatism is frequent. Megalocytes in pernicious anæmia are probably evidence of megaloblastic degeneration of the bone-marrow. (See Fig. VIII, Plate XVI.) *Megaloblasts* are found but rarely in large numbers, seldom more than six or eight being seen in a single preparation (Ehrlich); in rare cases they may be abundant, as just before death, whence their generally ominous significance; they may be absent for months or weeks at a time. *Normoblasts* occur in nearly all cases, especially in the "blood crises" of von Noorden, but are not numerous; they are immature cells and indicate activity of the blood-forming organs. Microblasts are frequently absent; their teleologic significance lies in the increase of the respiratory surface of cells thus afforded. Cell divisions are found. (Plate XVI, Fig. 3.) Granular deposits (staining with basophilic dyes) in the protoplasm are noted as in malaria, cachexia, leukæmia and suppuration; according to Ehrlich, they are not due to polychromatophile degeneration but to nuclear degeneration. The cells may stain only at their margins. Decreased formation of rouleaux is observed as well as amœboid movement of the red cells. The *white corpuscles* are decreased absolutely, the lowest record being 1,500; the polymorphonuclears are decreased. Leukocytosis is a distinct complication, is often ominous and indicates inflammation or suppuration. Apart from complications, the lymphocytes are relatively increased, even to 62 per cent. of the total white cells. The leukocytes are readily increased in children in any variety of anæmia. Marked decrease of the eosinophiles is an unfavorable sign (Neusser). Pernicious anæmia due to parasites shows increase in the eosinophiles from 1 to 4 (normal) to 6, 36 and even 50 per cent. (See Plate XVI, Fig. V.)

2. Subjective Symptoms.—The most marked are general weakness, muscular adynamia on exertion, vertigo, palpitation, dyspnœa and the ordinary signs of anæmia.

3. Skin.—The skin presents a *peculiar pallor,* often of a lemon color, which is somewhat characteristic. Slight icterus is not infrequent. The skin, usually dry, may become drenched upon exertion. *Œdema is invariable,* especially about the ankles and eyelids. It is usually not marked, but is obstinate and may produce pronounced general anasarca; it is due to vascular degeneration and permeability. Hemorrhages, usually punctate, are frequent in the skin and mucosæ. Large hemorrhages occur in 25 per cent. of cases. Pigmentation is sometimes as marked as in Addison's disease. The author once saw herpes zoster and once herpes along the crural nerve. Sometimes before death the skin emits a foul, cadaverous odor (Eichhorst).

4. Metabolism.—The nutrition is maintained in 61 per cent. of cases because tissue waste is lessened and water is retained in the tissues; in 39 per cent. (Cabot) the weight and strength finally decline with persistent vomiting and diarrhœa. Opinions differ as to the destruction of the albumins. In the *urine* indican, putrescin, cadaverin, leucin and sometimes tyrosin, acetone and albumose are found. Albuminuria, due to tubular degeneration, is frequent. Urobilin is frequently increased, sometimes imparting a dark color to the urine, and indicates hæmolysis. The chlorides are decreased. The iron in the urine and uric acid are sometimes increased and the phosphates are greatly increased. *Fever,* present in 80 per cent. of cases, bears no constant relation to the severity of the disease. It may rise considerably; the temperature may fall below normal in the ante-mortem coma. The type is irregular. Its cause is ill-understood; it is probably due to the production of a ferment from the destroyed red cells or, according to Schmidt, Ponfick and others, from a fibrin ferment formed from the leukocytes (*v. s.*).

5. The Circulation.—*Palpitation, precordial anxiety* and *dyspnœa* are observed, sometimes only on exertion. The heart impulse is fallaciously strong to the palpating hand. Its outlines are usually normal, save that dilatation of the right heart or hydrops pericardii may develop. Almost always a blowing systolic murmur is heard, usually with a clear diastolic tone; the murmur is rarely presystolic or diastolic. The second pulmonic sound is variable, sometimes accentuated. No parallelism exists between the degree of anæmia and the loudness of the murmur. Leube reports cases of chlorosis and pernicious anæmia in which there was relative tricuspid insufficiency; the positive venous pulse was present, but no cyanosis. A venous hum in the neck, carotid pulsation and thrills, a loud, clear systolic tone and murmurs and double tones over the peripheral arteries are often present. The *pulse* is collapsing and decreased in tension and its *rate increased,* being rarely below 80 and usually between 90 and 120. *Anatomically* the heart shows vestiges of hemorrhage, degeneration and atrophy, from hæmolysis. The *fatty,* "tabby-cat heart"

is almost constant. Degeneration in the smaller vessels has been noted, especially in the brain.

6. Digestive Tract.—Anorexia, distaste especially for meats, gastric achylia, pain and sensitiveness throughout the alimentary tract are noted. The tongue and gums are sensitive and circumscribed or punctate hemorrhages are often noted in them. Pain in the mouth (50 per cent.) and œsophagus is due to the anæmia or administration of arsenic. The gums may ulcerate; erythema bullosum and other eruptions may occur. *Vomiting* is frequent in various stages of the disease and is often continuous or incoercible; the vomitus consists of food, greenish mucus and sometimes traces of blood. The writer has observed profuse hæmatemesis and cyclic vomiting as the first symptoms. *Crises* of abdominal pain occur in 60 per cent. Epigastric pulsation, moderate abdominal distention and achylia are observed. The author once saw expansile abdominal pulsation, wrongly diagnosticated as aneurysm. The glands in the gastro-intestinal tract may atrophy, which probably toxæmic condition is coördinate with and not causal of pernicious anæmia. The mesenteric glands are not infrequently enlarged. *Paroxysmal diarrhœa* occurs in 50 per cent., constipation in 35 per cent. and an alternation of these conditions in 15 per cent. The stools are not characteristic, although sometimes acholic from poor absorption of fat. Parasites or their eggs may be noted. Leucin and tyrosin crystals are frequently caused by diarrhœa, but are not characteristic of the disease. Fermentation in the bowel (with indican in the urine), some œdema, hemorrhage and follicular swelling in the bowel are frequently observed. Some ascites is found in the terminal stage. A deposit of iron (siderosis) in the liver, in the outer and middle zones of its lobules, in the spleen, bone marrow, kidneys and lymphatic glands is usual and results from hæmolysis; it is very unusual in other anæmias. There is most hepatic siderosis in acute cases. The liver is slightly swollen and tender in one-third of the cases.

7. Blood-making Organs.—The *lymph glands* are not usually enlarged. The prevertebral hæmolymph glands have been carefully studied by Warthin, who found their sinuses dilated and evidences of hæmolysis in their tissue, shown by increased phagocytosis and sometimes hyperplasia of the glands; Warthin holds that the unknown poison stimulates the phagocytes of the spleen, lymph nodes and hæmolymph glands to increased hæmolysis. The *bones are tender,* especially over the sternum and tibiæ and sometimes painful and their fatty marrow may become lymphoid (leucoblastic degeneration); this transformation may occur in other forms of anæmia, in cachexia or in fevers; in pernicious anæmia the red cells of the marrow are abnormally large, abnormally dark from increase of the hæmoglobin and are oval rather than round; the megaloblastic degeneration develops at the expense of the other elements in the bone marrow. These probably secondary bone changes were first described by Wood, Pepper and Tyson (1875). The *spleen* is not enlarged

save from accidental causes; in it are found phagocytosis and evidences of hæmolysis.

8. Sexual Sphere.—The menses cease, otherwise no characteristic changes are observed.

9. The Nervous System.—The psychical functions are decreased, the memory is weak and psychoses, delirium, hallucinations, mania and intractable insomnia may develop either temporarily or antecedent to the terminal coma. Sometimes improvement is noted after severe mental symptoms or coma. The pupils are rather wide and sometimes their reflexes disappear. Paresis, apoplectiform attacks, cramps, convulsions, paræsthesia, aphasia, vertigo or in women and children the hydrencephaloid state may develop. The *cord* is more frequently involved (in 84 per cent. of cases) than the brain. The weakness may amount to paresis, not due simply to muscular adynamia but to spinal changes. The picture may resemble tabes as concerns the eyes, patellar reflexes, ataxia, bladder and rectal changes, pains, anæsthesia and paræsthesia; or, less often, the symptoms of spastic spinal paralysis prevail, as increased reflexes and spastic paresis. In one group of cases the nervous symptoms are slight, and in a second they are so conspicuous as to even overshadow the anæmia itself. Early paræsthesia occurs in all cases, usually in the hands and feet, sometimes in the mouth; sometimes there is hyperæsthesia or disturbance of the temperature sense, spasticity and ataxia. The sexual function is decreased in males and amenorrhœa is regular in women. There is a sclerosis of the posterior and lateral columns, most marked in the lower cervical and upper dorsal segments, not always diffuse but sometimes focal, and involving chiefly the white substance, but not the entire neuron. Degeneration is found in the upper part of the cord or possibly throughout its length. The white substance suffers more extensively than the gray; the changes usually occur in the posterior column, which rarely wholly escapes. Small foci of degeneration with secondary connective tissue growth occur about the bloodvessels, which foci may fuse into plaques of small size with secondary degeneration, hence the symptom complex of a systemic disease.

The discrepancy between the anatomical and clinical findings is remarkable, *i. e.,* there may be many symptoms with few autopsy findings and the converse is also true; consequently it is probable that the spinal and nervous symptoms are less sequences of anæmia than coördinate toxic manifestations. The peripheral nerves present few symptoms. Anatomically, capillary hemorrhages and disseminated foci of softening may be observed in the brain and meninges.

In *the eyes* the general symptoms of anæmia occur; hemorrhages in the retina are almost constant; occurring on a pale background, with tortuous veins and a strong light reflex from the vessels, the hemorrhages form a striking picture (see Plate VIII, Fig. 9). They are punctate and their resorption is infrequent. The other senses are obtunded; labyrinthine hemorrhage with vertigo is recorded.

Clinical Course and Prognosis.—The pallor and weakness which are first observed increase insidiously and later dyspnœa, weak heart-action, œdema, complete anorexia, langor, spinal and violent gastro-intestinal disturbance and finally capillary hemorrhages and fever develop; this vomiting and diarrhœa may at times resemble acute poisoning. In other instances remissions may occur with treatment or independently of it, perhaps after violent gastro-enteric manifestations. The symptoms may partially or wholly disappear, as in Ehrlich's case, where the red cells in seventeen days rose from 1,340,000 to 4,115,000. According to Laache, in simple anæmia the red corpuscles gain more rapidly than the hæmoglobin, but in pernicious anæmia improvement increases both equally. The disease with remissions may cover a period of years, the longest duration being 15 years. Death usually occurs within half a year. The writer observed recovery for five years after a count of 400,000.

The *prognosis* is usually poor when many megaloblasts are present or when the color index is over one. In some cases the blood is rapidly regenerated.

Cabot's description of the disease recognizes *two forms:*—

	THE SEVERE, RAPIDLY FATAL TYPE	AND THE LESS SEVERE, SLOW TYPE.
(*a*)	Extreme progressive anæmia.	Remissions.
(*b*)	Color index high.	Normal or low.
(*c*)	Red cells increased in size.	Normal.
(*d*)	Degenerative changes.	None.
(*e*)	Many megaloblasts.	Many normoblasts, few megaloblasts.
(*f*)	Lymphocytosis.	Normal ratio of the various white cells.

Death usually occurs in coma or possibly from cerebral hemorrhage or other rare complications. Pye-Smith collected 20 cases of recovery; there are only 6 recoveries, according to Cabot, yet he found 27 cases which lived 8 or more years. The mortality in the parasitic form is 17 per cent.

Diagnosis.—No single finding is absolutely characteristic, though the pallor, apathy, adynamia, œdema, weak heart, retinal hemorrhages, maintained nutrition and the blood findings are highly suggestive. The yellow color is very often mistaken for icterus. Ewing states no importance should be attached to the increased diameter of the red cells unless 33 per cent. of the red disks show it. Megaloblasts are highly suggestive. The diagnosis must be made from the negative as well as from the positive findings.

DIFFERENTIATION.—(*a*) From *metastatic bone-tumors,* in which the white cells are increased (not merely the polymorphonuclear but the neutrophile and eosinophile marrow cells); (*b*) from *parasitic anæmia,* for which the stools should be examined for parasites and their eggs; (*c*) *fever,* if dominant, may suggest typhoid or meningitis; acute endocarditis may be simulated by the fever, anæmia, heart murmurs and cardiac insufficiency of pernicious anæmia; (*d*) from *latent carcinoma,* the gradual cachexia, anorexia, nausea, vomiting, hæmatemesis and decrease or absence of hydrochloric acid may

suggest gastric carcinoma, but the blood examination with thorough physical examination usually determines the point; (*e*) *spinal disease,* Addison's disease and hemorrhagic pleuritis are usually excluded with ease; and (*f*) *from other anæmias.* The following is constructed after Cabot's table:

	Pernicious Anæmia.	Chlorosis.	Secondary Anæmia.
Red Cells Number.	Average number 1,000,000.	Rarely under 2,000,000.	Findings as in chlorosis May be 1,000,000 or less, though rarely.
Shape.	Poikilocytosis great, sausage-shape, etc.	Somewhat deformed.	
Size.	Average increased, some dark.	Diminished, light colored.	Various, not increased.
Hæmoglobin.	Increased per corpuscle yet many Ponfick's shadow corpuscles.	Reduced per corpuscle.	
Staining	Polychromatophile cells.		
Nuclei.	Micro-, megalo-, normoblasts. Megalo- more numerous than normoblasts and constitute majority of nucleated R. B. C.	Megaloblasts rare though recorded. Normoblasts more than megaloblasts.	Normoblasts far more than megaloblasts in malignant diseases. Normoblasts common (more than in chlorosis).
Rouleaux.	Not present, or decreased.		
Color index.	High color index (not in chronic or mild cases).	Index less than 1; always low; lower than in secondary anæmias.	Relatively low (this rule does not hold positively).
Leukocytes.	W. B. C. decreased, except in blood crises, hemorrhage, suppuration.	Usually normal.	Polymorphonuclear leukocytosis in malignant disease, when R. B. C. are low in number, *e. g.* one million, speaks for secondary anæmia; positive of more value than negative findings (*i e.* no leukocytosis).
Lymphocytes.	Lymphocytosis (relative).	Lymphocytosis	Rarely increased (no decrease).
Myelocytes.	Small number only.	Rare; less than in pernicious anæmia.	Myelocytes very rare.
Adult Leukocytes	Rarely increased. Usually decreased.	Decreased.	Usually increased.
Coagulation	Slow.	Rapid.	Often rapid.

Aplastic anæmia, generally regarded as a type of pernicious anæmia, is characterized by its low blood count and reduced hæmoglobin. It was so named by Ehrlich because the bone marrow was aplastic; therefore in the blood there are no evidences of regeneration, such as nucleated red cells. The white cells may number only 200 per c.mm. and 80 to 90 per cent. of them are lymphocytes. Two-thirds of the cases are women. There may be hemorrhages, fever or hyperplasia of the spleen and lymph glands. The outcome is rapidly fatal, 80 per cent. dying within three months.

Fig I

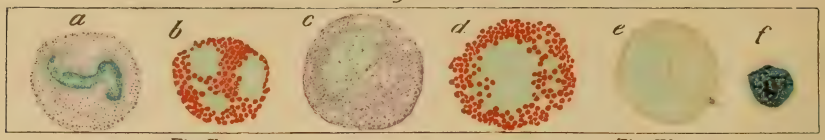


Fig II.

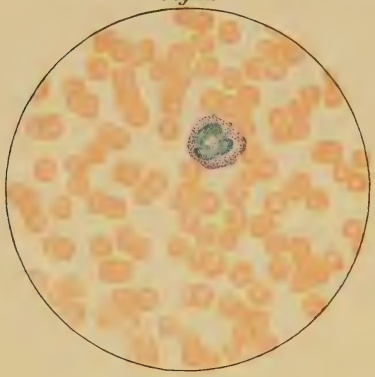
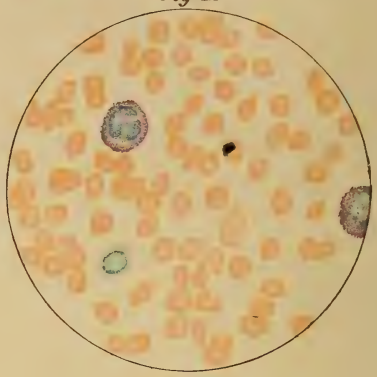


Fig III



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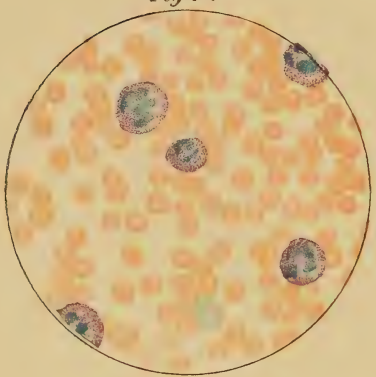


Fig.V.

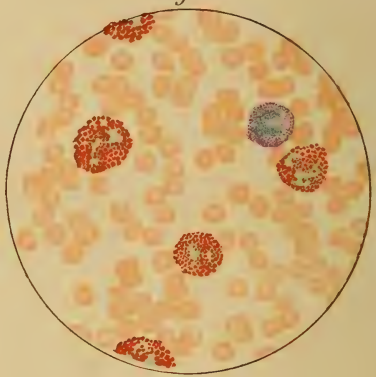


Fig VI.

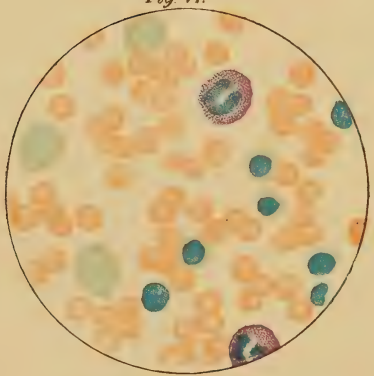


Fig.VII.

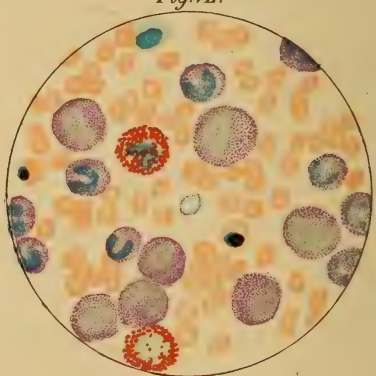
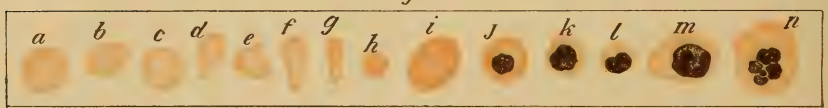


Fig.VIII.



DRAWN BY J.N.Z. CHASE

PLATE XVI

BLOOD.

(Ehrlich triple stain.)

(Prepared by Dr. I. P. Lyon.)

Fig. I. TYPES OF LEUCOCYTES.

a. Polymorphonuclear Neutrophile. *b*. Polymorphonuclear Eosinophile. *c*. Myelocyte (Neutrophilic). *d*. Eosinophilic Myelocyte. *e*. Large Lymphocyte (large Mononuclear). *f*. Small Lymphocyte (small Mononuclear).

Fig. II. NORMAL BLOOD.

Field contains one neutrophile. Reds are normal.

Fig. III. ANÆMIA, POST-OPERATIVE (secondary).

The reds are fewer than normal, and are deficient in hæmoglobin and somewhat irregular in form. One normoblast is seen in the field, and two neutrophiles and one small lymphocyte, showing a marked post-hæmorrhagic anæmia, with leucocytosis.

Fig. IV. LEUCOCYTOSIS, INFLAMMATORY.

The reds are normal. A marked leucocytosis is shown, with five neutrophiles and one small lymphocyte. This illustration may also serve the purpose of showing the leucocytosis of malignant tumor

Fig. V. TRICHINOSIS.

A marked leucocytosis is shown, consisting of an eosinophilia.

Fig. VI. LYMPHATIC LEUKÆMIA.

Slight anæmia. A large relative and absolute increase of the lymphocytes (chiefly the small lymphocytes) is shown.

Fig. VII. SPLENO-MYELOGENOUS LEUKÆMIA.

The reds show a secondary anæmia. Two normoblasts are shown. The leucocytosis is massive. Twenty leucocytes are shown, consisting of nine neutrophiles, seven myelocytes, two small lymphocytes, one eosinophile (polymorphonuclear) and one eosinophilic myelocyte. Note the polymorphous condition of the leucocytes, *i.e.*, their variations from the typical in size and form.

Fig. VIII. VARIETIES OF RED CORPUSCLES.

a. Normal Red Corpuscle (normocyte). *b, c*. Anæmic Red Corpuscles. *d-g*. Poikilocytes. *h*. Microcyte. *i*. Megalocyte. *j-n*. Nucleated Red Corpuscles. *j, k*. Normoblasts. *l*. Microblast. *m, n*. Megaloblasts.

Leukæmia (see DIFFERENTIATION OF LEUKÆMIA).

Treatment.—1. HYGIENIC.—The patient should have absolute rest in bed and if possible should be in a warm climate. An excessive nitrogenous diet is unwarranted because it increases tissue waste, patients have an instinctive disinclination to nitrogenous foods and albumins frequently produce diarrhœa. A vegetable diet is better tolerated, with bread, zwieback, beef tea, peptones, milk, honey or porridge. Nutrient enemata are indicated in the gastric crises. In pregnant women labor should not be induced.

2. HÆMATINIC.—(*a*) *Transfusion* of defibrinated human blood has been practiced, though seldom beneficial. Human blood may be transfused *in toto.* (*b*) Lépine recommends the physiological *salt solution,* based on Maragliano's findings that the serum in cases of pernicious anæmia has a globulicidal action on the red blood cells. The serum destroys the hæmoglobin in pernicious anæmia (the blood of a patient clears in a test tube on standing). Salt solution should be given frequently and acts temporarily on the serum. (*c*) Phosphorus is without value and iron is often injurious. (*d*) *Arsenic,* recommended by Bramwell, is said to have cured the disease, though most cases have not been followed out thoroughly; it is, nevertheless, the best remedy known, probably acting upon the blood-forming centres. Birsch-Hirshfeld explains its action by assuming that it operates in some way upon the serum, while Litten holds that it increases the number of white cells.

Physiological Action of Arsenic.—(*a*) The smallest dose merely increases the appetite. (*b*) Small doses stimulate the appetite by gastric irritation, by increasing secretion and stimulating peristalsis; tissue waste and elimination of nitrogen are lessened. It is an alterative, increases the muscular strength and endurance, improves the quality of the blood, the complexion, sexual powers and lung capacity. (*c*) Continuous administration produces constitutional effects, as puffiness of the eyes, first seen in the morning (Wier Mitchell); this may be followed by general œdema, due to vascular and sometimes renal alteration. Administration should be stopped when puffiness is observed. (*d*) Large doses result in anorexia, nausea, diarrhœa, abdominal uneasiness or colic and sympathetic headache.

The circulation is stimulated by small doses. Arsenic is absorbed by the bloodvessels. Full doses lessen the force and rate of the heart, for they act directly upon the heart muscle. Arterial pressure is reduced by peripheral vasomotor paralysis. Respiration and digestion are stimulated by small and depressed by large doses, although some deny the stimulant action of any dose. Elimination is very rapid and mostly by the kidneys and intestinal mucosa, but also by the skin, bile, lungs, saliva and mother's milk.

Administration.—Children tolerate large doses, though the aged stand the drug poorly and their digestion suffers; after the fifth year adult doses are given; girls tolerate the drug better than boys. When susceptibility is marked a few drops of laudanum may be added, but

its administration should always be stopped when (*a*) the eyes become puffy or (*b*) itch and (*c*) the tongue becomes furred. It may be given in the following forms:—(i) Arseni trioxidum gr. ⅓₀ p. c., with ext. glycyrrhizæ and piper (black pepper) āā gr. iv ("Asiatic pill"). (See formula on page 417.) (ii) *Liquor potassii arsenitis* (Fowler's solution, 1786); for hypodermic use it must be sterilized, because fungi are likely to grow in it. Even with great care abscess may develop. Minims v equal gr. ⅓₀ of arseni trioxidum. Treatment should commence with ♏iij and each day the dose should be increased a drop until ♏x or xij are given; rarely can larger doses (♏xv–xxv) be given. (iii) The cacodylate of soda pill (gr. ⅙) may be given one to five times daily (Gautier).

3. Symptomatic Treatment.—This includes opiates for diarrhœa, cardiants for weak heart and dilute HCl for gastric achylia.

SECONDARY ANÆMIA.

I. Acute Post-Hemorrhagic Anæmia.—Etiology.—It may result (*a*) from trauma of large vessels; (*b*) from post-partum hemorrhage, abortion or tubal pregnancy; (*c*) from hæmoptysis, aneurysm, cardiac disease or tuberculosis; (*d*) from carcinomatous ulcerations in the uterus, gastro-intestinal tract, etc.; (*e*) from round ulcer of the stomach or duodenum; (*f*) from varicosities, as in the leg, stomach or œsophagus and liver cirrhosis; (*g*) from blood conditions, hemorrhagic states or constitutional diseases.

Symptoms.—1. *The blood* is (*a*) *hydræmic,* the loss of fluid being supplied by the tissues, and its specific gravity is lowered. (*b*) The *red disks* are reduced to 50 or even to 20 per cent., are often irregular in contour (poikilocytosis), pale and poor in hæmoglobin; their diameter may be increased; generally there are some normoblasts. (*c*) The *hæmoglobin* is lessened even more than the red cells. (*d*) The leukocytes at first show a moderate increase; the polymorphonuclears are decreased and the mononuclears increased. In sudden anæmia with rapidly resulting death the red cells are seldom nucleated and there is no leukocytosis, because the bone marrow makes no effort to regenerate the blood. (See Plate XVI, Fig. 3.)

2. There is *marked anæmia* of the *skin and mucous membranes.* Some œdema results from chemical changes in the blood or malnutrition and increased permeability of the vessel walls. The pulse is weak and becomes frequent with the least exertion. Nevertheless, the vasomotor system accommodates itself to a loss of one-quarter of the blood volume; a greater loss produces cardiac weakness. Sometimes the heart action seems more violent and respiration deeper and more rapid than normal.

3. *Albuminuria.* After a large hemorrhage no obvious microscopic change in the renal epithelium is detected, whence it seems that albuminuria is due to undue permeability of the renal vessels.

4. The *nervous* and *eye symptoms* may be marked. Amblyopia

and amaurosis, known to Hippocrates, are at first functional, but usually later degeneration or inflammation of the retina develops; as the blood pressure rises, retinal hemorrhages may follow. The patient sees spots before the eyes. Occasional nervous manifestations include aphasia or vertigo; delirium and hallucinations are chiefly terminal events; in some cases lasting psychoses develop.

5. The *autopsy findings* in fatal cases are fatty degeneration, particularly of the heart, endothelium of the vessels, liver, brain, stomach and kidney cells, which is usually caused by imperfect oxygenation. Hemorrhages in the uterus, gums and retina are due to impaired nutrition of the vessel walls. The bone marrow changes from a yellow to a red color and abounds in normoblasts.

Prognosis.—The patient may survive the loss of more than one-third or one-half of the blood; Hayem reports recovery after the number of red cells fell to 550,000. Men stand hemorrhage better than women and young children; the loss of a pint may cause death in arteriosclerotics. Recovery is more rapid in acute than chronic hemorrhage. In moderately severe hemorrhage recovery may be complete within a week; water and albumin are readily yielded to the blood by the tissues; the red cells regenerate more slowly and the hæmoglobin is the last element to reach normal. Increase in the coagulability of the blood is a favorable prognostic. According to Bierfreund, a loss of 10 to 15 per cent. of the blood is restored in eight days; of 16 to 20 per cent., in twenty days; of 21 to 25 per cent., in twenty-two days and of more than 25 per cent., in twenty-nine days or more.

Treatment.—This has been considered under gastric ulcer and intestinal hemorrhage in typhoid, *viz.*, absolute rest, morphine to insure quiet and salt infusions only in extreme cardiac failure.

II. Chronic Secondary Anæmia.—Etiology.—Its possible causes are numerous; the chief types are (*a*) *hemorrhages* like those listed under acute post-hemorrhagic anæmia, but frequent and persistent, as chronic hemorrhoidal bleeding. (*b*) *Poor hygiene;* overwork, long hours in poorly-ventilated, sunless offices or factories; insufficient food or food poor in iron. (*c*) *Exhausting discharges or loss of albumin,* as frequent pregnancies with prolonged lactation, albuminuria, respiratory or alimentary catarrh or protracted suppuration, with its attendant loss of nuclein. (*d*) *Acute infections,* as typhoid, diphtheria, rheumatism or malaria, in which the activity of blood-making centres is diminished and food is poorly assimilated; chronic infections, as syphilis and tuberculosis. (*e*) Gastro-intestinal and possibly other *intoxications.* (*f*) *Neoplasms* acting as mechanical obstructions (as those of the œsophagus or pylorus), affecting sometimes the bone marrow by metastases deposited there, sometimes causing repeated losses of blood or elaborating toxins, which break down the tissue albumins; in neoplasms the reduction of the blood in red cells and hæmoglobin averages 55 to 66 per cent. of normal. (*g*) *Parasites* (oxyuris, ascaris, echino-

coccus, etc.); and (*h*) *poisons* (alcohol, lead, arsenic or phosphorus) may produce anæmia.

Symptoms.—The *blood is pale* and its specific gravity is lowered. As a rule the red disks and hæmoglobin are reduced in rather close parallelism. The hæmoglobin may fall to 18 or 14 per cent. of normal and the red cells may, in rare instances, approach the count of pernicious anæmia. The red cells stain unequally and their umbilication is clearer, so that the hæmoglobin may even appear as a slight peripheral ring. They show less poikilocytosis than in acute post-hemorrhagic anæmia and many of them are undersized. Some nucleated erythrocytes appear, but less than in the acute post-hemorrhagic form. The white cells vary with the causal conditions. As a rule the polymorphonuclear neutrophiles are increased and the eosinophiles (normally 4 to 6 per cent.) are decreased. The latter are often increased in parasitic anæmias, up to 10 or even 50 per cent. In some chronic anæmias the leukocytes are diminished.

As to *general symptoms* it is frequently difficult to decide whether they result from the anæmia or from its cause. An anæmic skin, muscular adynamia, irritable weakness and a tendency to œdema and hemorrhage into the skin, mucosæ and serosæ may be noted. The appetite is poor and tenderness over the stomach, nausea and decrease or increase of the HCl may be observed. The intestinal functions are usually normal, though the motor power may be tardy. The aromatic sulphates of the urine are frequently increased. Exceptionally symptoms resembling those of pernicious anæmia may develop, as alteration of the bone marrow, fatty heart, cerebrospinal degeneration, atrophy of the gastro-intestinal cells and retinal hemorrhage. For *differentiation* see PERNICIOUS ANÆMIA.

Treatment.—The treatment depends upon the cause, for which thorough and repeated search should be made. The hygiene of the home and place of work should receive consideration. Fresh air, sunlight and proper food are often difficult to obtain for the lower classes. Iron and arsenic are valuable in simple types (*v.* CHLOROSIS AND PERNICIOUS ANÆMIA).

LEUKÆMIA.

Definition.—Leukæmia (leukocythæmia) is a disease of hæmogenesis, characterized by an increase of the leukocytes, a decrease in the red cells and hæmoglobin, hyperplastic alteration of the bone marrow, lymph glands and spleen and by secondary infiltration into various organs and tissues. It was discovered by Bennett and independently by Virchow (1841).

Classification.—The older classification embraced (*a*) the splenic and (*b*) the glandular (Virchow) types to which Neumann added (*c*) the medullary form; Béhier described a gastro-intestinal form and Phillippart, Nachter and Kaposi a dermal variety. This classification is clinical, but that of Ehrlich is based on the blood findings; he classifies the disease as follows:

I. Lymphatic leukæmia. (i) The acute form, which runs a very rapid course and resembles an acute infection, with lymphæmia, moderate or no splenic enlargement, fever, hemorrhages, lymphatic enlargement and stomatitis. (ii) The chronic form, with lymphæmia, enlarged spleen and glands and a protracted course.

II. Myelogenic leukæmia with great polymorphism in the blood.

I. Acute Lymphatic Leukæmia.—Acute lymphatic leukæmia (*acute lymphœmia*) includes nearly all acute leukæmias. The term does not imply that the lymph glands are the cause, because there is growing evidence that the bone marrow may be the seat of the causal change and that it may contain a parent cell for both leukocyte and lymphocyte. The chief pathological finding is *hyperplasia of the lymphadenoid tissue.* The course is that of a *rapid infective disease,* usually with an acute onset, lymphæmic blood findings, asthenia and often fever; it is fatal in a few days to a few weeks. The disease occurs at any age, but especially in *children,* and 66 per cent. of cases are in males. The bacteriological findings are inconstant; the amœba described by Löwit is considered an artifact by hæmatologists. The disease is infrequent.

Symptoms.—The prodromal symptoms may be headache, pain in the neck, spleen, joints or head, epistaxis, mental obscuration, dyspnœa or stomatitis, after which four cardinal findings develop:—

1. Lymphadenoid Changes.—*Lymph gland* intumescence is very frequent, is usually moderate and sometimes disappears after the initial swelling; *splenic enlargement* occurs in 66 per cent. and is of slight degree; in children it may be enormous; *tonsillar* hypertrophy occurs in 50 per cent., sometimes with hemorrhage and necrosis. Reed published a case of acute lymphæmia without glandular enlargement.

2. Hemorrhages.—Hemorrhages are most characteristic and frequent; they occur (*a*) in the skin, as petechiæ or larger effusions, with necrosis or ulceration in areas of lymphæmic infiltration; (*b*) in the mouth, gums, palate and pharynx, as petechiæ, erosions or extensive necrosis, developing in areas of lymphæmic infiltration; (*c*) in the retina in practically 100 per cent. with the same anatomical basis; (*d*) visceral hemorrhages, in the brain, cerebral nerves, labyrinth and spleen; (*e*) from mucous surfaces, the vagina, urinary passages and intestines; and (*f*) from the serous membranes.

3. Necrosis.—Necrosis in the infiltrated foci of the mouth occurs in 70 per cent. and is *per se* highly suggestive in the diagnosis (Bradford and Shaw).

4. Lymphæmia.—Lymphæmia is the *crucial finding.* The ratio between the white and red cells is 1 to 2, 10 or 20; the number of lymphocytes may reach 918,000 to the cubic mm. In general, the increase of leukocytes is less than in the splenomedullary form and averages 350,000. The polymorphonuclears are usually normal or decreased, the eosinophiles constitute about 1 per cent. of the white cells, a few myelocytes may be found, but the salient increase is in the lymphocytes and chiefly in the larger forms; the lymphocytes may

constitute 90 or even 99 per cent. of the leukocytes. Some very large primordial cells are found, from which the red and white cells are probably derived. The red cells vary between 1 and 3 million and some nucleation may occur. The hæmoglobin usually falls below 50 per cent., or much lower in peracute cases. (For varieties of leukocytes, see Plate XVI, Fig. I. For blood picture see Plate XV, Fig. 2, and Plate XVI, Fig. VI.)

Other findings are pallor, frequent pulse, priapism, exophthalmos, swollen, infiltrated liver, increased flow of urine, with increased urea and uric acid (increased nuclein destruction); albuminuria, nephritis and endocarditis are sometimes observed. Lymphomatous masses may cause dyspnœa. The fever is irregular, both in type and occurrence. Intercurrent sepsis reduces the splenic and lymphatic enlargements, the white cells decrease even to normal and sometimes an ordinary polymorphonuclear leukocytosis replaces the typical blood findings. The *bone marrow* is *constantly involved,* especially in the long bones; it is mostly red and jelly-like in color, more rarely red-gray or hemorrhagic. The typical granular neutrophile myelocytes are few, while large and small lymphocytes abound as in the circulating blood; this is known as "the lymphoid marrow." Opinions vary as to whether the lymphatic or the medullary lesions are the primary change. Lymphatic deposits occur in the mouth, stomach, intestines (simulating typhoid ulcers), muscles, liver, kidneys and thymus. In a personal case the teeth were hidden by the infiltrated gums. The patient usually emaciates. Fatty and degenerative changes in the heart and other viscera are observed.

Diagnosis.—Though the splenic and lymphatic enlargements, petechiæ, stomatitis, fever, ulceration and hemorrhages are frequently most suggestive, the blood findings alone are distinctive; they exclude typhoid, purpura, scurvy, trichinosis, diphtheria, septicæmia, severe acute anæmias and chloroma (*v. i.*).

II. Chronic Lymphatic Leukæmia.—Chronic lymphatic leukæmia is rarer than acute lymphæmia and runs a clinical course of from months to years, with lymphadenoid hyperplasia and lymphæmia; it is closely associated by some writers with malignant lymphoma and Hodgkin's disease.

Etiology.—No constant bacteriological cause has been proven.

Symptoms.—The symptoms begin insidiously and the disease may be first detected by (1) swelling of the *lymph glands,* usually in the neck, which may reach enormous dimensions. The axillary glands are large, while the inguinal are less massive; glandular swelling, as a rule, extends from above downward. Sometimes the internal glands alone are swollen and in rare cases the lymphatics are not enlarged. The glands are discrete, without periadenitis, oval and soft, being much less hard than in pseudoleukæmia. Sometimes moderate pain is experienced and there may be compression of vital structures, as the vagus, trachea and portal vein. The glands continue to grow until death, unless an intercurrent affection, as erysipelas, causes them to shrink temporarily.

2. *Splenic hyperplasia* and intumescence is practically constant, though less extreme than in the myeloid type or in Hodgkin's disease. The spleen often projects from beneath the costal arch but an inch or so, it grows as the disease progresses and is usually proportionate to the duration of the disease. In a case of Virchow the spleen was not enlarged. Intercurrent sepsis, cholera, erysipelas, etc., may cause the splenic tumor, lymphæmia and the lymph nodes to temporarily disappear, but all three findings may persist in such infections. Diarrhœa and treatment by arsenic or quinine may likewise effect splenic reduction. Perisplenic adhesions and thickening are common and the organ is soft or hard according to the duration of the disease. The tonsils are often swollen, in which hemorrhages, with or without necrosis, are frequently observed.

3. The changes in the *bone marrow* seldom cause pain. The essential character of the marrow alteration is undetermined.

4. The *blood* reveals an *absolute and a relative lymphæmia.* The red and white cells may have a ratio of 100 to 1, and yet the lymphocytes constitute 90 to 99 per cent. of all the white cells. They are mostly small lymphocytes (Pinkus), whereas the larger forms occur in the acute lymphatic leukæmia. (Ehrlich holds that large lymphocytes predominate.) Many of them are altered, degenerated or swollen. The polymorphonuclears are reduced from 70 to 10 or even 1 per cent. Myelocytes are usually absent. The red cells average 4,000,000, but seldom fall lower than 2,000,000. The lymphocytosis may temporarily disappear during infection and in one instance this was permanent. (See Plate XVI, Fig. 1.)

5. The *skin* may itch or be the seat of papular eruptions. Lymphomata form in the skin, sometimes of considerable size; infiltrated reddish deposits may precede the lymphæmic blood findings. Œdema occurs, while hemorrhages are rarer. Facial growths are observed similar to chloroma (*v.* Medullary Form, Diagnosis) or to the symmetrical swelling of the lachrymal and salivary glands.

6. The heart is degenerated, as are the vessels, from which hemorrhages may occur. Intraperitoneal hemorrhages from ruptured adrenals are reported. The nerve trunks may degenerate with bulbar, spinal and nerve trunk symptoms. Retinitis leukæmica or optic nerve involvement may impair vision. The liver is swollen from degeneration and from lymphatic infiltration. Dyspnœa is most common and occurs early from the anæmia, mechanical compression of the air tubes, vessels or nerves or from leukæmic changes within the follicles of the mouth and air passages. Renal infiltration is frequent.

Course.—The *course of the disease* is slow; it develops gradually and passes slowly into cachexia, though now and then extension in the glands is "explosive" (Trousseau). Remissions of weeks or months are not uncommon. Intercurrent disease may be fatal, as pneumonia and mechanical stasis, suffocation and acute exacerbations of the disease itself may hasten the course.

Diagnosis.—The diagnosis, suggested, perhaps by the skin lesions,

lymphomata, splenic enlargement or hemorrhagic tendency, is positively made only by the blood examination.

III. Myeloid Leukæmia.—Myeloid leukæmia (*myelæmia, myelogenic or medullary leukæmia*) is the most common form of leukæmia and occurs for the most part between the twentieth and fiftieth years; 67 per cent. of cases occur in males. Bacteriology has shown no cause. The disease is frequent among the Polish Jews and the poorer and working classes. A history may be obtained of malaria, syphilis, typhoid, influenza, stomatitis, tonsillitis, digestive disturbance, pregnancy, disturbed menstruation, the climacteric and trauma, though they are only accidental occurrences. The disease has been noted in three generations. Leukæmia has been also observed in animals.

Symptoms.—1. Blood.—The specific gravity is decreased from 1,055 to 1,045–36; it is watery or sometimes chocolate-colored; it coagulates slowly. In extreme cases the coagula are white (leukæmia) or slightly greenish. Large white clots are observed at autopsy and Virchow thought he had incised an abscess when he opened the left ventricle in his first autopsy. Microscopically the white cells are enormously increased, even as much as 1 to 1, or in Robin's case, 2 whites to 1 red cell. The highest count the author has seen was in Mercy Hospital in 1908; the whites and reds numbered 1,200,000 each. Amœboid movement is greatly decreased. (Plate XVI.)

The *characteristics of the white cells* are: (*a*) The *large mononuclear neutrophiles* ("mark cells," myelocytes), are found early and amount to 30–50 per cent. of the leukocytes. They are not pathognomonic, having been found (in small numbers) in pneumonia, uræmia, chlorosis, skin diseases, syphilis and in normal blood. (*b*) *Mononuclear eosinophiles* occur in other diseases, but in myeloid leukæmia are absolutely, though not necessarily relatively, increased. They are absolutely necessary to the diagnosis. (*c*) The "*mast cells*" must be absolutely increased; they are polymorphonuclears with basophile granulations which do not show with the tri-acid stain. Often they are relatively increased from 0.28, the normal percentage, to 1 or indeed 18 per cent. They are constant in this type. (*d*) The *polymorphonuclear* neutrophiles and eosinophiles constitute 30–60 per cent. of the leukocytes. (*e*) The *lymphocytes* are increased. (*f*) Atypical leukocytes, dwarf forms and white cells containing mitoses, fat globules or red cells may also be found. (See Plate XVI, Fig. I.)

The *red cells* are decreased to three or two million, though leukæmic patients are not always anæmic and an excess of red cells has been described. Toward the end the red cells may decrease to even 300,000 and thus resemble pernicious anæmia. The reds may become degenerated, necrotic, polychromatic or acquire amœboid movement, nuclei and mitoses. Nucleation occurs more frequently than in any other anæmia (Ehrlich), normoblasts being found regularly. The color index is 1 or less than 1. (See Plate XV, Fig. I, and Plate XVI, Fig. VII.)

2. Splenic Enlargement.—This is usually the first symptom

to attract attention; it appears with the blood findings and is most constant; spleniculi, if present, are enlarged. Instead of a weight of 5 to 7 ounces, 18 to 28 pounds are reported; from the normal measurements, 3 to 5 inches, dimensions of 15 inches are frequently observed. The form of the organ is preserved. The notches are usually clearly palpable and the edges are plump, rounded and hard. It may reach to the navel or into the pelvis, displacing other organs. The size varies during intermissions; without cause; from administration of arsenic and quinine and from diarrhœa or hemorrhages. It is enlarged after meals. A systolic murmur or pulsation is sometimes detected. Crackling under the fingers and tenderness indicate soft adhesions (perisplenitis). Firm adhesions may develop and the capsule may thicken. Though at first soft, it later becomes very firm, the pulp and trabeculæ hypertrophy and new lymphoid deposits are clearly marked against the reddish pulp (myeloid metaplasia). The author has seen three cases of leukæmic floating or wandering spleen, two of which had been operated on as uterine tumors. Rupture is a rare complication.

3. Glands.—The glands are enlarged in 33 per cent. According to the most recent views, the glands are heteroplastic, due to newformed myeloid tissue, "metastases" from the bone-marrow. Soft at first, they later become hard. Caseation and suppuration are most infrequent. As in the other types of leukæmia, recurrent compression of the vagus, trachea, portal vein and bronchi may result. Retrosternal dulness has been observed.

4. Bones.—Bone changes, recognized first by Neumann, occur in the spongy and long bones (ribs, tibiæ, sternum and vertebræ). The bone is mostly rarefied and there may be tender or yielding areas. The histological changes in the marrow are (*a*) the *lymphoid* (Neumann), in which the marrow resembles currant jelly, is reddish and consists of small lymphocytes; it is sometimes hemorrhagic; the normal fatty appearance disappears. (*b*) The *pyoid,* which is seen mostly in the myelogenic type; the marrow is not transparent, but gray; its cells are rich in protoplasm with one or many nuclei. In both forms nucleated red cells abound, also eosinophile cells and large cells with neutrophile granules and large nuclei.

5. Vascular System.—The heart is moderately distended with white clots and, as Virchow described in his first autopsy, opening the heart was like incising an abscess. The muscle is pale and fatty and nodules of leukæmic infiltration in the myocardium, foci of hemorrhage and pericardial nodes and transudation are frequent. Cardiac dislocation from pressure by enlarged glands and spleen, anæmic bruits, venous pulsation, *bruit de diable,* palpitation, systolic murmurs from pressure by the glands on the vessels and centripetal venous pulse are observed. The radial pulse is rapid. Dyspnœa results from weak heart, vagus pressure, nodules in the lung, bronchitis, hydrothorax, pressure of the spleen on the diaphragm, tilting of the heart upward and compression by the glands.

6. Respiratory Tract.—Nodules are frequently seen in the larynx, trachea, thyroid and thymus, often with hemorrhage, sometimes resulting in tracheal, bronchial or laryngeal stenosis. In the lungs (Deiter) lymphoid nodules may necrose and cavities like those of caseous tuberculosis may appear (Böttcher). Small myeloid foci of leukocytes may resemble miliary tuberculosis (Virchow). Bronchitis with many eosinophile cells in the sputum, nodes along the pleural vessels and pleural hemorrhage, transudation and exudation have been observed.

7. Fever.—Fever occurs in the majority of cases. It is rarely high; temperature may be normal for months or it may resemble recurrent fever, typhoid, sepsis or malaria, though it follows no one type.

8. Digestive Canal.—Anorexia, stomatitis or pharyngitis with ulceration of the lymphoid deposits, dysphagia from glandular pressure on the œsophagus, eructations and vomiting are occasional symptoms. *Diarrhœa* is very common, leucin and tyrosin being found in the stools. An enteric type is described by Béhier and others, in which the bowel symptoms are conspicuous; the lymphadenoid structures in the ileum are prominent and Friedreich once confused this type with typhoid on account of the ulceration, enlargement of the spleen and hemorrhage from the bowel. The abdominal lymph glands may be enlarged alone. The *liver* is much enlarged and may weigh 13 to 25 pounds. Its surface is smooth, its consistence increased and its edges plump. Myeloid deposits occur in the liver and dislocate or deform the rows of liver cells. The portal lymph nodes may be enlarged. Icterus is infrequent and ascites is common from cachexia or leukæmic peritonitis. The fluid contains many white cells and the myelocytes predominate as in the blood.

9. Kidneys.—The kidneys are frequently involved; there may be "myeloid" infiltration and cloudy, fatty and amyloid alteration. The urine is decreased, pale and strongly acid. The *uric acid* may amount to 20 or 105 grains (Virchow, Laache). Normally uric acid is as 1:50–80 of urea, but in leukæmia it is 1:16. The uric acid results from increased formation of the acid. The xanthin bodies are increased. Albuminuria is usual only in the last stages. The amount of urea depends on the degree of cachexia. Priapism occurs from nervous lesions or thrombosis of the dorsal vein. Amenorrhœa is frequent in women.

10. Nervous Symptoms.—Nervous symptoms may develop, as delirium, neuralgia, headache, syncope, vertigo or mania or rarely bulbar palsy, facial paralysis, apoplexy or symptoms like those of brain tumor.

The *Eye*.—Lymphoid nodules may develop in the orbit, possibly with exophthalmos. The *retinitis leukæmica*, first described by Liebreich, is not inflammation, but leukæmic infiltration and occurs in 33 per cent.; the retina is pale, its arteries small and veins large; the papilla is "washed" and yellow spots with red borders appear; hemorrhages also occur (Plate VIII, Fig. 9).

The *Ear.*—Vertigo, deafness, tinnitus and an atactic gait may result from growths in the labyrinth; sudden deafness from hemorrhage is described by Steinbrügger, Pepper, Wagenhausen and Lanois; the author saw three such cases within one year.

11. Skin.—There may be acne, pigmentation, œdema, pruritus, lymphoderma perniciosa (diffuse or tumor-like infiltrations) or sweats.

12. Hemorrhages.—Mosler found them in 52 and Cabot in 80 per cent. Epistaxis is the most common and may be fatal. Stomach and bowel hemorrhages rank next and those from the respiratory and genito-urinary tracts, the skin, joints, muscles, subcutaneous tissues or brain are rarer. The writer has seen three massive hemorrhages under the skin of the thorax, one in Leube's clinic and two in his own practice. Death has followed the extraction of teeth in a leukæmic subject. The writer saw Kolisko autopsy a case in which a slow ascending paralysis followed hemorrhage between the spinal pia mater and arachnoid.

Complications.—Complications are nephritis, pneumonia, endocarditis, amyloid degeneration, diabetes and gangrene. Sussmann could collect but 25 cases of complicating tuberculosis; this number was increased by Dock to 27; the author saw a case with E. W. Andrews, in which tuberculous pleurisy developed; tubercle bacilli and mononuclear white cells were recovered from the blood-stained fluid. In another case miliary tuberculosis developed.

Diagnosis.—The diagnosis was formerly a matter of great difficulty, because the difference between leukæmia and leukocytosis was made one of degree only and various limits were placed, beyond which leukocytosis was said to cease and leukæmia begin. The mere number of white cells is no criterion, because a ratio of 1 to 20 may be leukocytosis (as in Strauss' case of gastric cancer), and on the other hand, 1 to 200 may be leukæmia (von Noorden). Leukocytosis from digestion, fasting, marasmus, pregnancy, acute infections or in the death agony is of the ordinary polymorphonuclear neutrophilic type. (Plate XVI, Fig. IV.) Leukocytosis is a symptom, but leukæmia is a disease. The *polymorphism* of the blood is striking; according to Lazarus, there must be (*a*) mononuclear leukocytes with granulations (myelocytes); (*b*) increased mono- and polynuclear eosinophiles; (*c*) absolute increase in the "mast cells," and (*d*) nucleated red cells must be readily found. The gross splenic, medullary or lymphatic lesions are of but presumptive value in diagnosis and the polymorphic blood picture alone differentiates from pseudoleukæmia, typhoid, purpura, anæmia, sepsis, etc.

Caution should be exercised (*a*) during remissions, when the blood is rarely morphologically normal, no matter what the number of leukocytes. In one of the author's cases the leukocyte count dropped from 1,250,000 to 9,000 in a week, though the myelocytes still constituted 33 per cent. of the white cells. (*b*) Care is also necessary during acute infections when the leukæmic characteristics may disappear, as during typhoid, acute miliary tuberculosis, influenza or sepsis. (*c*) The blood findings may become atypical before death.

Similar Conditions.—1. Chloroma.—Chloroma is considered a malignant type of leukæmia or a "neoplastic hyperplasia of the red bone marrow," by Dock and Warthin; 46 cases are on record. It is closely related to acute lymphæmia. Lymphomata form, causing orbital pain, tinnitus, deafness, temporal and orbital swellings and such ocular symptoms as exophthalmos, strabismus and disturbed vision. The lymphomata are greenish, whence the name chloroma. It usually develops before the twentieth year. Retinal infiltration and hemorrhages occur. Other findings are ataxia, pallor, sternal tenderness, hemorrhages, severe anæmia, often but not always changing to leukæmic characteristics, greenish urine and greenish lymphomatous infiltration of the bone marrow, periosteum (sometimes with Charcot-Leyden crystals), the liver or lungs and greenish lymphadenoid changes. The hæmoglobin and red cells are reduced, even to 15 per cent. and 500,000 respectively. The leukocytes number about 50,000, but have been as numerous as half a million; they are chiefly lymphocytes. The affection is always fatal, usually within six months.

2. Leukanæmia.—Leukanæmia is described by Leube as a severe blood disease in which the myelogenous formation of both the red and white cells is disordered, *i. e.*, it combines some features of both leukæmia and pernicious anæmia. Its features are (*a*) a progressive anæmia, waxy color and asthenia; (*b*) numbers of normoblasts, but especially megaloblasts; (*c*) no abnormal pigments in the urine, no enlargement of the lymph glands (though since reported by Mattirolo) and no siderosis of the tissues; (*d*) no true leukæmic blood findings, but considerable myelocytosis and lymphocytosis; (*e*) firm connective tissue in the bone marrow and (*f*) enlargement of the spleen and prevertebral hæmolymph glands.

3. Secondary Cancer in Bone.—There are 13 cases in the literature in which cancerous bone metastases have produced blood findings rather like those of pernicious anæmia and increase of the leukocytes (of which the myelocytes constituted 4 to 17 per cent.).

Prognosis.—The prognosis is bad. It may last 5 to 8 or even 10 years after a diagnosis has been made, but a little over 1 year is the average *course*. The *beginning is insidious* and enlargement of the abdomen, dyspnœa, anæmia and enlarged glands are the cause for seeking medical treatment. The nutrition is usually good. *Sudden death* may occur, either without discernible cause or from hemorrhage of the brain, hæmatemesis, marasmus, anasarca, diarrhœa, pleuritis, peritonitis, deep coma or pneumonia. *Remissions* occur, but the blood findings persist. Ewing observed 3 *acute cases* and Billings and Capps published 2 and collated 7 others from the literature (which resembled an infection with hemorrhages, moderate splenic and lymphatic hyperplasia, severe anæmia, often necrosis of the throat and jaw, with 16,000 to 540,000 leukocytes and often with low fever). A permanent cure after erysipelas has been reported by Richter.

Treatment.—*Hygienic* measures, rest and a careful diet are of some

benefit. Fats and carbohydrates cause dyspepsia, whence Litten gives animal foods, eggs, milk, raw chopped meat, koumyss, peptones, oysters, beer, wine, zwieback, spinach, etc.; an ice-bag to the spleen may mitigate pain over it. *Splenectomy* is most irrational, for leukæmia is a primary disease of the bone marrow; 94 per cent. of Bissel-Hagen's group of 35 died. The great danger is hemorrhage. Great *caution* must be observed in using *purges,* which, as in Addison's disease, may cause sudden collapse and death. Inhalations of oxygen, blood infusions, spleno- or organotherapy and iron are inferior to *arsenic,* which decreases the number of white blood cells and the size of the spleen. Hemorrhages may cease and the number of red cells increase. Ebert gives arsenic with quinine. It is injected locally by Mosler into the glands and spleen, but in the author's experience without success. Phosphorus may soften the glands. Special care should be employed in tapping an ascites, for peritonitis is prone to develop. The *x-rays* may prove decidedly beneficial. In 1 case already alluded to the patient lived 19 months after the day he seemed to be dying. The way it helps is still a mystery. The *x-rays* produce a leukolytic substance (Capps and Smith). Wendell thinks 90 per cent. are benefited. In some cases the glandular and splenic enlargements and the blood findings have wholly disappeared, as in 1 case observed by the writer.

PSEUDOLEUKÆMIA.

Definition.—A disease resulting in chronic anæmia or cachexia, of which the chief symptoms or signs are enlargement of the spleen or lymph structures. The definition of the disease as "leukæmic appearance without leukæmic blood findings" no longer holds. Such conceptions would include glandular tuberculosis, Kundrat's lymphosarcomatosis, etc. It is also called Hodgkin's (1832) disease, pseudoleukæmia (Cohnheim), adenia (Trousseau), malignant lymphoma (Billroth) and anæmia (cachexia) lienalis or lymphatica (Wilkes).

Etiology.—The etiology is not clear. The disease occurs after local lymphatic disturbances in the neck, following otorrhœa, coryza or tonsillitis. Like chloroma and leukæmia, 75 per cent. of cases occur in males between twenty and thirty years of age. The disease is not uncommon in children and often occurs in the lower animals. It also sometimes runs through a family. Malaria, syphilis, scrofulosis, alcoholism, rhachitis, diarrhœa, typhoid, measles and scarlatina are often noted in the patient's history, but their relation, if any, is obscure. Suspected to be mycotic, many negative and variant examinations have been made. Spirochætes are recently reported. The disease has no well-established relation to leukæmia, as Cohnheim held, yet Fleischer, Penzoldt, Mosler and Senator have observed transitions from pseudoleukæmia to leukæmia. Neumann held that the leukæmic blood changes depended solely on the hyperplastic marrow changes and that as long as the hyperplasia involves only the spleen and glands

pseudoleukæmia results. Sternberg and others have maintained that it is tuberculosis, a theory disproven by Dorothy Reed and Simmons.

Symptoms.—Its types are (*a*) lymphatic; (*b*) lienal, pseudoleukæmia splenica, splenomegalia; (*c*) mixed and (*d*) myelogenic.

1. Lymph Glands.—Swelling of the lymph glands is generally the earliest symptom; the cervical glands first become enlarged, then the axillary glands after a variable period and later the subpectoral, inguinal and other groups. They are soft and elastic; at first the individual glands are round or oval, discrete and smooth; later, with increasing size, they fuse into harder, irregular masses as large as the fist or a child's head, particularly those in the neck. They may be painful from periadenitis, but never transcend the gland capsule and seldom caseate or suppurate. From the neck, where the jugular veins may be compressed, the glandular enlargement often passes to the intrathoracic glands, which are more often enlarged than the other internal nodes; this often occasions dyspnœa, compression of the superior cava (whence the development of a large collateral circulation on the chest-wall), dysphagia, bronchial compression and recurrent laryngeal or vagus paralysis. The veins of the arm may be compressed, causing œdema. In the abdomen the retroperitoneal, mesenteric, periportal, iliac and other groups may enlarge greatly; according to their location, abdominal pain, œdema of the legs, paraplegia, hydronephrosis, ascites and jaundice may ensue. *Histologically,* the lymphadenoid changes are (*a*) increase in the endothelial and reticular cells; (*b*) hyperplasia of the lymphoid cells; (*c*) formation of peculiar giant cells; (*d*) fibrosis and chronic inflammation and (*e*) great increase of the eosinophile cells (Dorothy Reed).

2. Spleen.—The spleen is enlarged in 75 per cent. of cases. It reaches dimensions rarely attained in leukæmia, but maintains its form, is hard and tender and presents the same histological appearance as in leukæmia, *viz.,* nodes of lymphoid cells in a fibrous reticulum. The greatest swelling of the spleen is usually observed when the lymphatic glands are large and hard, yet splenomegaly may be the sole symptom; splenic hypertrophy alone, with relative increase in the lymphocytes, may be classed as Hodgkin's disease, according to Pinkus.

3. Bone-marrow.—The bone-marrow is less frequently involved than in leukæmia. A myelogenous pseudoleukæmia has been recorded, but its exact status is still doubtful. The eosinophiles are abundant in the lymph nodes and bone-marrow.

4. The Blood.—The ratio between the red and white cells is normal. The hæmoglobin is decreased and the color index is usually 1; the hæmoglobin may be rapidly decreased as in Litten's case, from 85 to 40 per cent. within a week. There is usually moderate anæmia, especially in advanced cases. Poikilocytes and microcytes often occur, megalocytes rarely and normoblasts sometimes, but the eosinophiles are not increased. The blood plates are increased, but the Charcot-Leyden crystals are never found. Concerning the white

cells, there is a relative increase of lymphocytes, the chief criterion of the disease, according to Pinkus and Ehrlich. The polymorphonuclear neutrophiles are decreased. These changes may not be observed in the earliest stage of the disease. The total of white cells is not essentially increased; in the case of Askanazy the disease was pseudoleukæmia for two and a half years, when it became leukæmia and lasted another one and one-half years. Leukocytosis in some instances is a leukocytosis of the death agony or a terminal infection.

5. Other Organs.—(*a*) The urticarious eruptions and *lymphatic growths in the skin* occur rather more frequently than in leukæmia; the lymphoderma perniciosa of Kaposi may be considered pseudoleukæmic in cases with an absolute lymphocytosis. Erythema, purpura, furuncles, pemphigus, prurigo from irritation of the lymphatic deposits around the sweat glands and pigmentation have been observed. According to Dubreuilh (1905), 18 cases of prurigo are reported. Severe *sweats* are frequent. (*b*) Symptoms of *anæmia* occur, as a feeling of cold, vertigo, syncope, dyspnœa, œdema, hemorrhage, palpitation, anorexia, dyspepsia and severe diarrhœa. (*d*) *Local pain* over the *liver* and *spleen* may indicate perihepatitis or perisplenitis. Swelling of the liver may be marked and is due to lymphoid deposits. Severe muscular pain has suggested trichinosis. (*d*) Gangrenous stomatitis or pharyngitis may occur in degenerated lymph nodes in the mouth, tonsils and throat. Gastric and intestinal ulcerations and sometimes perforation are noted, though less frequently than in lymphosarcoma. Neusser noticed gastric crises like those of tabes. (*e*) Ross and Osler described lancinating pains in the feet and œdema from involvement of the spine and cava by the lymph nodes. (*f*) Amyloidosis, bronchial catarrh and pulmonary phthisis have been noted. (*g*) The uric acid in the urine is not increased. (*h*) The lymphatic hyperplasia may also involve the lymphatic structures in the tonsils, circumvallate glands of the tongue, the muscles, mammæ, eye-lids, thyroid or thymus; secondary deposits may be noted in the lungs, liver, heart, pleura and more rarely in the brain, genitalia and pelvis.

6. Fever.—Fever is common. Gowers and Murchison observed a chronic recurrent type, later described by Pel and Ebstein; the term is a misnomer and classification of this febrile variety as a separate form is incorrect, for it has been observed in chronic malaria, lymphosarcoma, cancer and glandular and splenic tuberculosis. The urine may show the diazo reaction with the fever. When the disease involves the internal glands only, the fever may simulate typhoid. Winnewarter stated that fever occurred when the glands decreased in size under the administration of arsenic. Fever is rather indicative of glandular tuberculosis or of intercurrent (terminal) infections.

Course.—The clinical course is long. The glandular swelling sometimes decreases with or without therapeutic measures. The extension from the cervical to other lymphatic glands is generally gradual, but may occur with great suddenness, as in Trousseau's case. An acute

form of Hodgkin's disease is not well established; many are sepsis, tuberculosis or acute sarcomatosis. *Death* may occur from intercurrent diseases, but it most frequently results from chronic cachexia. Obstruction of the air passages occurs more frequently than in leukæmia. Death may also occur from twisting of the pedicle of the spleen or from hemorrhages from the nose, uterus, stomach, intestines or larynx, which sometimes resemble purpura hemorrhagica of Werlhof. Very exceptionally transition into leukæmia is observed. It is a question whether patients with well-developed cases ever recover. Pleural effusions, diarrhœa, dysentery and vomiting from pressure of the splenic tumor are unfavorable complications.

Diagnosis, Allied Affections and Differentiation.—There are two criteria; (*a*) the lymphadenoid hyperplasia and chronic inflammation observed when an excised node is examined histologically; other kinds of cells or other degenerations excite suspicion that the disease is not pseudoleukæmia; and (*b*) according to Pinkus, an absolute lymphocytosis, with a practically normal number of white blood cells. Differentiation from similar affections is as follows:

1. Glandular Tuberculosis.—Hodgkin's disease may be complicated by or confused with, tuberculosis; in pseudoleukæmia the typical structure of the tubercle is lacking, but in tuberculous adenitis there may be an accumulation of large endothelioid cells between which lie tubercle bacilli (Ziegler); after a long course caseation and giant cell formation may result; the forms of glandular tuberculosis are (*a*) Ziegler's large cell infiltration, (*b*) purely hyperplastic lymphoid proliferation or (*c*) anatomical tubercles. Of late the number of reported instances of glandular tuberculosis simulating Hodgkin's disease has increased enormously. The author examined 2 cases of supposed pseudoleukæmia in which tubercle bacilli (without anatomical tubercles) were found in the spleen and lymph nodes. In the neck tuberculous adenitis is more often in the submaxillary than in anterior and posterior glands, in the latter of which pseudoleukæmia is more common. Tuberculosis is more frequently a local than a general adenitis and is more often attended by secondary suppuration. Türck believes that fever and glandular tenderness indicate tuberculosis and that node-like swellings of the lymph vessels in the mouth are symptomatic of pseudoleukæmia. Excision of a node determines the question.

2. Anæmia Infantum Pseudoleukæmica.—This disease was first fully described by von Jaksch, though earlier observed by Senator; it is known in Italy as *anæmia splenica infettiva dei bambini;* it is called "anæmia splenica" or "pseudopernicious anæmia of children" by Ehrlich. It is a profound anæmia in which the blood findings suggest pernicious anæmia; there are normoblasts, megaloblasts, decrease of the red blood cells (1½ to 3 million) and sometimes poikilocytes or polychromatophilia; there is often increase of the leukocytes, but rarely more than 50,000. It occurs in children from nine to eighteen months of age; the convalescence is very slow

or death may result. Its symptoms are hardness and enlargement of the spleen, swelling of the liver (less than in leukæmia and less than the swelling of the spleen), profound anæmia and hemorrhages into the skin or from the nose, mouth, stomach or intestines. The status of the disease is not yet determined, because the lymphocytes are frequently increased in children and leukocytosis in general has less significance than in adults. The diagnosis is especially difficult when it occurs in syphilitic children; syphilis, rickets and dyspepsia are common antecedents.

3. Anæmia Splenica.—It is doubtful whether it should be classed as an essential anæmia, liver cirrhosis, chronic splenitis or as a splenic form of Hodgkin's disease. (*a*) The *spleen is enlarged,* its capsule thick, its stroma increased and the endothelium of the lymph sinuses sometimes proliferated (endothelioma). The cause is unknown; possibly it is toxic. (*b*) There is an *anæmia* of the *secondary* or chlorotic type; the red cells average 3,500,000, the hæmoglobin is even more reduced and the whites are decreased to 3 or 4 thousand. (*c*) *Hemorrhages,* particularly from the stomach, occur in one-third to one-half the cases. (*d*) In many cases there is a terminal *liver cirrhosis* (Banti's disease), with icterus and ascites. (*e*) Its course is *very chronic,* covering years. In some cases there are profound alterations of nutrition, as stunted growth, clubbed fingers or pigmentation of the skin (in 8 of Osler's 18 cases). In several cases Dock and Warthin found portal thrombophlebitis and hyperplasia of the hæmolymph glands. Of Armstrong's cases 23 recovered and 9 died.

4. Lymphosarcomatosis.—This is sometimes difficult to differentiate and some, as Hoffmann, class the two diseases together. According to Kundrat, lymphosarcoma is largely a local disease, with local glandular involvement, especially of the cervical glands, leading to infiltration of the throat by contiguity; the disease may begin in the tonsils, air-passages, portal glands, etc. Lymphosarcoma ulcerates and transgresses the gland capsule; this never occurs in leukæmia or in pseudoleukæmia. The glands in leukæmia and pseudoleukæmia may compress neighboring structures but never actually invade them. Unless lymphosarcoma extends beyond the capsule of the glands it cannot be distinguished by physical examination unless it lies in parts which can be easily inspected, as the mouth. The spleen may enlarge enormously. According to Pinkus, the leukocytes are increased in pseudoleukæmia, while in lymphosarcoma they are decreased.

5. Other Splenic Enlargements.—The most common are: Cirrhosis of the liver, malarial spleen, sometimes carcinoma, chlorosis with enlarged spleen (Chvostek), chronic polycythæmic cyanosis and rarer forms, as miliary tuberculosis of the spleen, actinomycosis, syphilis, rhachitis or echinococcus. (See Diseases of Spleen.)

6. Multiple Myeloma.—This is characterized pathologically by lymphoid hyperplasia of the bone marrow, in the ribs and body-ends of the long bones chiefly and more often in the proximal than in the distal bones; no metastases occur. The bones soften or sometimes

sclerose. Clinically, it occurs in males over forty-five years and is characterized by severe, *deep-seated pain* in the body, chest, spine, neck and more rarely in the extremities. Prominences in the bones, *spontaneous fractures,* especially of the sternum and ribs, and kyphosis, from softening of the vertebræ, may result. The growths may pulsate or rupture externally. The *x-rays* may clearly define them. Severe anæmia without leucocytosis; neuralgia, motor and sensory paralyses, which develop late and involve the opticus, hypoglossus, facialis, cord (paraplegia dolorosa) and sciatic nerve; fever and the *Bence-Jones' albumosuria* are observed. To call this disease a myelogenic form of pseudoleukæmia or myelogadenia there must, according to Pinkus, be a lymphocytosis.

7. Other Affections.—Syphilitic lymphatic glands; symmetrical simple lymphoma of the lachrymal and salivary glands of Mikulicz, reacting to arsenic, and resembling pseudoleukæmia; and secondary carcinoma or sarcoma must be differentiated. *Difficulties* of diagnosis are met when the enlarged glands in internal parts produce topical symptoms, thus simulating hypertrophic cirrhosis, etc.

Treatment.—1. Local Applications.—These are useless, excepting the *x-rays;* W. A. Pusey states that two of his cases, seen by the author nine years ago, wholly recovered. In some cases the rays have no influence on the spleen and glands and in others the cachexia increases, though there may be improvement in the local findings. Inunctions of green soap, once or twice daily, are sometimes beneficial.

2. Drugs.—*Arsenic* is most efficacious (*v.* Pernicious Anæmia for details); it acts on the glands more than on the spleen. Some believe that the fever which it sometimes produces, reduces the adenopathy. Winnewarter and Billroth recommended its parenchymatous injection into the glands, but the results are not very encouraging. Iodides are seemingly injurious. Berberin hydrochlorate gr. xv increases the appetite and contracts the spleen (Litten). For hemorrhages Litten uses a decoction of quercus cortex, which disturbs digestion; formalin and rectified spirits āā locally (which may irritate a delicate skin) and guacamphol gr. iij to iv once daily may be given.

3. Surgical Treatment.—Extirpation of the glands is seldom beneficial. The removal of the spleen is attended by constantly decreasing mortality. In 1900 Bessel-Hagen collected 360 operations for various indications; the average mortality was 38 per cent. (for splenomegaly, 61 per cent. before 1891 and since then 13 per cent.). In Lichty's 25 cases 8 were operated on with 37 per cent. mortality. Scott added 6 cases, of which 33 per cent. died. Carstens, 1905, collected 739 cases of splenectomy; 72 per cent. recovered.

THE HEMORRHAGIC DISEASES.

The hemorrhagic diseases may be classified as follows:

(A). Acquired types. { 1. Purpura. 2. Scurvy.

(B). Congenital (hereditary) type. 3. Hæmophilia.

PURPURA.

Purpura includes a number of affections, more or less similar in kind and differing chiefly in degree, some of which are symptomatic, while others are independent, though obscure affections.

I. Symptomatic Purpura.—Symptomatic purpura is observed as a symptom in:—(*a*) Various *infections,* as endocarditis, scarlatina, measles, smallpox, rheumatism, sepsis, yellow fever, plague, syphilis or tuberculosis. (*b*) *Toxic* conditions, as nephritis, cholæmia, snake-bites and drug-poisoning (copaiba, ergot, belladonna, quinine, mercury, iodides, bromides or arsenic). (*c*) *Cachexiæ;* old age, malnutrition, carcinoma, sarcoma, leukæmia, pseudoleukæmia or pernicious anæmia. (*d*) *Nervous* states; cerebrospinal lesions, as tabes, myelitis; neuroses (bloody stigmata of hysteria); neuralgias or vasomotor fluxions. (*e*) *Mechanical* conditions, as trauma, embolism or stasis.

II. Purpura Simplex.—Purpura simplex (Zeller, 1684) is purpura in which cutaneous hemorrhages are the chief symptoms. Its *etiology* is obscure. It develops most often in middle-aged males. As in the other forms, whose description follows, there is no constant or characteristic *pathology,* though vessel thickening, hyaline and fatty degeneration, thrombosis, hemorrhage and pigmentation sometimes occur. In the blood itself no change is observed other than its tardy coagulation; this requires 10 to 30 minutes instead of 3 to 5, which is the normal time. The blood platelets are decreased (Pratt, Hayem). The *eruption* begins as small red spots, which gradually become green and black; they occur largely on the extensor surfaces of the limbs and more often on the legs than on the arms or trunk. They do not fade upon pressure. The initial eruption may be macular, papular or urticarious (purpura maculosa, papulosa or urticans), but soon becomes blood-tinged. Constitutional symptoms are usually absent; there may be slight fever, gastric disorder or diarrhœa (Graves). The usual *duration* is 1 to 2 weeks, the *prognosis* is good and the *treatment* hygienic rather than medicinal. Relapses are common.

III. Purpura (or Peliosis) Rheumatica.—Purpura rheumatica, described by Schönlein, is still listed by itself because of the articular symptoms. As joint symptoms also occur in Werlhof's disease (*v. i.*), scurvy and hæmophilia, its separate classification is somewhat arbitrary. Most cases develop in males between fifteen and thirty years of age. It has no relation to rheumatism, being a pseudorheumatism. Its pathology is obscure.

Symptoms.—(*a*) As *prodromes,* indisposition, low fever, muscular pains, colic and, particularly, sore throat may be noted. The developed disease is manifested chiefly by various forms of purpura and by joint symptoms; (*b*) the *purpura* may occur before, with or after the joint-symptoms. It occurs as simple hemorrhages of the skin, which vary from red to green-black, according to their age, or as urticaria, erythema, pemphigus or œdema, which become stained with blood; the legs are most often involved, largely on the extensor surfaces. (*c*) *Articular symptoms* are most common in the knees and ankles, being less frequent in the elbows and rare in other joints; there may be only stiffness and pain or actual swelling; if the joints are first involved their symptoms frequently abate with the appearance of the purpura. (*d*) *Other symptoms* are inconstant, as fever, which may rise to 102° or 103°, splenic tumor, albuminuria or nephritis. The author had 3 cases at one time in the Cook County Hospital, in which there was severe sloughing of the tonsils. Osler (1904) described 29 cases in which *visceral lesions* occurred with purpuric, urticarious and erythematous symptoms; they occurred chiefly in children and relapses were frequent; (i) digestive complications occurred in 25 cases; vomiting in 15; colic in 8 (resembling renal stone or appendicitis); blood in the stools in 8 and diarrhœa in 15; (ii) acute nephritis occurred 14 times and 5 cases died of uræmia; (iii) arthritis occurred in 17 cases; (iv) swelling of the fauces, œdema of the larynx, aphasia, transient hemiplegia and least often endocarditis may occur.

Diagnosis.—The diagnosis chiefly concerns affections characterized by hemorrhages of the skin, articular symptoms and perhaps temperature. Acute leukæmia, septicopyæmia and endocarditis must be excluded. Genuine rheumatic polyarthritis is differentiated with ease by the earlier migrating, inflammatory joint involvement, the temperature, sweating, characteristic cardiac complications and the later, rather accidental and wholly secondary occurrence of the purpura.

Prognosis.—The prognosis is generally good, though relapses are common and sometimes periodical. Its course is rarely longer than a week or two.

Treatment.—Treatment is symptomatic. The joint-symptoms yield somewhat to acetanilide, though the salicylates exert no specific influence.

IV. Purpura Hemorrhagica.—Purpura hemorrhagica (*Morbus maculosus Werlhofii,* Werlhof, 1775) closely resembles the types already described, but differs in that not only are the skin and joints the seat of hemorrhage, but also the mucous membranes, serous membranes and perhaps the viscera.

Etiology.—Of this little is known; staphylo-, strepto- and pneumococci and Bacillus pyocyaneus and coli have been found. It occurs oftener in young weakly women than in robust adults. Exposure to cold or dampness and malnutrition seem predisposing causes. It

usually develops as an apparently primary affection, but it may occur in some of the conditions listed under symptomatic purpura.

Symptoms.—Perhaps after inconstant and variable prodromes, as fever, vomiting or vertigo, (*a*) *hemorrhages* appear on the *extremities;* the face is not often involved; they occur as spontaneous petechiæ, blood-stained bullæ or vibices or may result from slight trauma, as by pinching the skin. (*b*) The *mucous membranes* bleed to a varying degree; the nose and mouth bleed most often; less frequently there is hæmoptysis, hæmatemesis, enterorrhagia, hæmaturia or metrorrhagia in various combinations, sequences and degrees. The conjunctivæ may be blood-stained. The writer saw one rapidly fatal case in which there was hemorrhage from the middle ear. (*c*) The *serous membranes* and the joints, particularly the knees, ankles and elbows, are least often implicated. Ecchymoses in the endo- and pericardium, pleura, peritoneum and meninges are common findings at autopsy. (*d*) *Other hemorrhages* are much less common, as retinal hemorrhages, hemorrhage into the brain, cord or testis. The connective tissues, fasciæ and bones are practically exempt.

General symptoms and *complications.* As to the blood, there is delayed coagulation and some anæmia, but the red cells and hæmoglobin are rarely much reduced; some nucleated red cells may be found, as well as leukocytosis, reduction in the blood plates and some methæmoglobinæmia. Fever is inconstant and atypical. The pulse rate may be raised; palpitation, delirium, vertigo, albuminuria, hepatic enlargement, splenic tumor, subicterus and nephritis are occasional symptoms. In J. S. Cutler's case there was extensive intestinal ulceration; perforation also has been reported.

Diagnosis.—Most errors in diagnosis result from confusion with purpura cases symptomatic of sepsis, hemorrhagic exanthemata or blood affections. The difficulties in diagnosis are often great; in an anæmic old man, who suddenly vomited blood and passed bloody urine, the diagnosis of purpura was made; the autopsy revealed a round ulcer of the stomach and a papilloma of the bladder. *Scurvy* is distinguished by its gingivitis and muscular hemorrhages, and *hæmophilia* by reason of its hereditary facts. *Henoch's purpura* occurs chiefly in children and is characterized (*a*) by cutaneous lesions, which resemble erythema nodosum rather than purpura; (*b*) by hemorrhages from the mucous membranes; (*c*) by gastro-intestinal crises of vomiting, diarrhœa and abdominal pain; (*d*) by pains in the joints and (*e*) by frequent relapses, often extending over a number of years (*v. s.* Osler's group). Of *chronic purpura,* Bensaude and Rivet collected 34 cases (1905).

Prognosis.—The prognosis is uncertain. According to Gross, 50 per cent. die. Unfavorable symptoms are high temperature, nephritis and cerebral hemorrhages. *Purpura fulminans,* of which Stybr (1906) collected only 14 cases, may be fatal in 24 to 48 hours; it was originally described by Guelliot, 1884. Recurrence is not uncommon.

Treatment.—This has advanced but little beyond Werlhof's therapy

of dilute sulphuric acid ♏x and quinine gr. v after meals. Everything of a supportive nature, as sufficient nourishment, rest in bed, iron and arsenic, is helpful. The hemorrhages are arrested with difficulty and the author doubts the value of drugs in their control. Turpentine and ergot may be given (as in hæmoptysis, *q. v.*). Adrenalin solution to the nose and mouth, gelatin by mouth (see NEXT SECTION), and especially calcium lactate gr. xv, t. i. d., to increase the coagulability of the blood, are indicated.

HEMORRHAGIC DISEASES OF THE NEW-BORN.

Syphilis Hemorrhagica Neonatorum.—Syphilis hemorrhagica neonatorum appears soon after birth. In some instances the syphilitic child may be born with hemorrhages in the skin, mucosæ, internal organs and from the navel. Occasionally deep jaundice develops. The cause is probably vascular disease; Mrâcek found endarteritis, especially of the capillaries, in 14 per cent. In other cases an added sepsis is apparently causative.

Winckel's Disease.—Winckel's disease, epidemic hæmoglobinuria, is (*a*) an affection of the new-born of unknown causation; (*b*) it appears epidemically in hospitals; (*c*) it develops in the first four days of life; (*d*) it is characterized clinically by fever, gastro-intestinal symptoms, icterus, dyspnœa, cyanosis, albuminuria, methæmoglobinuria and large mortality, and (*e*) pathologically by internal hemorrhages, fatty degeneration and splenic tumor.

Morbus Maculosus Neonatorum.—Morbus maculosus neonatorum is no clearer etiologically than Werlhof's disease in adults; some cases are congenital syphilis; others with fever, jaundice and epidemic occurrence are infections (pyogenic cocci, pneumococci, a bacillus like Friedländer's, pyocyaneus and colon bacilli) and still others seem embolic, thrombi forming in the auricles when the heart is weak (Landau). Injuries to the head, causing hemorrhages in the fourth ventricle and cerebral peduncles, and other vasomotor disturbances (von Preuschen and Pomarski) are questionable causes. It occurs once in 500 to 700 births.

Symptoms.—In the first week of life hemorrhages occur from the navel and mucous membranes; in Townsend's 50 cases they occurred in the following frequency: from the bowel (melæna), 40 per cent.; navel, 36 per cent.; mouth, 28 per cent.; stomach, 28 per cent., and nose, 24 per cent. Death occurs within a week in 63 per cent. Dusser, in 78 cases, found the mortality was 55 per cent.

Treatment.—Iron, ergot and adrenalin are useless. Diffuse stimulants should be given when there is a weak, rapid pulse. Injections of gelatin are dangerous, for it possibly contains some ptomaine; its local use and administration by mouth are safer (Abt).

SCURVY.

Definition.—A general disease, more often endemic than sporadic, caused by neglect of hygienic conditions and characterized by gingivitis, hemorrhages, inflammations, cachexia and chronicity of course.

Etiology.—It was first observed in the Crusades and was described most fully by Vasco de Gama; in his voyage around the Cape in 1498, 33 per cent. of his sailors were stricken with scurvy. It has developed among prisoners, Canadian lumbermen, Pennsylvania miners and among workers on the Chicago Drainage Canal, where in 1893–95 the author saw 100 cases. Insufficient, monotonous, possibly spoiled food causes the majority of cases, sometimes in conjunction with lack of fresh meat, impure water, damp quarters and over-exertion; a lack of fresh meat, vegetables (potassium salts, malic and citric acids) and fats predispose to scorbutus, though no single group is solely responsible. The Drainage Canal cases were all Poles and lived on coffee, bread and preserved and fresh meats. It develops principally in middle-aged males of the poorer classes. The disease is becoming less frequent. In 1780 Admiral Gleary had 2,400 cases in the English fleet. In 1849 there were 260,444 cases in Russia and 60,598 deaths; it is still endemic there and is regarded as an infection.

Symptoms.—Scorbutus begins slowly and progresses insidiously; its earliest symptom is (1) *anæmia* (cachexia scorbutica). The skin is scaly, pale, lustreless and may become pigmented. The mucous membranes are livid and the expression is apathetic. The blood findings are not characteristic; coagulation is slow, the red cells and hæmoglobin are proportionately reduced and leukocytosis is absent, except with intercurrent inflammation or severe hemorrhage. Emaciation, mental depression, muscular weakness, anorexia, reduction of HCl in the gastric juice, intestinal fermentation, constipation, palpitation, weakness with irregularity of the heart's action, a systolic functional murmur and œdema about the ankles accompany the anæmia. Splenic enlargement, albuminuria, peptonuria and oliguria are common.

2. *Gingivitis* begins with turgescence of the gums, which is due to a serohemorrhagic infiltration, usually noticed first about the canine teeth; the gums are spongy, painful, particularly on eating, bleed readily and may become so swollen as to hide the teeth. Gingivitis is absent when there are no teeth or roots. The breath is extremely fœtid. In severe cases the gums necrose and bleed profusely; deep ulcers, on healing, leave cicatrices; the teeth may loosen and fall out, the salivary and cervical lymph glands and tongue may swell.

3. *Hemorrhages* may occur (*a*) in the *skin,* especially on the extensor surfaces of the legs; small hemorrhages often occur, first about the hair follicles; they are at first small petechiæ, but later become larger and stretch the skin. Less frequently the first eruption may resemble acne, herpes or pemphigus, hemorrhages appearing later in

these exanthems. The trunk is less frequently involved and the face is usually exempt. As the hemorrhages increase, the legs are diffusely mottled with red, green and black and are swollen and tense. Similar stains may be noted running upward in streaks along the lymph vessels. Some of these changes are due to (*b*) muscular hemorrhages (*scorbutic sclerosis*). They occur mostly in the calf and thigh, less in the buttocks or abdominal wall. The legs are large, extremely hard, blood-stained and later pigmented. As the disease regresses the muscles usually recover their function, but in some instances ulceration (scorbutic ulcers), suppuration, muscular cicatrization or shortening of the tendons may leave permanent deformity and contractures. (*c*) *Mucous membrane hemorrhages* are less common than in purpura or hæmophilia, though epistaxis is fairly frequent. In some cases hemorrhages occur in the conjunctiva, air passages and stomach, but in more cases in the bowels, perhaps with intestinal ulceration. (*d*) Of serous membrane hemorrhages, articular hemorrhage (hæmarthrosis) is the most frequent. Next in order of frequency are those in the pleura, pericardium, peritoneum and meninges. They are usually sudden in onset, extreme in degree and unfavorable in outcome. (*e*) Parenchymatous hemorrhages are least common, as in the eye-tissues, brain and other viscera. Subperiosteal and epiphyseal hemorrhages are rare, unless Barlow's disease is included (see NEXT SECTION). Scorbutic ulcers in soft tissues may reach the bone, causing necrosis.

4. Scorbutics are subject to *various inflammations.* (*a*) Painful *joint* swellings are the most frequent and occurred in 35 per cent. of the author's cases and chiefly in the knees; they may be serous or hemorrhagic or in severe cases the cartilages or bones may become eroded and ulcerated; sometimes suppuration intervenes; deformity and ankylosis are possible sequences. (*b*) Hemorrhagic pericarditis and pleuritis are very dangerous. (*c*) Onychia, paronychia, choroiditis and panophthalmitis are occasional incidents. Fever is uncommon, except from extreme inanition or terminal infection.

Course and Prognosis.—The onset is insidious, the course chronic and convalescence protracted. Most cases recover and recurrence is not frequent. Death may occur (*a*) from cachexia, and the autopsy then shows fatty degeneration of the heart, liver and bloodvessels; (*b*) from profuse hemorrhages, into the pleura, pericardium, gastrointestinal tract or rarely into the brain or meninges; (*c*) from infections, as dysentery, pleurisy, pneumonia or septicopyæmia, probably of the terminal type, promoted by the reduced physiological resistance.

Diagnosis.—The diagnosis is made without difficulty by (*a*) the anæmia or cachexia, (*b*) gingivitis, (*c*) tendency to hemorrhage and (*d*) inflammation, (*e*) the chronic course, and (*f*) the temporary nature of the condition (as contrasted with the permanent tendency to hemorrhage in hæmophilia).

Treatment.—(*a*) *Hygienic;* avoidance of "cured" or smoked foods is both prophylactic and curative. In Nansen's polar expedition,

which lasted three years, not one case of scurvy developed. His meats were cooked and plenty of cooked and raw fruits and vegetables were used. Berries, sauerkraut, potatoes, radishes, cress, apples, oranges and lemons are especially valuable. A monotonous dietary is particularly to be avoided. (*b*) The *anæmia* (*q. v.*) should be treated by strychnine, quinine, iron and arsenic. Yeast, ʒij three times daily, is recommended. (*c*) The *gingivitis* is treated by local applications of silver nitrate (10 per cent.). Cocaine may be painted on the gums before meals so that the patient may eat, but great care must be taken lest the habit be contracted or toxic symptoms develop. (*d*) *Symptomatic treatment;* hemorrhage is benefited by rest only. Stimulation is required in intercurrent infections. Drastic cathartics are to be avoided. Complicating arthritis necessitates rest and the application of cold, but rarely aspiration or drainage.

INFANTILE SCURVY—BARLOW'S DISEASE.

Möller (1852–62) described the affection as acute rhachitis, and Ingerslev (1871), Jalland (1873), Cheadle and Gee (1878), but particularly Barlow (1883), described a cachexia with hemorrhages, which occurred in the very young. In 1898, 379 cases were collected in the United States.

Etiology.—Unlike scurvy in adults, the hygienic conditions are good in 80 per cent. of cases, and it occurs largely in the better classes. (*a*) The obvious *cause is feeding* with malted, condensed or cooked milk or baby foods; these foods lack some unknown substance essential to health. In 713 cases only 2.5 per cent. occurred in infants at the breast (S. Weiss, 1905). (*b*) Ninety per cent. of cases occur between the sixth and twelfth months (Heubner and Neumann).

Symptoms.—These are not always typical; there may be anæmia without hemorrhages or hemorrhages without anæmia, though in typical cases they occur together.

1. *Subperiosteal hemorrhage* is the leading symptom. The primary vague symmetrical swelling over the epiphyses later becomes more obvious, though no œdema is noted. There is pain and tenderness usually in the thighs or legs, which are drawn up, flexed or everted, immobile and semiparalytic (pseudoparalysis). At autopsy the anatomical findings are subperiosteal hemorrhages; a colloid and fibrillar alteration in the marrow of the diaphyses; arrest of ossification; decrease of the osteoblasts; rarefaction of the bones, sometimes leading to fractures (spontaneously or after slight trauma) or to epiphyseolysis (usually at either end of the femur or the upper end of the tibia); the joints are normal. Seventy per cent. of the lesions are in the thigh, 24 per cent. in the leg and 6 per cent. in the orbit; the arms, ribs, spine, skull, sternum and scapulæ are seldom involved. Rib involvement results in depression of the sternum.

2. The *cachexia* is evidenced by the anæmia (usually moderate and sometimes attended by slight lymphocytosis), sallow skin, asthenia,

anorexia, constipation, irregular temperature (in 66 per cent.) or subnormal registration and the almost invariable severe sweats.

3. *Gingivitis* is noticed when the teeth have already erupted or are about to erupt; the gums are normal in but 15 per cent. (Heubner and Neumann).

4. Other symptoms embrace hemorrhages in the skin in 50 per cent., from the bowels (10 per cent.), nose (9 per cent.), kidneys (9 per cent.) and rarely in the conjunctivæ, muscles or meninges. The spleen is rarely swollen.

Diagnosis.—Diagnosis is not difficult, after one typical case has been seen; it is based upon the etiology, subperiosteal hemorrhages, gingivitis and anæmia. Confusion with genuine paralysis is unnecessary, for the immobility is voluntary to escape the pain of movement. Syphilitic bone disease (syphilitic pseudoparalysis or Parrot's disease) is rather similar as to the crepitus, epiphyseolysis and pain, but other syphilitic stigmata or scorbutic symptoms are present.

Prognosis and Treatment.—Proper treatment secures marked and immediate improvement in the great majority of cases. Death is uncommon; in unfavorable cases it occurs in the third or fourth month of the disease. The diet consists of raw cow's milk, potato purée and beef juice (not broth or extracts); orange juice, apple sauce, pears, cooked berries and spinach are most beneficial. Treatment by drugs is generally superfluous.

HÆMOPHILIA.

Definition.—A congenital and hereditary affection, characterized by deficient coagulability of the blood and a consequent tendency to profuse hemorrhages, which are spontaneous or traumatic and most difficult to arrest. It was named by Schönlein.

Etiology.—Its real cause is unknown and the few pathological changes described in the blood and the bloodvessels are insufficient factors. Sahli believes that there is an hereditary deficiency in the fibrin-forming substances (thrombokinase). (*a*) *Heredity* is the only clear etiological fact, which Fordyce noted in 1784; as Grandidier (1877) remarked, hæmophilia is "the most hereditary of all hereditary diseases." Osler quotes Brown as tracing hæmophilia through seven generations in 200 years. Sometimes a generation may be exempt. (*b*) Broadly speaking, the *males* of the "bleeder families" are the "bleeders," and the females—the "conductors"—transmit the disease to their male offspring without manifesting the disease themselves. In 200 families of "bleeders," Grandidier found 609 cases in males and 48 in females (8 per cent.); Stahel in 1,880 families found only 24 cases in males. In the Mampel family, first reported by von Chelius in 1827, Lossen (1905) found 212 members of which 111 were males; of these exactly one-third were bleeders and 48.7 per cent. of the male bleeders died of hemorrhage; none of the females were bleeders and all transmission of the disease occurred

through the women. (*c*) The disease occurs most frequently in Germany, England, North America and France. Consanguinity and family tendencies (gout, rheumatism or tuberculosis) have been suggested as causes. "Bleeders" are generally healthy individuals with soft, blond, delicate skins and blue eyes.

Symptoms.—In 75 per cent. of cases, *hemorrhages develop* in the *first two years of life;* rarely do they first appear after puberty, in the third decade or after the seventieth year; only 46 congenital cases are known (Pratt). The hemorrhages are spontaneous or traumatic; they most often occur as capillary oozing from a mucous surface; deep interstitial bleeding is most unusual. They are copious and hard to still. Epistaxis is the most common (50 per cent.); hemorrhages from the gums (12 per cent.), intestine (12 per cent.), lungs, kidneys and stomach (each 6 per cent.) and uterus are next in frequency. Arthropathies are the rule. According to König, the joint findings are (*a*) an hæmarthros, with pain, swelling and fluctuation; (*b*) then panarthritis, whose gross pathology resembles tuberculous arthritis, with flexion of the joint; (*c*) regression, or ankylosis and deformity, which rather resembles arthritis deformans. The knees and elbows are most often affected. Subcutaneous hemorrhages may occur. The first bleeding may occur from the navel or may result from vaccination, extraction of teeth, circumcision, from a trifling prick or cut, from delivery or from rupture of the hymen on the wedding night. In various attacks, one part after another may bleed or the hæmophilic disposition may be confined to one part, as a kidney (renal hæmophilia) or nose; in Agnew's celebrated case, the patient bled from cuts on the head but never from lesions lower. Hæmophilia differs from purpura, in that the bleeding seldom occurs into a serous membrane (excepting the joints) or into the viscera. The amount of blood lost may be enormous and in Litten's case amounted to 24 pounds in eleven days. The coagulation time of the blood is delayed to thirty or sixty minutes (Wright); the clot, when formed, is porous, but retracts normally; the coagulum does not retract in purpura and the blood plates are reduced.

Of *general symptoms* there are few except changes in the joints (like those in subacute rheumatism), a neuropathic tendency (neuroses and neuralgia, particularly of the fifth nerve) and anæmia (though hæmophilics tolerate loss of blood better than other individuals, and recover from it more readily).

Diagnosis.—The diagnosis may be for a time uncertain, when hemorrhage develops after an operation or occurs from one organ only, as the kidney. The family history clearly stamps the affection and readily differentiates it from scurvy, purpura and hemorrhages in the new-born. It is peculiar that patients often deny that they are bleeders. The 16 cases of multiple hereditary telangiectases with recurring hemorrhages, described by Osler, may be hæmophilic.

Prognosis.—The prognosis is grave. Any hemorrhage may prove fatal. Sixty per cent. of cases die before the eighth year and only 11

per cent. reach the twenty-second year; if middle life is reached the danger gradually lessens. It is more favorable in girls, despite the apparent menstrual dangers. De Bovis (1905) found 69 excessive hemorrhages among 150 deliveries in hæmophilic women; he believes pregnancy has a rather favorable effect on the disease. De Lee has reported premature detachment of the placenta in hæmophilia. The rudimentary or local forms are more favorable than general hæmophilia.

Treatment.—1. Prophylaxis.—Grandidier lays down the following rules: (*a*) Women, with an hæmophilic family tendency, whether they are "bleeders" or not, should not marry. (*b*) Men, if not "bleeders" themselves, may marry. (*c*) Men should not marry when their family history indicates that male "bleeders" have begotten hæmophilic children. It is obvious that in hæmophilic families, general measures to elevate the physiological resistance and avoidance of trauma or any but life-saving operations, are indicated. Vaccination is rarely contra-indicated.

2. Control of Actual Hemorrhage.—This is almost impossible. (*a*) Wounds should be cauterized or enlarged, as Fordyce discovered that in hæmophilics large bleed less than small wounds. Other surgical hæmostatic measures need not be further considered. The Esmarch constrictor controls some hemorrhages of the extremities. (*b*) Styptics, as Monsell's solution, or tannic preparations are useless. (*c*) Adrenalin, locally and internally, has no apparent effect. (*d*) Calcium lactate is more beneficial; grs. xv t. i. d. should be given intermittently, because protracted administration decreases the coagulability of the blood. (*e*) Gelatin may be given by mouth or in unusually severe hemorrhages by subcutaneous infusion (*v.* page 832). (*f*) Saline cathartics are sometimes efficacious, possibly by diverting blood from the seat of hemorrhage. (*g*) Very acute anæmia necessitates injection of salines under the skin; when the hemorrhage is over, regeneration of the blood is remarkably rapid. (*h*) Joint involvement is treated by rest, immobilization and rarely aspiration.

SECTION VII.

DISEASES OF THE DUCTLESS GLANDS.

DISEASES OF THE SUPRARENAL GLANDS.

ADDISON'S DISEASE; OTHER AFFECTIONS OF THE SUPRARENAL GLANDS.

Definition.—A disease characterized (*a*) pathologically by lesions (usually tuberculous) in the adrenal glands or by degenerative changes in the adjacent abdominal sympathetic ganglia, and (*b*) clinically by muscular and mental adynamia, digestive disorders, pigmentation of the skin and a fatal issue.

Etiology.—1. Suprarenal Disease.—Suprarenal disease was first associated pathologically with melanoderma by Addison in 1855. By far the most common lesion is (*a*) chronic fibrocaseous *tuberculosis* of the adrenal glands; the tubercles of the adrenal glands are usually bilateral, destroy most of the suprarenal structure and frequently involve the semilunar ganglia and solar plexus by the attendant formation of connective tissue; in rare cases only is the adrenal tuberculosis primary (Symes and Fisher); there is a primary focus in the lungs in over 50 per cent., in the genito-urinary, alimentary or glandular system. (*b*) Less common are tumors, simple atrophy, fibrous induration (usually tuberculous), cirrhosis (26 cases recorded), gumma, amyloid degeneration, acute inflammation (as by contiguity from perinephric abscess), acute hemorrhage, embolism, echinococcus, adenoma or other very rare lesions. In what way suprarenal lesions produce Addison's *melasma suprarenale* is still a disputed question, for our knowledge of adrenal physiology is incomplete. It seems (i) that the glands contain a substance (sphygmogenin) which maintains vascular tonus and stimulates the vasomotor centres, the vagus and respiration; when secreted in excess, as in hypernephroma, the blood tension may be much elevated (Fränkel, Neusser, Kolisko) and this may induce cerebral hemorrhage; (ii) the glands apparently elaborate an internal secretion necessary to life which renders less toxic substances produced by other tissues, *e. g.*, the "fatigue stuffs" of muscle metabolism. Chemically, sphygmogenin, pyrokatechin, neurin and lecithin have been found. Able holds that the active glandular substance is epinephrin.

There are, however, cases of Addison's disease in which the adrenals are normal. Lewin's statistics from 500 cases are as follows: In typical cases the adrenals are diseased in 88 per cent. and apparently

sound in 12 per cent.; on the other hand, disease of the adrenals is attended by pigmentation of the skin in 72 per cent. and unattended by it in 28 per cent. There is no wholly satisfactory explanation for such cases. Perhaps compensatory hypertrophy or accessory glands may prevent symptoms in some cases of adrenal disease.

2. The Adjacent Sympathetic Structures—the solar plexus and semilunar ganglia,—may be involved directly, or indirectly as by fibrous changes in the adrenals and pressure from contiguous aneurysm or lymphomata. Pigmentary atrophy, hyaline degeneration and round cell infiltration of their vessels and degeneration extending upward to the cord or downward along the nerves have been described. Some writers explain Addison's disease as a disease of the sympathetic system; others endeavor to reconcile conflicting facts by assuming a decreased adrenal activity plus sympathetic disturbance.

3. Minor Etiological Factors.—(*a*) *Age;* most cases occur between the fifteenth and fortieth years; the youngest case recorded was congenital and the oldest was eighty years; (*b*) *sex;* 66 per cent. occur in males; and (*c*) 90 per cent. in the laboring classes.

Symptoms.—1. Adynamia.—Marked *physical and mental adynamia* is usually the initial symptom and persists, with increase after possible remissions, from onset to death. The insidious languor, weakness, apathy, lack of initiative and muscular relaxation contrast sharply with the healthy appearance of the patient in the early stages. These symptoms are probably toxæmic, partly from reduced internal secretion and partly from the tuberculous lesion. Obstinate early insomnia and headache and somewhat later attacks of vertigo, tinnitus, spots before the eyes and syncope are frequent. The intellectual functions are weakened, neuralgias occur in the epigastrium, intercostal spaces, back and extremities, periodic attacks of irritability are frequent and the deep reflexes are usually somewhat reduced.

2. Digestive Symptoms.—Digestive symptoms generally appear next. The tongue is coated and moist; there are anorexia, thirst, a sense of abdominal fulness, eructations and sensitiveness over the abdomen. *Paroxysmal pains* in the epigastrium or back radiate widely, especially into the chest; they resemble tabetic crises and are most severe. *Vomiting* is the most distressing, ominous and intractable alimentary symptom; it may follow eating or occur independently of it. At first intermittent, it later hastens the fatal issue. Early constipation gives way to terminal diarrhœa.

3. Pigmentation.—Pigmentation (melanoderma, bronzed skin, *melasma suprarenale*) develops after the adynamia and alimentary disturbance. Its cause is obscure; Neusser refers it to involvement of the sympathetic nerves or ganglia. It is found in the deep layers of the rete Malpighii, to which it is apparently brought from the blood by leukocytes. At first the *skin* has a dirty, yellow-gray color and small areas appear which gradually fuse and then slowly become darker and more diffuse; extended pigmentation may occur with focal areas of deeper discoloration. Pigmentation develops particularly (*a*)

where it is normally deepest, as about the nipples, anus and genitalia; (*b*) where pressure or friction is greatest, as over folds of the skin, over the clavicles, spine, ischia, scapulæ or waist (from belts or corsets); (*c*) where there is exposure to the sun, as in the face, neck or arms; (*d*) where accidental irritation occurs, as by blistering, eczema or parasitic affections. The soles of the feet, palms of the hands, nail-beds and hair are seldom stained. Previous scars may or may not be stained; sometimes they are surrounded by a zone of deeper discoloration. The skin is dry, is sometimes bathed with profuse perspiration and may have a fish-like odor. Scattered spots of pigment may atrophy (vitiligo) and impart to it a strange appearance. The *mucous membrane* of the lips, gums, cheek and palate show pigmentation later than the skin. The spots and horizontal ink-like stains on the cheek where the teeth touch it are particularly of diagnostic value. The conjunctivæ are pearly white and are usually said to be exempt, though frequently small aggregations of pigment can be seen on careful examination (Leva). Staining of the larynx, nymphæ, vagina, glans penis, serous membranes and internal organs is infrequent.

4. Other Findings: (*a*) The *heart* shares in the general adynamia; it frequently shows brown atrophy at autopsy; its tones and apex are weak. Pain over the heart is frequently reflected from the abdomen; palpitation and dyspnœa are common, but functional murmurs are rare. The pulse is compressible and somewhat accelerated, seldom slow or arhythmic. The peripheral vessels and abdominal aorta pulsate actively. Atheroma is common, even in young subjects (Kolisko). (*b*) *Anæmia* was considered characteristic by Addison, though Nothnagel and others have demonstrated its infrequency; in a recent case the writer observed a red-cell count of 2 million. Increase in the red cells (polycythæmia rubra) and in the hæmoglobin results from concentration of the blood, due to vomiting and diarrhœa. The lymphocytes are somewhat increased. (*c*) The *lymph nodes* and Peyer's patches are sometimes hyperplastic. The spleen, thyroid, thymus and pineal glands are often enlarged, probably vicariously. (*d*) *Emaciation* and *cachexia* are terminal conditions. Œdema of the ankles is infrequent even toward the end. Characteristic metabolic changes are not found; pigment in the urine is rare, acetonuria is occasional, the urea is often decreased and terminal albuminuria may develop. (*e*) The temperature is most often normal or subnormal and a subjective chilliness is experienced. (*f*) The genital functions decline as in any cachexia. (*g*) Tuberculosis of the lung or bowel may somewhat modify the symptoms.

Clinical Course.—The cardinal symptoms described progress slowly, with occasional intermissions, and in nearly all cases death results after a year or two; the terminal symptoms are often violent; there may be delirium, hallucinations, convulsions, incoërcible vomiting and diarrhœa, irregular respiration, small pupils, occasionally pseudomeningitic symptoms and finally collapse and coma. The course may

vary: (*a*) *acute cases* may resemble typhoid, digestive disease or acute poisoning; (*b*) *chronic cases* sometimes last ten or thirteen years; (*c*) *sudden death* may occur before pigmentation develops, the suprarenal lesion at autopsy disclosing Addison's disease; (*d*) the course may be modified by *pulmonary* or intestinal *tuberculosis.*

Diagnosis.—Diagnosis is based on (*a*) the prostration, psychical and physical; (*b*) digestive disorders, vomiting, diarrhœa and neuralgia; (*c*) pigmentation of the skin and oral mucosa; (*d*) cardiac weakness and (*e*) the chronic lethal course.

As Addison's disease usually indicates the presence of tuberculosis, the advisability of injecting tuberculin may be considered; a reaction may easily occur from tuberculosis in other tissues. Adrenal tumors may cause metastases in the brain or (like tumors of the thyroid, prostate and mammæ) in the long bones, skull and spine.

Differentiation.—*Pigmentation* occurs in numerous other conditions: (*a*) poisoning by silver (*argyria*), in which the reduced metal forms a gray deposit in the extracellular tissue of the skin, sweat-glands, conjunctivæ and nail-beds; by *arsenic,* which seldom stains the mucosæ; in *pellagra,* which is endemic and intermittent in occurrence, with exacerbations in the spring and fall, is preceded by erythema, is rarely associated with tuberculosis, but with eosinophilia, diarrhœa and voracious appetite and is exempt from mucous membrane pigmentation (Neusser). (*b*) Chronic *icterus* (which, however, stains the scleræ and urine), *cyanosis* (arteriosclerosis and heart disease, also sometimes pigmenting the oral mucosa), various physiological pigmentations and dirt and parasites, as in tramps (vagabondism). (*c*) *Abdominal diseases;* dyspepsia, gastric ulcer, tumor (cancer, lymphomata and Hodgkin's disease), hypertrophic cirrhosis and hæmochromatosis (hepatic and pancreatic cirrhosis, diabetes and skin pigmentation, *diabéte bronzé*). (*d*) *Pelvic conditions;* pregnancy, uterine and ovarian tumors (after the removal of which pigmentation may regress). (*e*) Phthisis, malaria, melanosarcoma of the skin, exophthalmic goitre or rarely scleroderma. In these conditions, pigmentation of the oral mucosa is rare.

Treatment.—1. Supporting and Tonic.—Absolute rest is imperative because of exhaustion and proneness to lethal syncope. Alcoholic stimulation is poorly tolerated. Careful feeding is necessary because of the sensitiveness of the stomach and bowels. Strychnine, arsenic and nitroglycerine may be given.

2. Symptomatic.—Narcotics for pain, gastric sedatives for vomiting (*v.* Gastritis), astringents and opiates for diarrhœa (*v.* Enteritis) should be administered; strong cathartics are always contraindicated, for they may precipitate fatal collapse; one case in the Cook County Hospital died suddenly after taking a dram of compound jalap powder.

3. Organotherapy.—Adrenal extract by mouth has surely helped some cases and apparently arrested the development of others (Oliver, Sansom, Lloyd, Jones, Osler, Ringer, Langlois, Dupaigne).

In Adam's review of 97 cases, one-third were benefited and one-sixth recovered. The medullary portion of the gland is best. It may increase the vascular and muscular vigor or even lessen pigmentation. Some practitioners advocate the hypodermatic administration of adrenalin chloride, 3j of 1 to 1,000 in salt solution on alternate days. Some advanced cases are unfavorably affected by organotherapy.

Other affections of the suprarenal glands are of slight clinical interest. (*a*) In *developmental anomalies* the glands are sometimes absent or hypoplastic, chiefly with other defects, as encephalocele, etc. (*b*) *Hypertrophy* in one gland may compensate for hypoplasia in the other. *Supernumerary* glands are not uncommon. (*c*) *Atrophy* is usual in advanced age. (*d*) *Degenerations* embrace the parenchymatous, fatty, hyaline and amyloid types. (*e*) *Circulatory disorders* are hyperæmia, embolism, thrombosis and hemorrhage, which latter may occur in trauma in the new-born, tumors, inflammation and the hemorrhagic diathesis. (*f*) *Inflammation,* acute and chronic, is uncommon. (*g*) *Granulomata,* chiefly syphilitic and tuberculous, are rarely primary. (*h*) *Tumors,* carcinoma, sarcoma (20 cases recorded, Affleck and Leith) and hypernephroma (*v.* page 792) are chiefly of anatomical importance. In 23 published operations the mortality was 50 per cent., recurrence developed in 21 per cent. and 29 per cent. recovered (in nine months to six years). At the Johns Hopkins Hospital three tumors of the suprarenal glands were successfully operated on (Osler). (*i*) Parasitic disease (echinococcus) is extremely infrequent.

DISEASES OF THE SPLEEN.

Lesions of the spleen are usually secondary.

ACUTE SPLENIC TUMOR.

This occurs (*a*) in *acute infections,* due to the local action of microörganisms and their toxins or to toxæmic paralysis of the splanchnic nerve, which normally controls the vascular supply of the organ; it is most common in typhoid, malaria, recurrent fever and septic conditions; (*b*) in *acute stasis,* usually from portal obstruction; and (*c*) in *embolism* (*v. i.*) and trauma. *Symptoms* are infrequent; much pain usually indicates involvement of the capsule (perisplenitis). On objective examination: (*a*) The spleen may sometimes be seen during deep inspiration. (*b*) Palpation alone is wholly reliable. By the bimanual method and with the examiner sitting at the right side of the patient the edge of the spleen comes in contact with the fingers and slips sharply down below them; splenic crenations are seldom palpable. A palpable spleen always indicates

enlargement or much less often, downward dislocation by pleurisy, emphysema, pneumothorax or thoracic tumor. (*c*) Percussion, valuable in outlining the upper splenic level, is unreliable in determining the lower border.

CHRONIC SPLENIC TUMOR.

Chronic enlargement is symptomatic of (*a*) *chronic infections,* as malaria, tuberculosis and syphilis, (*b*) *continued portal stasis,* (*c*) *repeated embolism,* (*d*) *blood affections,* as leukæmia, pseudoleukæmia or splenic anæmia, and (*e*) rickets, amyloidosis, neoplasms, cysts, polycythæmia (*v. i.*), arthritis deformans and chronic acetanilide poisoning.

EMBOLISM.

Embolism occurs frequently in the spleen, because of the wide arteries and slow current; it ranks second in frequency to kidney embolism. It usually develops in the course of acute endocarditis or chronic valvular lesions and seldom follows local lesions, as sinus thrombosis or splenic vein thrombosis. The *symptoms* vary somewhat as the embolus is simple or infective; sudden chill, fever, pain in the side, acute splenic tumor and perisplenitic friction may develop, which with a cause or embolic symptoms in other organs justify a diagnosis of embolism. Frequently no distinctive symptoms occur. If septic, symptoms of abscess may be present (*v. i.*). Treatment is symptomatic.

ABSCESS (SPLENITIS APOSTEMATOSA).

Abscess of the spleen may result from embolism, trauma or invasion by contiguity. It occurred in 9 per cent. of 430 cases of pyæmia (Paget). It is largest in non-embolic cases; in embolic the abscesses are small and multiple. Symptoms are: (*a*) those of the cause, which may mask the lesion; (*b*) of embolism; (*c*) of splenic tumor, perisplenitis, friction or pain; (*d*) of rupture into the pleura, kidney or peritoneum; (*e*) aspiration may determine the diagnosis and surgical evacuation is then indicated.

PERISPLENITIS.

This affection may be primary or secondary, simple or suppurative, an isolated lesion or part of a chronic peritonitis (see "Icing Liver," Chronic Perihepatitis). It usually accompanies the various splenic affections enumerated. Without local peritonitic friction, pain or tenderness its diagnosis is impossible. There is no therapy.

AMYLOID SPLEEN.

Its etiology is considered under amyloid liver and kidney. Pathologically its two types are (*a*) the sago spleen, in which there are amyloid deposits in the vessels of the Malpighian tufts, and (*b*) the more important, diffuse amyloid infiltration. It is diagnosticated by (i) the etiological factors, (ii) its usually large size, hardness, smoothness and thick edge, and (iii) coincident amyloidosis of the liver and kidneys.

SPLENIC ANÆMIA.

Springthorpe observed six cases in one family. Umber advises operation in every case, because he believes that the spleen destroys the red cells. The *x*-rays have been of therapeutic value. (See page 827.)

TUMORS AND CYSTS.

New growths are very infrequent. They are more often secondary than primary. They include carcinoma, sarcoma (of which 19 primary cases were reported by Adolph, 1905), enchondroma, fibroma and lymphangioma. They are rarely differentiated from splenic anæmia and like affections. The chronic endothelioma of Gaucher clinically resembles splenic anæmia; pathologically, large endothelial cells replace the splenic tissue; this was seen macroscopically in Stengel's case.

Echinococcus cysts are found in but 0.2 to 3 per cent. of echinococcus disease and usually with concomitant cysts in the liver. One dermoid cyst is reported (Andral, 1829). Only 38 non-parasitic cysts are found in the literature.

Granulomata (see TUBERCULOSIS AND SYPHILIS).

RUPTURE OF THE SPLEEN.

Rupture may be traumatic or spontaneous; the latter variety has been observed in embolism, malaria, typhoid, acute leukæmia and pregnancy. It is more common in India and tropical climates than in temperate zones. Aspiration and even palpation have occasioned rupture. The treatment is surgical; 104 splenectomies for rupture are reported, with 29 per cent. mortality (Hörtz).

MOVABLE OR FLOATING SPLEEN.

Permanent mobility (*lien mobile*) and dislocation are due to the same factors observed in enteroptosis, with which it may be associated: (*a*) lax, elongated or ruptured ligaments (ligamentum gastrolienale, phrenicolienale and most important, ligamentum phrenicocolicum); (*b*) relaxed abdominal walls; and (*c*) enlargement and increased weight of the spleen operating with the above factors.

Symptoms.—Symptoms are often absent. Pain or a dragging sensation is frequent, which is either local or radiates to the thorax, left shoulder or legs, similar to that observed in neurotics or women with uterine or ovarian disease. There may be headache, insomnia, psychical alteration, dyspepsia, constipation, menstrual anomalies, vesical tenesmus and paræsthesia in the legs; most of these are neurotic. *Objectively:* (*a*) The most important sign is *palpatory detection of the spleen in an abnormal location.* (i) It is located most often in the left ilio-inguinal region, near the ilium, but may lie just below the left costal arch, near the navel, in a hernial sac, in the pelvis or above the pubes. (ii) Its hilum usually points upward, either directly, if it lies across the vertebræ or in the pelvis, or with slight inclination to the right or left if it lies to one side. (iii) The form, notches and possibly the pulsating vessels are those of the spleen. (iv) In size it is usually enlarged, either from some causal hypertrophy or some later congestion; torsion of its pedicle may lead to softening, atrophy, gangrene or even liberation of the spleen as a free body. (v) As a rule it can be replaced and its position changes with posture; perisplenitis, causing a friction rub, may result in firm adhesions to the colon, bladder, rectum or uterus, thereby causing insuperable diagnostic difficulty; in such cases the spleen ceases to "wander." (*b*) The *spleen is absent from its normal location,* the splenic area being replaced by tympany from the colon (which changes to dulness when water is introduced into the colon). (*c*) *Pressure symptoms* include intestinal obstruction, gastric dilatation, vesical and rectal compression.

Diagnosis.—Diagnostic confusion with floating kidney and pedunculated ovarian tumors is infrequent when the abdomen and pelvis are carefully examined. The author knows of three laparotomies performed for supposed uterine or renal disease in which abundant adhesions rendered differentiation impossible. On the other hand, a hard, immovable suprapubic tumor was correctly diagnosticated as leukæmic spleen from the glands and blood findings.

Treatment.—(*a*) *Bandages* are more difficult to apply than for movable kidneys, though some cases can be benefited by them. (*b*) The spleen may be replaced by operation and its vicinity well packed with gauze; the resulting adhesions often retain the viscus. (*c*) *Splenopexis* (Rydygier) may be performed, the spleen being sewed in place or deposited in a peritoneal pocket. Of 43 cases operated upon in the last decade only 3 died (Bessel-Hagen). (*d*) *Splenectomy* (see PSEUDOLEUKÆMIA) may be performed as a last resort, the mortality being about 27 per cent.

SPLENOMEGALY, WITH CHRONIC POLYCYTHÆMIA AND CYANOSIS.

This condition is also known as erythemia and Vaquez's disease (1892). Anders (1907) collected 53 cases. (*a*) The *red cells are in-*

creased, even to 9 or 13 million (polycythæmia rubra or polyglobulism); the hæmoglobin may reach 200 per cent.; the white cells number from 4,000 to 31,000. The viscosity, specific gravity and volume of the blood are increased. (*b*) There is *chronic cyanosis,* without adequate cause and (*c*) *splenomegaly.* There may be headache, dyspnœa, enlarged liver, mental and physical weakness, albuminuria, constipation, vomiting and an hemorrhagic diathesis. The arterial tension is increased and the heart hypertrophies. De Lee sent the author what appeared to be a *forme fruste,* a case with splenomegaly and cyanosis but without polyglobuly. The *etiology* is obscure; in about one-third of the cases it was tuberculosis of the spleen and 8 of the 11 operated cases recovered; it seems probable that in most cases the cause is an undue activity of the bone-marrow, which at autopsy is livid purple and hyperplastic. The *treatment* is *x*-rays, venesection, nitrites, iodide of potash or splenectomy.

LYMPHATISM (STATUS LYMPHATICUS).

Lymphatism is a condition in which there is hyperplasia of the various lymphoid and kindred tissues and a tendency to sudden death. It is also known as the *constitutio lymphatica, status thymicus* and *status lymphaticus.* (See Thymus Gland.)

Its *etiology* is obscure. The affection is rare and occurs most often in women and children. Felix Plates (1614) observed it as a familial affection.

Symptoms.—1. There is *lymphoid hyperplasia.* (*a*) The *lymph glands* of the chest and abdomen are more often involved than the external, cervical, axillary or inguinal glands. (*b*) The *tonsils,* nasopharyngeal, lingual and intestinal lymphadenoid tissues are frequently hyperplastic. (*c*) Moderate *splenic enlargement* is usual. (*d*) The *thymus* is swollen, sometimes giving retrosternal dulness. (*e*) The *bone-marrow* often changes to a reddish color.

2. *Associated findings* may include hypoplasia of the heart and aorta, thyroid enlargement, poor development, infantilism, rickets, tetany and laryngismus.

3. *Sudden death* is often the first evidence of the condition and may occur without seemingly adequate cause. (*a*) It has occurred during or after *anæsthesia,* especially with chloroform. In one case a patient with enlarged glands died suddenly after receiving but 20 drops of chloroform. (*b*) Operations with *local anæsthesia* have also been fatal. (*c*) Administration of *antitoxin* has caused death, as in the well-known case in Professor Langerhans' family. (*d*) It may occur while *bathing* or after *falling in the water.* (*e*) Sudden deaths during convalescence from *acute infections* or some collapses during hydrotherapeutic treatment (Escherich) may be attributed to the lymphatic constitution. (*f*) Children with eczema may die suddenly; lymphatism causes 75 per cent. of these deaths. It is said to be due to cardiac excitability (Kundrat), to lessened physiological resistance

with a tendency to heart paralysis (Paltauf) and to a toxic condition (Blumer, who suggests the term lymphotoxism).

DISEASES OF THE THYROID GLAND.

GOITRE.

Definition.—Hypertrophy of the thyroid gland. Sporadic cases are common in Europe and America. Goitre occurs endemically in Switzerland and Italy, though we have no knowledge of its cause; the drinking-water has been thought causal. It is usually acquired and is seldom congenital.

There are *three main forms:* (*a*) The *parenchymatous* or follicular type, with more or less symmetrical enlargement and formation of colloid material in the hyperplastic follicles; (*b*) the *cystic;* and (*c*) *vascular* or congestive type, with dilatation of the bloodvessels, which form borders closely on those physiological variations observed at puberty, in defloration, pregnancy, chlorosis and psychical trauma.

Symptoms.—1. The *thyroid enlargement* may be diffuse, as in the soft, symmetrical and moderately vascular form, or partial or nodose, implicating but one lobe or the isthmus. In most cases there are only local findings.

2. *Compression* symptoms are not common. The carotid artery and jugular vein are pushed to one side and the sternomastoids become thin. The trachea may be compressed, its lumen becoming shaped like a sword scabbard; asthma, dyspnœa, bronchiectasis and dilatation of the right ventricle follow stenosis of the air-passages. Laryngeal paralysis occurs in over 10 per cent. Fränkel states that in 25 per cent. of cases in which there is tracheal stenosis, there is also laryngeal paralysis. From the isthmus or lower horns of the thyroid, goitres may grow in a retrosternal or retroclavicular direction (5 to 10 per cent.) causing compression of the vagus, sympathetic nerves and vena cava. A goitre may disappear into the chest with each inspiration and appear on expiration (*goitre en dedans*). It may become incarcerated in the chest.

3. An *aberrant or accessory thyroid* may occasionally form a tumor anywhere from the tongue to the aorta, from the spine to the anterior chest wall, in the retropharyngeal or retroœsophageal tissues (causing dysphagia or dysarthria), in the trachea, larynx, root of the tongue, pleura or mediastinum; it may compress an entire lung; 75 benign and 16 malignant growths are recorded in retrosternal accessory thyroids. Storrs (1904) collected 32 lingual goitres; there are three cases in the literature of retrosternal accessory thyroids, which became cystic and calcareous (Collins, 1905), and fourteen which were intratracheal (Pfeiffer, 1905).

4. *Sudden death* may occur in large goitres; it may be due to pressure on the vagus, spasm or œdema of the glottis, sudden hyperæmia of the gland and pressure on the trachea, hemorrhage dissecting into the chest or status lymphaticus.

Diagnosis.—Confusion is possible with *tumors,* as sarcoma, carcinoma or adenoma, for whose differentiation and symptoms reference to surgical treatises should be made. There are 25 cases in the literature of *echinococcus cysts.* In 83 per cent. of cases *cancer* develops from a preëxisting goitre; early metastases are specially common and characteristic; only 10 per cent. recovered among 110 operations (Madelung). There are 20 cases recorded in which *metastases* throughout the body occurred *from benign tumors* of the thyroid (chondroma or myxoma). Thyroid inflammation (*strumitis*) is usually metastatic or traumatic, rarely primary; it may occur in sound or goitrous glands; it is most often due to the streptococcus but may be caused by the colon, typhoid, pneumonia or other organisms.

Treatment.—In regions where goitre is prevalent the drinking water should be boiled. *Medical treatment* is of uncertain value. Iodine may be used externally or by injection (Maunoir, 1825). Potassium iodide (Coindet, 1820) is beneficial in some subacute cases, according to Bruns, in 75 per cent.; Kraus found it particularly beneficial in the vascular type, with cardiovascular symptoms, as moderate tachycardia and full arteries. The *x*-rays are valuable in some cases. Thyroid extract is occasionally beneficial. *Surgical treatment* is indicated in large or growing goitres. Reverdin's collection of 6,103 operations shows a mortality of 2.8 per cent. and in Kocher's series, now several thousand, one death occurred in each 600 operations. See Cachexia Thyreopriva, Myxœdema.

EXOPHTHALMIC GOITRE.

Definition.—A malady due to excessive activity of the thyroid gland and characterized by three cardinal symptoms, exophthalmos, enlarged thyroid and tachycardia and by other accessory symptoms, as tremor, increased metabolism and ocular, vascular, digestive and other symptoms. It was first described in England by Parry (1786) and Graves (1835) and later in Germany by Basedow (1840). The claim of the Italians that Flajani (1800) first described the affection is ill-founded.

Etiology.—(1) The most probable cause is *over-activity or perversion of thyroid secretion* (Möbius, Johnston and Greenfield). Exophthalmic goitre contrasts sharply with myxœdema (*v. i.*) of which the anatomical basis is thyroid atrophy, as shown below:

Exophthalmic Goitre.	*vs.*	Myxœdema.
(*a*) Hypertrophy of gland; excessive internal secretion (hyperthyrea or hyperthyroidism).		Atrophy; deficient (hypothyrea) or absent secretion (athyrea).

Exophthalmic Goitre.	*vs.*	Myxœdema.
(*b*) Increased nervous excitability.		Dulness, apathy, cretinism.
(*c*) Vascular erythrism; flushed, moist, warm skin; rapid pulse.		Skin dry, thick and cool, and pulse slow.
(*d*) Increased diuresis and active metabolism.		Decreased.
(*e*) Thyroid extract increases symptoms; it may produce in normal individuals symptoms like exophthalmic goitre, tachycardia, tremor, even exophthalmos.		Thyroid extract improves or cures.
(*f*) Surgical (partial) excision relieves, and often cures.		Some cases result from removal of thyroid. Transplantation of new gland improves or cures.

(*g*) Exophthalmic goitre (glandular hypertrophy with excessive function) may occasionally become myxœdema (insufficient function from glandular atrophy).

2. *Other etiological explanations* are less probable, as (*a*) vasomotor neurosis or (*b*) lesions in the corpus restiforme of the medulla.

3. *Disposing or exciting factors* are: (*a*) *Sex;* in Eschner's 200 cases 80 per cent. were women (95 per cent. in other series). Fibroids, pregnancy and puberty are sometimes promoting factors. (*b*) *Age;* most cases occur between the twentieth and thirtieth years. Barrett (1904) could collect only 42 cases in persons under fifteen years. (*c*) *Psychical* trauma, as fear, excitement or concussion, are apparent causes. (*d*) Anæmia, debility, excesses, neuropathic family tendencies, other neuroses or psychoses favor its development. (*e*) After thyroidectomy, symptoms resembling those of exophthalmic goitre may develop; this postoperative thyroidism is rare.

Symptoms.—1. Cardinal Symptoms.—(*a*) *Tachycardia* is the earliest and most constant cardinal symptom. The pulse ranges from 100 to 120 and in severe cases to 140 (or even 200); it varies greatly from time to time. It is associated with other cardiovascular symptoms, as (i) increased cardiac action, manifested by a diffuse cardiac impulse, sharp tones sometimes heard at a distance of several feet (Graves) and hypertrophy of the left ventricle eventuating in dilatation; (ii) a functional systolic murmur over the apex; (iii) palpitation, which usually antedates tachycardia and is an evidence of intoxication (thyroidism); (iv) vascular symptoms; a pulse rather weaker than the apex beat; active arterial pulsation in the carotids and abdominal aorta; systolic capillary pulsation in the fingers, forehead or even in the retina, palate, liver and spleen; even the peripheral veins may pulsate; (v) irregular and failing heart action occurs late and in severe cases. Hemorrhages, notably epistaxis, are not uncommon.

(*b*) *Exophthalmos* is usually a later symptom than tachycardia or sometimes than the goitre. It is present in about 90 per cent. of

cases and results from dilatation of the orbital vessels. In developed cases the protrusion of the eyeballs and the staring, fixed expression are very characteristic (exophthalmos may also occur in extreme myopia, tumors of the orbit and sympathetic irritation). The condition is generally bilateral, though often unequal; in one instance the author saw unilateral exophthalmos with homolateral goitre. The eyelids are unable to cover the eyeball entirely and a rim of the white sclera shows about the iris. The protrusion may be very extreme, even amounting to dislocation of the bulb; in these cases there may be corneal ulceration and loss of the eye by panophthalmitis. Under this topic may be mentioned the *associated eye-symptoms,* which are: (i) *von Graefe's sign,* the failure of the upper lid to follow the eye-ball when it is rolled downward, is present in at least 33 per cent. and is of considerable diagnostic value, though present also in Thomsen's disease. (ii) *Stellwag's sign,* which is rarely absent, is widening of the interpalpebral fissure (Dalrymple, 1849) with infrequent winking (Stellwag, 1869); it also occurs in mania and in old age. (iii) *Möbius' sign* consists of weakness of the internal recti, whereby convergence is less complete or unsustained when a finger on which the eyes are focused is brought close to the nose; it occurs also in other conditions, as paretic dementia. (iv) *Becker's sign* (1873) consists of pulsation of the retinal vessels. (v) In *Joffroy's sign* the patient's forehead remains smooth (instead of becoming wrinkled) when he glances upward with the head bent somewhat downward. (vi) *Gifford's sign* is the difficulty in everting the upper lid; (vii) Jellinek and Rosin describe an œdema and pigmentation of the upper lid. Boston noted a spasm of the lid in eliciting v. Graefe's sign. Tremor of and a sense of pressure behind the eyeball are common. Eye paralyses and retinal or pupillary changes are infrequent.

(*c*) *The goitre* is rarely an initial symptom and usually develops later and in a remittent fashion, *i. e.*, swelling, recession and renewed swelling. It is present in 100 per cent. (i) Its typical form is *vascular,* though it may develop upon any of the ordinary forms of goitre, as in an acute fatal case, seen in 1905, with Dr. Dorsey of Streator, in which exophthalmos, tachycardia and thyroid thrills and murmurs developed in a chronic cystic goitre. Acute thyroidism may develop in thyroid malignancy. The goitre is *smaller* than the ordinary bronchocele; it is diffuse or oftener more developed on the right side (an accentuation of physiological proportions), shows a vascular, rough surface and is dry, gray and granular on section (instead of the normal amber-red color); microscopically, according to MacCallum, the alveoli are not rounded, full of colloid matter or lined with cubical epithelium, but are extremely irregular in size and form, smaller or ramifying and encroached upon by epithelial proliferations and the epithelium becomes columnar; in extreme or severe cases there is some epithelial desquamation; the colloid matter is scanty and stains faintly and some lymphoid tissue

develops; in the later stages some fibrous tissue may develop. (ii) It is usually *soft, elastic, variable* in size from time to time, *pulsating* on inspection, *thrilling* on palpation and revealing on auscultation a loud systolic bruit or a double murmur, which, according to Guttmann, is pathognomonic. The goitre may be retrosternal.

2. Accessory Symptoms.—(*a*) *Tremor,* described first by Basedow, is classified as a cardinal symptom by Charcot and Marie. It is present to some degree in most cases and is conspicuous in 50 per cent. It is characteristically fine, regular and rapid, numbering 8 or 9 vibrations to the second. It affects the wrist more than the fingers; sometimes it affects the trunk. (*b*) *Nervous* symptoms. Some *cerebral symptoms* are usual, as headache, vertigo, impaired memory, insomnia, irritability, depression or change in disposition. Psychoses are infrequent, as the mania sometimes observed in fatal cases. Choreiform movements were described by Kahler. "Giving way of the legs" (Charcot) and weakness of the respiratory muscles (scant inspiratory increase in the chest measurement, Bryson's sign) are expressions of *weakness of the muscles,* whose strength is reduced to one-third or one-fifth of normal (Müller). The reflexes are often increased. Rheumatic pains may be noted. *Vasomotor symptoms* are common, as violent reddening of the face, subjective warmth, dry mouth, sweats of a peculiar, penetrating odor, or intermittent swelling of the joints. Rarer complications are cerebral paralyses, tabes, syringomyelia or various neuroses. (*c*) *Respiratory* symptoms include dyspnœa and dry cough. The author has seen two incipient cases diagnosticated as phthisis. (*d*) *Digestive* symptoms. Severe nervous vomiting and diarrhœa may simulate organic disease; their persistence is ominous. (*e*) *Cutaneous* symptoms. Pigmentation may be extreme (Drummond, Chvostek). Pigmentation of the eyelids, vasomotor œdema, urticaria (particularly in acute cases), itching and falling of the hair may also occur. A peculiar fulness above the clavicles may develop, sometimes with pain in the neck. The electrical resistance of the skin is decreased, perhaps to one-fifth of the normal (Charcot, Chvostek, Vigoroux); it is probably occasioned by profuse sweating and has no pathognomonic importance. (*f*) *Constitutional* symptoms. Emaciation and anæmia are common, especially in blondes. Metabolism is almost always increased; this is evidenced by the nitrogenous excretion, increased diuresis and the frequent, slight, but wholly atypical, elevation of temperature; these symptoms may be intermittent (Huchard). Gilman Thompson describes fever and symptoms resembling ulcerative endocarditis. Amenorrhœa, albuminuria, glycosuria, persistence of the thymus gland (in 80 per cent.) and the occasional hyperplasia of the spleen are difficult to explain. In the leucocyte count the polymorphonuclears are decreased and the lymphocytes increased (Kocher).

Diagnosis.—The diagnosis is unequivocal when the cardinal triad of symptoms is present, reinforced by the secondary symptoms. Atypical forms (*formes frustes*) may cause confusion, but careful

watching of the clinical course usually settles all doubt. The affection is often incorrectly diagnosticated in young girls at puberty, in whom thyroid turgescence, moderate tachycardia and vascular excitability occur without particular significance. Incipient tuberculosis and neurasthenia may be simulated.

Course and Prognosis.—(*a*) The onset is usually insidious and the course chronic. Remissions are very common. Chronic cases sometimes begin rather acutely. Acute cases are unusual; they occur chiefly in men; Lloyd records a case which was fatal in three days and Trousseau one fatal in eight days. The author has seen three cases in which death occurred in from two to five weeks. The acute type is usually fatal, though Solbrig mentions a recovery after ten days. (*b*) *Death* occurs in 12 per cent. of cases; it is almost always due to cardiac failure, hypertrophy passing into dilatation. (*c*) The outlook is best in the less typical (rudimentary) forms. (*d*) Complete recovery is rare, though 20 to 50 per cent. of cases improve (relative recovery). (*e*) Unfavorable prognostics are its occurrence in the very young and in men, emaciation, extreme tachycardia, persistent vomiting or diarrhœa, fever, corneal disease, lymphatism and intercurrence of myxœdema (*v. i.*).

Treatment.—1. General.—*Rest* is the first indication. Travel, sojourn at the various springs and resorts, gymnastics and excitement are always injurious. A simple diet and interdiction of alcohol, coffee, tea and tobacco are essential. A quiet life at a moderate elevation or at the sea-side is very beneficial.

2. Drugs.—Except for symptomatic indications, drugs have a limited use. The heart tonics are disappointing; digitalis fails to slow the heart and strophanthus and spartein sulphate are rather more efficient but do not adequately control tachycardia. Belladonna, in large physiological doses (until it produces mydriasis and dry mouth), is recommended by Gowers; in personal experience the annoying palpitation and abdominal throbbing are most relieved. Ergotin, quinine, arsenic, iron and strychnine are of uncertain value, but the latter seems rather more beneficial; iron is apparently useful only in chlorotic girls (Möbius) and is directly injurious in men, for it aggravates digestive and vasomotor disturbances. Constant application of an ice-bag to the heart is very helpful; it relieves palpitation and somewhat slows the heart rate.

3. Electricity.—This is chiefly valuable as a suggestive remedy; galvanization is particularly recommended by Erb, who uses the anode over the cervical spine and the kathode over various peripheral areas. Electrotherapy effects only transient results.

4. Organotherapy.—Thyroid extract aggravates the symptoms in nearly all cases. Thymus extract has apparently helped some cases, but recent reports concur with the rather negative results obtained by Mackenzie and Kinnicutt. The use of serum or milk from thyroidectomized animals is still in the experimental stage; Baumann (1895) demonstrated that the thyroid gland contained an

iodine body which Ross proved was the main functional constituent of the organ. The active element of the gland presents two clinical phases: (*a*) it neutralizes toxins (failing in which myxœdema results), and (*b*) *if not itself neutralized,* Basedow's disease results; to neutralize the hyperthyrea of this disease, it was suggested that the serum of thyroidectomized animals be used (Ballet, Enriquez, Lanz, Burghart and Blumenthal); some good results have recently been obtained (Rogers).

5. Thyroidectomy.—The earlier objections to partial thyroidectomy are now weakening because of convincing surgical statistics. The average mortality is about 5 per cent. In Kocher's large experience of 315 operations, naturally on severe types, only 3.5 per cent. died; not one case died in the last 91 operated upon; 83 per cent. were cured. Kocher considers (*a*) the blood pressure, size of the heart and tachycardia; (*b*) he hesitates to operate if there is great nervousness, intoxication or absence of lymphocytosis, and (*c*) if the gland is very large, pulsating or vascular. The Mayos' last series shows 8 per cent. mortality; they consider general preferable to local anæsthesia. Death may also occur under local anæsthesia, which latter is a very unpleasant procedure. Removal under nitrous monoxide gas gave excellent results in two of the author's cases. Kocher prefers trying the *x*-rays first and then proceeding by stages, first ligating the thyroid arteries (after which tetany may occur and once within half an hour) and later performing partial thyroidectomy; the danger of a "heart" death is avoided by relatively early operation. Hirst states that the mortality of operation, for any indication, in an exophthalmic subject, is increased 15 per cent. Division of the cervical sympathetic is inadvisable.

MYXŒDEMA.

Definition.—Myxœdema is a chronic constitutional disease, characterized (*a*) by loss of thyroid function (athyrea), (*b*) by myxœdema (an elastic œdematous change in the subcutaneous tissue), (*c*) by cachexia or (*d*) by cretinism (mental failure). It was described by Gull (1873) as a cretinoid manifestation and later by Ord (1877) in England and in France by Charcot, Bourneville, d'Olier and others.

The symptoms vary as to cause and combination, whence (i) cretinism, (ii) adult myxœdema and (iii) operative myxœdema will be considered separately.

Etiology.—The thyroid may be aplastic, hypoplastic, cirrhotic from an infective sclerosing thyroiditis (syphilis, tuberculosis, rheumatism or erysipelas) or hypertrophied (goitre) or it may be removed by operation, but in all cases the thyroid function (internal secretion or antitoxic action) is suspended or lost.

CRETINISM.

Cretinism may be congenital, in which instance life is not long protracted; it usually develops in the first year and is noticed near the time of weaning (infantile form); it may develop between the fourth year and puberty (juvenile form). The endemic form occurs in regions where goitre is prevalent, in Switzerland, Italy, France, Great Britain, Spain and Sweden; according to Kocher, when both parents are goitrous, the child is invariably a cretin, is myxœdematous and may also have goitre. The sporadic form may develop from thyroid aplasia, sclerosing thyroiditis or from goitre. In 1905, 100 cases were collected in America (Howard).

Symptoms.—In the last part of the first or in the second year of life, retardation of mental and physical development is apparent. The head is disproportionately large, the fontanelles persist, the forehead is narrow and the base of the skull is shortened. The face is turgid, pale and imbecile, the lids are swollen, as are the nose, the protruding tongue and pouting lips. Dentition is retarded and caries is frequent. The skin loses its tone and is dry, and the hair is thin. The neck is short, which condition is accentuated by supraclavicular deposits of fat. The abdomen is large and pendulous; umbilical hernia is very frequent. The body is dwarfed and the extremities are short, stumpy and weak or helpless. Metabolism is slow, the gaseous interchange being reduced to 60 to 50 per cent. of the normal.

Diagnosis.—(*a*) The mental alteration is marked, though mild compared with the grimaces, grinding of the teeth and compulsory movements caused by *gross lesions of the brain.* (*b*) In hyperplasia of the bone cartilages (*chondrodystrophia fœtalis*) there are great enlargement of the head and joints, dwarfing of the body and shortness of the extremities (micromelia), but no essential mental change; court fools were probably of this type. (*c*) *Infantilism* concerns only the infantile appearance of the genitalia and the absence of the primary and secondary evidences of sex; general malnutrition, aplasia and cretinism are absent, though some cases of infantilism are due to thyroid hypoplasia.

MYXŒDEMA OF ADULTS.

Symptoms.—Myxœdema spontaneum adultorum, or *cachexie pachydermique* (Charcot) is characterized by (1) thyroid atrophy, (2) myxœdema (pachydermia), (3) intellectual and physical weakness and (4) certain accessory symptoms. It is most frequent in England and France. Most cases occur between thirty and fifty years of age.

1. Thyroid Atrophy.—This is difficult to detect clinically. It is less a clinical than a histological condition, as evidenced by the lack of function sometimes observed in gross enlargement of the gland; Magnus Levy, in 34 cases observed by him, found the thyroid hard and large in 5 cases. Eighty-six per cent. occur in women and it is possibly related to the uterine functions or to the physiological con-

gestive, thyroid enlargement observed in women. It may be hereditary or familial. It sometimes occurs in a family (or in individuals) in which there are cases of exophthalmic goitre.

2. Myxœdema.—Mucin has been found in the skin and subcutaneous tissues. These structures are inelastic, semigelatinous and semiœdematous, but do not pit upon pressure; the skin is pale, waxy, dry, rough, firm and its electrical resistance is increased; the hair and teeth may fall out and the nails thicken and break. The loss of the outer half of the eyebrow is described by French writers. The facies is somnolent, coarse and swollen, and the features are individually changed, rather resembling those of a cretin; the cheeks are patchy-red, pendulous and tremble like jelly and the tongue is big. The supraclavicular tissue is enlarged in lumps and rolls, though this is also observed in healthy persons (Verneuil's pseudolipomata). S. Kuh observed a painful swelling over the clavicle alone, which rather resembled keloid. The hands and feet are infiltrated. The swellings may change rapidly. The oral mucous membrane is dry and thick, sometimes also the pharynx, larynx and even the rectum; polypi are frequent and secretion of tears is scanty.

3. Mental Weakness.—Intellectual and physical weakness is apparent. Somnolence is more frequent than insomnia. Charcot compares the condition to hibernation. Headache is common. The patient is apathetic, irritable, weak in memory and slow in thought, speech and movement; sometimes delirium and mania precede the ultimate dementia. The gait is clumsy ("hippopotamus gait") and muscular fatigue follows moderate exertion.

4. Other Symptoms.—The accessory symptoms are variable, as cardiac hypertrophy, arteriosclerosis, palpitation; small, sluggish pupils, tremor, vertigo, night terrors, sensitiveness to cold; indigestion and constipation; anæmia; amenorrhœa; scanty urine with little urea and low specific gravity, sometimes albuminuria and less often glycosuria; hemorrhages, especially from the uterus; and subnormal temperature. The thymus is usually enlarged, sometimes the hypophysis; Hunt describes it with adrenal tuberculosis.

Course.—The course is chronically progressive and covers ten to twenty years. Remissions may occur in the warm weather, from change to a milder climate or during pregnancy. In the last stage, the myxœdema often disappears. Transient cases of acute myxœdema have been observed by Sollier, Osler and others. Osler reported an acute case with melæna, mania, glycosuria, tachycardia and death in six months. Sudden death is occasional, but most patients die of intercurrent tuberculosis or acute infections.

CACHEXIA THYREOPRIVA OR OPERATIVE MYXŒDEMA.

Horsley reported that myxœdema, tetany contractures, apathy and coma occurred in the monkey after complete thyroidectomy; when kept warm the monkeys did not manifest myxœdema, but a species

of cretinism. Cachexia follows extirpation in man and herbivora; convulsions develop in carnivora, resulting from the fact that the antitoxic elements of the thyroid are not present to neutralize the nuclein of ingested meat. The toxæmia strumipriva is manifested by anæmia (2 million red cells), leukocytosis (30 to 38,000) and degeneration in the walls of the bloodvessels. Kocher observed cachexia strumipriva in 70 per cent. of cases, more often after the complete than after the partial operation. Reverdin made similar observations and von Eiselberg reported it in about 23 per cent. of complete excisions made in Billroth's clinic. Accessory thyroids (*v.* GOITRE) may avert this operative complication. The author knows of two instances in which removal of an accessory lingual thyroid (under a mistaken diagnosis) caused myxœdema. Schiff (1884) and later von Eiselsberg cured operative myxœdema by transplantation of a thyroid. Bircher (1899) first successfully made a transplantation in the human being. Unfortunately, the function of the transplanted gland is seldom maintained, though Cristiani reports excellent results.

In some cases ablation of the thyroid gland seemed to produce tetany (*tetania thyreopriva*). Gley was the first to connect tetany with extirpation of the *parathyroid glands,* first described by Sandström (1880). Vassali and Generali maintained that absence or ablation of the thyroid causes cachexia and trophic disorder, and that absence or ablation of the parathyroid glands causes tetany (without cachexia). In thyroidectomy the thyroid should be removed carefully in order to spare the parathyroids located alongside the lower part of the lateral lobes.

Treatment of Cretinism, Myxœdema and Cachexia Thyreopriva.—For these allied conditions we possess in the thyroid extract (Glandulæ thyroideæ siccæ) one of the few actual specifics of medicine and most cases of cretinism, adult myxœdema and operative myxœdema can be cured—in a sense we can seldom employ this term. Horsley's pupil, Murray, first used the thyroid extract hypodermatically; this method soon gave way to its administration by mouth, advocated by Horwitz of Copenhagen (1892), and Mackenzie and E. L. Fox in London. The powdered extract should be prepared from the glands of young sheep, for the thyroid soon atrophies in older animals. Beginning with gr. ij t. i. d., the dose may be increased to gr. v or even xv. In a short time the evidences of increased metabolism appear, as loss of weight, increased diuresis, increased urea and elevation of bodily temperature; normal growth is stimulated, myxœdema and cretinism disappear, the pulse becomes full and lively, the skin moistens and regains its lustre and in every regard the results are magical. In some instances toxic effects (thyroidism) are apparent, as tachycardia, nervousness, delirium, dyspnœa, palpitation, flushing, sweating, tremor, less often tonic spasms and rarely exophthalmos, softening of the bones, particularly in young rhachitic subjects, or even death; these symptoms are allied to those of exophthalmic goitre

(hyperthyrea); thyroidism is seemingly more frequent in myocarditic and arteriosclerotic subjects. Ewald thinks that administration of arsenic lessens the liability of thyroidism, which after all is an infrequent condition. Larger doses may be given in winter than in summer. At first large doses are indicated and later, after relief is obtained, smaller doses should be given, averaging gr. j. to ij, or whatever size experience in the individual case may determine. Relapses are common after withdrawal of the extract and in most cases it becomes rather a food than medication. Change of climate and warm baths are of decided benefit. A milk or a salt-free diet is also advocated.

DISEASES OF THE THYMUS GLAND.

The **thymic functions** are unknown. Among other hypotheses it is said that the thymus is concerned in neutralizing infection or in certain vegetative functions, as the growth of bone and that it bears some relation to cerebral function. Some class it as a ductless gland. The gland is epithelial in origin, but becomes lymphoid in structure. Its secretion in the first two years of life is milky and contains iodine, cells and a molecular substance. It weighs two to four drams at birth, about an ounce in the second year, after which it wastes, weighing but one dram at twenty-five to thirty years of age.

There is an intimate, though obscure, relation between the thymus, thyroid, pituitary body, parathyroids, testes, ovaries and possibly also the bone-marrow. The thymus wastes as the testicles and ovaries develop. Thymic extract causes a fall in blood pressure, cardiac acceleration and, in large doses, fatal collapse.

(*a*) *Hemorrhages* into the thymus may be found in cases of congenital syphilis and asphyxia of the new-born. (*b*) Thymic *cysts.* Chiari claims that 50 per cent. are merely accentuation of the normal findings, in which the lymphoid elements overgrow into the convolutions of the gland. (*c*) *Abscess* of the thymus, described by Dubois (1850), is often mistaken for softened gummata, cysts and post-mortem softening. (*d*) The most frequent *tumors* are sarcoma and lymphosarcoma. Less frequent are dermoids and granulomata.

(*e*) *Persistence of the gland after puberty* is described; the term is ill-chosen, as the gland normally persists into adult life.

(*f*) Some cases of "persistent thymus" are **hypertrophy** or **hyperplasia,** which is the most common thymic lesion. Microscopically, we find lymphoid hyperplasia, Hassal's concentric epithelial corpuscles, eosinophiles and even myelocytes. (i) *Physical signs.* Dulness of more than 1 cm. to each side of the sternum is pathological. The gland may weigh one-half to five ounces. (ii) Plater (1614) associated enlargement of the thymus with *sudden death.* Grawitz, Jacobi and others have described cases of thymic hyperplasia which has caused sudden dyspnœa, usually in infants, followed by early death; the idea is advanced that the gland presses on the vagus, cava or trachea. The cases are thought to be lymphatism (*q. v.*), as are the cases described

by Nordmann, Recklinghausen, Rolleston, Paltauf and Keyer, who collected eight cases of sudden death in robust young men while bathing, by Kundrat, who found thymic hyperplasia in 10 cases of death from chloroform and by Schlömicher's similar series. (iii) The existence of *thymic asthma* (Kopp, 1830), also known as Millar's asthma, was denied by Friedleben (1858). The stridor, caused by thymic compression of the trachea, is pronounced, progressive and usually fatal from asphyxia. The enlarged gland may actually flatten the trachea (Summa and Benecke), as the distance between the sternum and spine is but little more than an inch and compression in this region is critical. Rehn, in a case of thymic asthma, stitched the gland to the skin, which relieved all symptoms; König and Ehrhardt each cured a case by partial excision of the gland; 9 operations are recorded. Hochsinger (1905) found 20 cases of *thymic stridor* in 26 enlarged thymi in children; enlargement was determined by the *x*-rays. (iv) Hyperplasia has been frequently found in Graves' disease, status lymphaticus, myxœdema, syphilis, acromegaly, epilepsy, myasthenia gravis and in atrophy of the splenic, lymphatic or myeloid structures. Besides operation, *x*-rays, quiet, iodides and mercury are advised.

DISEASE OF THE HYPOPHYSIS: ACROMEGALY.

Acromegaly (large extremities), first described by Marie (1886), is a nutritional affection, probably caused by disease of the hypophysis, and marked clinically by unusual overgrowth of the bones, tissues of the face, hands and feet and by general symptoms, glycosuria and sexual disturbance.

Etiology.—The hypophysis is almost always diseased. In 1902, Woods-Hutchinson collected 262 cases; in 77 autopsies the gland was diseased in 73. The process may be hyperplasia, adenoma or soft malignant sarcoma, less commonly colloid degeneration, fibrosis, softening or hemorrhage. Dean Lewis found hyperplasia of the chromophile cells, which confirms Benda's theory that acromegaly is due to excessive activity of the cells in the anterior lobe of the hypophysis. From its association with acromegaly, the gland has been thought to be the "growth centre." The growth may compress the cerebral nerves, cavernous sinus, bone, dura or even the brain. According to Sternberg, the sexes are about equally affected, and in 50 per cent. the affection begins between twenty and thirty, in 75 per cent. between twenty and forty and in 14 per cent. under twenty years of age.

Symptoms.—1. Acromegaly.—Enlargement of the extremities is usually the first clear symptom, but is preceded by paræsthesiæ and pains in the extremities, often extreme and due to expansion. (*a*) A remarkable change occurs in the *face;* the superciliary ridges protrude, the maxillæ enlarge and the nose, ears, lips, tongue and chin attain massive proportions. The general contour of the face is hexagonal. The teeth separate and the tongue sometimes protrudes be-

yond them. The eyes by contrast appear small. Pathologically, the change in the bone is simple hypertrophy; there is increase in the vascular furrows, widening of some of the foramina (with narrowing of others) and some roughening of the bones and localized protuberances which are not osteophytes; the skull is thicker and larger, especially the sphenoid, superciliary ridges, mastoid and occipital protuberances; the orbit may be narrowed by ethmoid bulging and the sinuses and sella turcica are increased. (*b*) *The hands and feet* are greatly enlarged, not deformed but symmetrically *hypertrophied.* The fingers and toes show two types (Marie), the long and the thick types; they are square at their ends—the "spade-like fingers." The palms and soles become flattened. The *x*-rays show simple hypertrophy of the periosteal and subperiosteal tissues and of the soft parts; in some cases the soft parts are more hyperplastic than the bones. A common statement in the history is that the patient "must each year buy larger-sized hats, gloves or shoes." The nails are thin and small. The antithesis of acromegaly is micromelia (Hutchinson). (*c*) The *changes* in the trunk are less conspicuous. The sternum is increased anteroposteriorly and the ribs and clavicles are thickened. The anteroposterior dimension of the thorax is increased, the head is held backward and the dorsal kyphosis is rather characteristic. The muscular weakness and lean arms and legs contrast sharply with the first impression of strength imparted by the massive face and extremities. The breathing is largely abdominal. (*d*) The *skin* is hyperplastic and also the mucous membrane of the nose, tongue, pharynx and larynx. The larynx is enlarged (Marie), which may explain the deep, low, rough, monotonous voice.

2. Nervous Manifestations.—(*a*) Symptoms of *brain tumor* (hypophysis) are common, as headache, vertigo, mental alteration, vomiting, periodic loss of consciousness, early and frequent bitemporal hemianopsia, optic atrophy (40 per cent.), ocular paralysis, anosmia, auditory disturbance and exophthalmos. Hertel in 175 cases found eye symptoms in 52.5 per cent. (*b*) *Changes in character,* anxiety, apathy and insomnia which deepens into terminal somnolence, are common; severe pains, paræsthesia, acroparæsthesia and subjective warmth are frequent.

3. Constitutional Symptoms.—(*a*) *Glycosuria* is more frequent than Hansemann's figures indicate (12 per cent.). Its relation to acromegaly is uncertain, probably being a coördinate manifestation. Sometimes polyuria occurs without glycosuria. (*b*) *Profuse sweats* are common, even with glycosuria; the skin is sometimes pigmented; obesity is common. (*c*) *Sexual disturbance* is the rule; in women amenorrhœa is practically constant and is often preceded by dysmenorrhœa; in men impotence is not unusual; the genitalia are much oftener infantile than hypertrophied. (*d*) The *heart* is often *dilated* and *hypertrophied* from the frequent concomitant *arteriosclerosis,* deformed chest and rarely from actual overgrowth (cardiomegaly). The vessels are wide and thick. Splanchnomegaly may be observed

(intestines and other viscera). In most cases the lymph nodes, particularly the cervical, are palpably enlarged, the tonsils, spleen, thymus and sometimes the thyroid are hyperplastic.

Course and Prognosis.—(*a*) Acromegalic subjects seek advice for the enlargement of the extremities or tonsils, sweats, pain, eye-symptoms or polyuria; (*b*) remissions are common, the constitutional symptoms and, to a slight extent, the enlargements subside; on the other hand, a fitful advance may be noted. (*c*) The average course is *chronic*, covering ten to twenty years, though relatively benign cases occur, lasting thirty or even fifty years, and acute malignant cases are known, which are always due to sarcoma; they may develop in a few weeks and average two to four years in duration. (*d*) *Death is inevitable;* it may result suddenly, as in brain tumor or from gradual marasmus; diabetic coma, intercurrent infection, cardiac insufficiency or pyelonephritis may explain the fatal issue.

Diagnosis.—In this connection, Strümpell quotes Charcot's maxim that physicians in general see and diagnosticate only what they have seen and learned to diagnosticate; the diagnosis is made at a glance by one who has seen a single case; undue regard to a single symptom, as the skin changes, amenorrhœa or glycosuria, may cause error, but the grouping of acromegalic findings is unmistakable.

Differentiation.—1. In the *cranium progenium,* the lower jaw protrudes and its teeth are directed forward; the lower jaw is hyperplastic, the upper jaw is hypoplastic. Cranium progenium may occur in acromegaly, cretinism, healthy individuals after variola, in degeneracy and in idiocy.

2. *Gigantism* occurs in over 20 per cent. of acromegaly, particularly when acromegaly develops early in life. Forty per cent. of giants are acromegalic (Sternberg). The interrelation of these conditions is incompletely understood. Gigantism proper implies symmetry of overgrowth; there are normal "giants" and giants with acromegaly, leontiasis (*v. i.*), facial hemihypertrophy and hereditary syphilis. Bassoe's report of the autopsy of the giant Wilkins covers the entire subject (*Journal of Mental and Nervous Disease,* 1903).

3. *Diffuse hyperostosis* (Starr, Putnam, Prince) occurs in the young, involves all the skull bones, and seldom the extremities or spine; exophthalmos, blindness, deafness, bilateral facial palsy, dysphagia, dyspnœa and other basilar symptoms are noted, which culminate fatally with stupor, idiocy, epileptiform convulsions and paralysis of the extremities. A sub-type (leontiasis ossea, Dana, 1893) occurs in the form of tumor-like protuberances on the skull and other bones. Prince (1902) collected 20 cases from the literature. He maintains that it is a form of osteitis deformans.

4. *Osteitis deformans* (Paget's disease, 1887) is a combined rarefying and hypertrophic osteitis. It begins slowly in subjects over forty years old, with pains and deformity of the thighs, legs and spine; the legs bow widely outward and somewhat forward, the cervical and upper dorsal spine becomes kyphotic, the thorax narrows

transversely and widens anteroposteriorly, the abdomen is diamond-shaped and crossed by a sulcus and the head hangs forward. The soft parts do not hypertrophy as in acromegaly, the face is rarely involved, the head is larger above than below and the arms are little altered. About 70 cases are on record.

5. *Hypertrophic osteoarthropathy,* known since the time of Hippocrates, was especially described by Bamberger and simultaneously by Marie, in 1889. The ends of the fingers and perhaps the toes become clubbed or "drumstick" in appearance. While the nails are thin and small in acromegaly, in hypertrophic osteoarthropathy they are large, thick, wide and curved downward. The alteration typically involves the terminal phalanges, but also in severe cases the ends of the ulna, radius, tibia and fibula. The pathology is a toxæmic, chronic periostitis, with hypertrophy and osteophytic growth. The most common cause is chronic lung disease (chronic phthisis, bronchiectasis, emphysema or fibrosis), whence Marie's name of *osteo-arthropathie hypertrophiante pneumonique;* it may develop in congenital heart disease or aneurysm or, much less frequently, in icterus, gastric dilatation, syphilis, cystitis or various infections.

Treatment.—The treatment of acromegaly is merely symptomatic. Extract of the hypophysis has been given and in a few cases with temporary success. Hochnegg operated upon one case successfully.

SECTION VIII.

CONSTITUTIONAL DISEASES.

DIABETES MELLITUS.

Definition.—A disease of metabolism characterized (*a*) by a permanent *lessened capacity* of the organs fixing, storing and consuming *grape sugar;* (*b*) by excess of sugar in the blood (*hyperglycæmia*), resulting in (*c*) grape sugar in the urine (*glycosuria*); and (*d*) *causal changes* in *the pancreas.*

Willis (1674) differentiated between diabetes mellitus and diabetes insipidus and Dobson (1776), of Liverpool, fully described the clinical symptoms, although Aretæus (150 A. D.) used the term diabetes.

The liver, the reservoir for carbohydrates, may contain even 14 per cent. of glycogen. It collects carbohydrates from the portal vein, stores them as glycogen and again imparts them to the blood as grape sugar, for use in the tissues. The muscles also manufacture glycogen from the grape sugar of the blood and are more tenacious of it than is the liver. Glycogen may also be produced from the albumins. When there is a temporary carbohydrate shortage, the reserve store of glycogen is drawn upon. Fat is converted in the liver into sugar when neither the carbohydrates nor the albuminates suffice to maintain the normal proportion of sugar in the blood. The fate of carbohydrates when ingested in excess is: (*a*) glycogen storage (up to 10 ounces); (*b*) conversion into fat; (*c*) glycosuria from hyperglycæmia, when the sugar in the arterial blood exceeds 0.2 per cent.; this is known as "alimentary glycosuria," which is physiological, wholly distinct from diabetes mellitus, and occurs when the blood-sugar exceeds the assimilation limit. The test for alimentary glycosuria consists of giving 3 ounces of grape sugar, after a light breakfast of bread and coffee. If more than 1 per cent. of sugar appears in the urine or if 2 or 3 per cent. of the entire amount administered is found it is indicative of diabetes rather than of glycosuria. Most absorbed sugar goes to the liver, but some absorbed by the lymphatics appears in the urine, whence its presence is not necessarily a sign of diabetes; in liver cirrhosis, sugar may reach the blood with the collateral circulation. Traces of sugar (0.01 to 0.02 per cent.) are normally present in the urine, but are not detected by the usual tests, whence healthy urine is regarded as free of sugar.

Etiology.—1. The *pancreas* (*q. v.*) has long been considered an etiological factor in diabetes. Opie (1900) discovered *atrophic*

changes in the cells of the islands of Langerhans, by microscopic examination. These cells are thought to produce an *internal secretion,* concerned in breaking up sugar in the tissues and muscles. This internal secretion from Langerhans' islands is wholly distinct from the external secretion of the pancreatic juice voided into the intestine. Lépine (1892) spoke of a "glycolytic ferment," which entered the blood from the pancreas and broke up the sugar molecules; in diabetes resulting from the absence of the ferment the sugar is not broken down. This ingenious argument has received much experimental corroboration, since (*a*) total pancreatic extirpation in animals and man produces glycosuria; (*b*) after partial extirpation and suture of the remaining portion to the abdominal wall, diabetes fails to appear, but develops if this portion is later removed; (*c*) when about a tenth part is left, a mild form of diabetes ensues and glycosuria occurs only after the ingestion of carbohydrates; (*d*) subsequent destruction of the remaining portion of the gland produces a severe form; (*e*) when more than one-tenth of the functionating gland is left behind no diabetes ordinarily results; (*f*) diabetes does not occur in simple ligature of the pancreatic duct, excluding the juice from the intestine, nor when the fluid escapes through a cutaneous fistula, whence the argument that there is an internal secretion (Minkowski and v. Mehring). (*g*) Cohnheim's experiments (1903) suggest that a ferment or pro-enzyme secreted by the pancreas, joins another ferment secreted by the muscles and that the united ferments break down the sugar molecules to produce heat and energy. In 167 patients, Thoinot and Delamare found "Langerhans' insufficiency" in 80 per cent., and chiefly in "lean diabetics."

2. There is a *neurogenous diabetes,* the relations of which (if there are any) to the pancreas are wholly obscure. This and the following type might be considered as symptomatic diabetes. Nervous lesions, as Claude Bernard's well-known puncture of the fourth ventricle (1844), may possibly alter the circulation in the liver. Among them are apoplexy, tabes, brain tumors (particularly those located near the base), general paralysis, multiple sclerosis, meningitis, syphilis, softening of the brain, diseases of the cord, vagus and sympathetic system, trauma, traumatic neurosis, paralysis agitans, mental diseases, Basedow's disease and acromegaly. Hoffman distinguishes between (*a*) *neurogenous diabetes,* which occurs chiefly in men, with no tendency to furuncles, carbuncles or cataract, with no connection with gout, and which passes away in a comparatively short time if the brain affection is curable; and (*b*) *diabetes of the obese,* which occurs as frequently in women as in men, often produces carbuncles, is often connected with gout and is very chronic, often lasting ten or twenty years.

3. *Obesity* may be a cause; according to von Noorden, "There are (*a*) cases in which the consumption of sugar and its conversion into fat are simultaneously restricted—glycosuria which varies in severity, and in degree of emaciation (ordinary diabetes); (*b*) cases in which

only the consumption of sugar and not its conversion into fat is interfered with—obesity without glycosuria (masked diabetes); these pass readily at a later period into (*c*) cases in which the consumption of sugar is restricted, and the collecting of carbohydrates in the fatty tissues is also more or less interfered with—obesity with consecutive glycosuria (ordinary diabetes of the adipose)."

4. *Gout, arteriosclerosis*, various *liver lesions* (cirrhosis) and possibly acute infections have apparent though obscure relations with diabetes. Adrenalin may produce glycosuria (Blum and Herter). Renal diabetes (glycosuria without excessive sugar in the blood) may occasionally develop in nephritis, renal hemorrhage, chyluria and from administration of phloridzin. *Lactosuria* may occur in late pregnancy, the puerperium and nurslings; von Noorden's hypothesis is that in the "puerperal state the capacity of the tissues for breaking up milk-sugar is diminished, an instance of adaptation to an end in that the cells of the mother refuse a material which is preëminently suited to the nutritive wants of the nursling."

5. *Predisposing factors* are (*a*) *sex;* men (3:2) are especially subject to diabetes; (*b*) *age;* though it occurs at any age, its frequency is as follows: In persons from thirty to forty years, 18 per cent.; forty to fifty years, 25 per cent.; fifty to sixty years, 30 per cent.; in Stern's collection of 117 infantile cases one was apparently congenital. (*c*) *Heredity.* A family history of obesity, consanguinity, neuropathic taint (20 per cent.) or gout is especially significant; cases are reported in which the grandfather was diabetic, the son gouty and the grandson again diabetic (hereditary alternating diabetes). (*d*) *Race.* Diabetes is comparatively rare in the colored race and is especially common in Jews. It is rather less common in America than in Europe. (*e*) *Occupation.* Diabetes is more often seen in private than in hospital practice. Wealth and culture increase ten-fold the predisposition to diabetes (von Noorden). It is frequently found in scientists, lawyers, musicians, poets, teachers, statesmen, merchants, speculators and in those leading a luxurious or self-indulgent existence with sedentary habits; it also results from overwork, excitement, over-eating and over-drinking. Diabetes is apparently increasing in frequency.

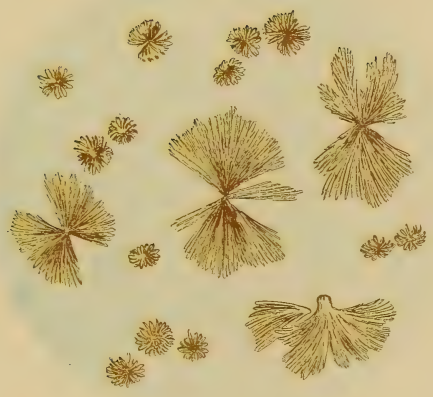
Symptoms.—1. The Urine.—*Glycosuria.*—Sugar in the urine is the leading symptom of diabetes. A daily excretion of 1 to 3 drams (5 to 12 grams) is not uncommon; the maximum amount recorded is 1,500 gm.; the largest quantity of sugar is voided late in the morning or after 6 P. M. A percentage of from 1 to 4 is not uncommon, 8 or 9 per cent. is rare and the maximum is 20 per cent. (personal observation). The estimation of a diabetic's capacity to consume sugar, suggested simultaneously by Seegen and Traube is as follows: Accordingly as the urine, after a diet absolutely free from carbohydrates, is free from sugar or not, a distinction is made between (*a*) mild and (*b*) severe cases of glycosuria. (*a*) *Mild glycosuria* is characterized by the urine becoming sugar-free within a few days after

carbohydrates are withdrawn and by sugar reappearing when carbohydrates are again ingested. The test diet consists of ham, eggs, roast beef, cucumber with vinegar, olive-oil and salt and pepper; brandy with Appolinaris water; coffee without milk or sugar; clear bouillon; butter, green salad, green vegetables and sardines; this diet is free from carbohydrates. With increasing additions of starch, sugar at some time is again voided, *i. e.,* the limit of tolerance or of assimilation is reached. (*b*) *Severe glycosuria* is characterized by continued excretion of sugar in spite of withholding all carbohydrates from the diet and signifies (i) that even those carbohydrates which are slowly formed from the albumin are not wholly consumed; (ii) that the functions of glycogen storage and (iii) of splitting up of sugar in the tissues are seriously affected; of this type there are many degrees. Fat and alcohol never increase the severity of glycosuria. Muscular exercise and massage usually decrease it. It is often increased by psychic disturbances and may be suspended or lessened during an acute intercurrent infection, especially typhoid; chronic diseases with fever, especially pulmonary tuberculosis, have less influence. It is reduced or absent in the final stages of diabetes and may be absent in diabetic coma. Glycosuria very often disappears when granular atrophy of the kidney develops (Frerichs, Stokvis, Fürbringer); the diabetes is then cured in some unknown way. Gouty diabetics as a rule pass no sugar during an attack of gout (diabetes alternans).

Tests for Sugar.—(*a*) For *Fehling's test,* two solutions are kept separate; one contains copper sulphate, 34.64 gm. in 500 c.c. of water, and the other sodium and potassium tartrate 173 gm., and sodium hydrate 125 gm. in 500 c.c. of water. Equal parts of these solutions are heated, but not boiled, and the urine is added drop by drop. The volume of urine should never exceed that of the solution. Reduction of the blue copper solution to the yellow-red suboxide of copper occurs when sugar (its aldehyd group) is present. Chloral, phenacetin, chloroform, formalin, morphine, uroleukinic and homogentisinic acids (the probable causes of alkaptonuria), glycuronic acid, cascara, rhubarb and salicylates may also reduce Fehling's solution, particularly after boiling. In doubtful cases, or as corroborative tests, (*b*) the *fermentation test* may be used; yeast is shaken thoroughly with a quantity of urine, the air allowed to bubble up and the mixture placed in a saccharometer,—a tube closed at the top and graduated for estimation of percentages. The sugar, fermenting, is measured by the amount of carbon dioxide formed. A control test should be made. (*c*) The *polarization test,* showing dextro-rotary reaction, may be employed. (*d*) The very delicate and reliable *phenylhydrazin test* of E. Fischer may be used.[1]

[1] Cipollina's method is the simplest: 5 minims of pure phenylhydrazin, eight drops (0.5 c.c.) of glacial acetic acid, and a dram (4 c.c.) of urine in a test-tube are gently boiled for one minute with frequent agitation. Four or five drops of sodium hydrate solution are added, the mixture is boiled again, and allowed to cool; in a few minutes (to a half an hour) yellow rosettes of phenylglucosazon precipitate; maltose and certain pentoses, the crystals of which melt at different degrees, may alone cause error. Plate XVII.

PLATE XVII



Crystals of Phenyl-glucosazone. (Musser.)

(Oc. 4, Obj. D.) Drawn by J. D. Z. Chase.

Quantity of Urine and Specific Gravity.—The urine is almost always acid even after fermentation (lactic acid) and has a sweet taste. As a general rule the quantity of urine secreted during the night is less than that secreted in the day-time; this is an important point because in other *polyurias* frequently ⅔ to ¾ of the entire amount of urine is secreted during the night (Lecorché and Quincke). A secretion of ten quarts is not rare. The largest amounts recorded are 17 or 28 quarts in a day. Higher figures suggest simulation, patients having been known to add sugar to the urine. *Pollakiuria* (frequency of urination) is common. The urine is *pale.* If dark and if uratic deposits are present, the prognosis is good (Seegen and Pavy). Foam often remains long on its surface and the sediment is very scarce or absent. Prout considered that the onset of diabetes began when the urine ceased having a turbid, uratic deposit. An increase in the amount of sugar usually brings an increase in the amount of urine, but ordinarily the amount of urine rises less rapidly than does that of the sugar. The specific gravity is higher, the greater the amount of urine secreted.

Amount in liters or quarts.	Specific gravity.	Sugar.
1.5 to 2.5	1.025 to 1.030	2 to 3%
2.5 to 4	1.030 to 1.036	3 to 5%
4 to 6	1.032 to 1.046	4 to 7%
6 to 10	1.036 to 1.046	6 to 9%

Two or three per cent. of sugar may cause no change in specific gravity. The average is 1,030–40, it is rarely more than 1,050, and the highest record is 1,074; very high specific gravity suggests fraud; the lowest records are 1,008 and 1,002. If under the influence of meat diet the urine becomes entirely or nearly free from sugar the quantity approaches normal. The specific gravity remains high because the urine is rich in meat end-products. There is no absolute parallelism between the percentage of sugar and the specific gravity; (*a*) glycosuria is often preceded by premonitory polyuria. (*b*) Though diabetes may be cured, polyuria may persist without glycosuria; this may be caused by the habit of drinking large quantities of water. (*c*) In some cases, though there is a large percentage of sugar, only a pint or a pint and a half of urine is excreted, described by Peter Frank a century ago as "diabetes decipiens." Crystals of sugar may be seen on the shoes, clothing or in the vessel.

Nitrogenous Constituents of the Urine.—Diabetic patients excrete considerable urea; 2 ounces are not infrequent and over 5 ounces have been noted; larger figures are reached in no other disease; it is occasioned by the abundant ingestion of proteids and possibly by fermentation of the tissue albumins; the condition is pathological when the amount of nitrogen in the urine exceeds that in the food. Naunyn and von Mehring deny that there is an increase in the urea save in severe and exceptional cases.

Ammonium, normally 8 to 15 grains *per diem,* may increase to

20 or 180 grains, although the highest figures are seen exclusively in diabetic coma. The organism excretes increased amounts of ammonia in order to neutralize pathological acids (acidosis). The diabetic diet is acid; phosphoric acid forms from the ingested meat, and much sulphuric acid is produced by oxidation of sulphur in the albumin molecule. The quantity of creatinin may reach 20 to 30 grains *per diem* for similar reasons. The amount of uric acid is nearly normal.

Albuminuria is found in 35 per cent. of cases; it is almost always slight and is due (*a*) to cystitis, pyelitis, intercurrent diseases as gangrene or infection, pulmonary tuberculosis or without relation to diabetes, as in the arteriosclerotic and gouty types of diabetes, liver cirrhosis, etc.; (*b*) to diabetes in nervous individuals, and (*c*) to circulatory disturbances; (*d*) in uncomplicated diabetes it may be due to the action of the sugar, β-oxybutyric and other acids and toxins on the kidneys. Albuminuria is a frequent forerunner of diabetic coma, as a coördinate manifestation or a determining factor (decreased renal permeability with retention in the blood of toxins). Diabetes may develop into chronic interstitial nephritis and the sugar then disappears.

Non-nitrogenous organic substances. Lipuria has been observed. Acetone, aceto-acetic acid and beta-oxybutyric acid are most frequent; they are derivatives of fat. *Beta-oxybutyric acid* is absent in diabetics in good physical condition and not infrequently also in those who are suffering from marked loss of albumin. The urine may be odorless or may smell like chloroform, fruit or wine. The daily amount excreted varies from a few grains to 2 to even 7 ounces. Its abnormal excretion once begun, is uninterrupted, sometimes lasts years and shows a constant tendency to increase. It is of the gravest prognostic significance because in most cases, at the end of days, weeks or months, fatal diabetic coma develops. An excretion of more than an ounce indicates imminent coma. Acidosis is most accurately estimated by the output of ammonium; according to Magnus Levy, 2 gm. of ammonium correspond to 6 gm. of β-oxybutyric acid, 5 gm. to 20 gm., and 8 gm. to 36 or 40 gm. β-oxybutyric acid is thought by some to be the parent substance of acetone and diacetic acid. *Acetone and diacetone* are present in the urine in diabetes, fevers, cancer, inanition or auto-intoxications and indicate malnutrition. When the amount of acetone progressively rises from 15 grains upward, the outlook is unfavorable. It is not proven that the acetones cause coma, but they at least accompany acidosis. They impart a fruity or wine-like odor to the urine and breath. Legal's test for acetone: To 10 c.c. of urine a few drops of a freshly made solution of sod. nitroprusside are added; the addition of caustic soda gives a red color if acetone is present.

Gerhardt's test for diacetone is as follows: a dram of urine is treated with a strong solution of ferric chloride added drop by drop. A precipitate of phosphates is filtered off and more of the iron solu-

tion is added to the filtrate. If a bordeaux-red color now appears, another portion of the urine is boiled and similarly treated. If in the second test no reaction is obtained a third portion of the urine is treated with sulphuric acid and extracted with ether. A positive reaction, when the ethereal extract is tested with ferric chloride, the color disappearing upon standing for twenty-four to forty-eight hours, indicates diacetic acid, particularly if the urine is rich in acetone.

Mineral Constituents.—Sodium chloride, sulphuric and phosphoric acids and the ethereal phosphates are abundant; they are caused by the increased amount of food ingested. The oxalates are increased in mild cases. Much lime is excreted, which suggests osseous waste.

Pneumaturia.—The butyric acid bacillus, the Bacillus coli, yeast fungus and other bacteria may cause fermentation in the bladder, producing carbon dioxide, hydrogen and methane gas.

2. Direct Consequences of the Glycosuria and Polyuria.—(*a*) The exaggerated *thirst* (*polydipsia*) is always secondary to the polyuria. (*b*) *Nutrition.* Much sugar is lost to the tissues; two quarts of urine with 5 per cent. of sugar produces loss of 400 calories, one-seventh of the total. Therefore the nutritive requirements are great, the appetite is excessive (*polyphagia*) and in severe cases emaciation and fatigue are pronounced. The loss of sugar must be met by increase in albumin and fat. Increase in weight may follow reduction in the sugar. The temperature in uncomplicated cases is subnormal from inanition.

3. Complications.—Complications and other symptoms result from various causes: (*a*) the sugar in the blood, (*b*) impaired nutrition and lowered physiological resistance; both conditions predispose to (*c*) infection, particularly by the bacillus tuberculosis, (*d*) poisoning (acidosis) and other ill-understood degenerations and toxæmia.

1. *Nervous.*—(*a*) *Intellectual depression,* insomnia, headache and neurasthenic symptoms are common; psychoses are less frequent. In rare instances focal symptoms may develop without anatomical findings, as in uræmia. Brain symptoms may result from coincident syphilis, arteriosclerosis, etc. (*b*) *Diabetic coma* occurs in 20 per cent. and causes about half the deaths in diabetes; it is most frequent in youth and in acute and severe types; Naunyn holds that it is favored by a strict antidiabetic diet. All coma in diabetes is not diabetic; patients may succumb (i) to cerebral *apoplexy,* embolism, thrombosis, sepsis or *uræmia,* (ii) to *heart failure,* which may cause sudden death, or coma with death after a short time or (iii) *diabetic coma* proper, which is probably due to acidosis (Stadelmann) and closely resembles experimental acid poisoning. Most writers believe that β-oxybutyric acid is causal, though in three of the author's cases it was not present in the urine (Long). Coma is imminent when the daily output of ammonium exceeds 45 grains. It may occur suddenly without obvious cause or may follow severe exertion, excesses, excitement or gastro-intestinal disorders. In some cases there are prodromal headache, vertigo, stupor, restless anxiety or symptoms re-

sembling those of acute alcoholism. The pupils are dilated, the reflexes abolished, the pulse is small and averages 100 to 120 and the temperature is usually subnormal. Convulsions are most uncommon. The breath has the wine-like acetone odor. The breathing is characteristic; inspiration is deep, long-drawn and energetic and expiration is short and slightly sighing; in this, *Kussmaul's dyspnœa* (1876), the inspiratory energy contrasts strongly with the general bodily weakness; respiration may be normal in rate, but is generally somewhat accelerated; there is no stridor, nor, at the onset, any cyanosis. The urine is generally albuminous and almost always contains small granular casts (Külz); the addition of a few drops of ferric chloride gives the Burgundy wine color; sugar is present, though it may disappear as the coma develops. Partial consciousness may return, but, with few exceptions, death occurs within a few hours to two days, with subnormal temperature, slowed respiration and cardiac weakness. The autopsy reveals no constant brain changes. (*c*) Degeneration (toxæmic) in the posterior columns of the cord may develop. (*d*) The *peripheral nerves. Neuralgia* is common and may indicate incipient neuritis or may appear and disappear, varying with the amount of sugar in the blood and urine. It is often an early symptom; it may be unilateral or more often bilateral, most frequently affects the sciatic nerves and is a most obstinate and recurrent symptom. *Neuritis* occurs in the severe forms of diabetes and in its later stages. It may be manifested only by obstinate neuralgia, painful paræsthesia in the legs, muscular cramps or in 50 per cent. of diabetics, decrease or abolition of the patellar reflexes (Bouchard). Neuritis may involve the cranial nerves, mostly the abducens, then the oculomotor, hypoglossus, spinal accessory and facial, or with greater frequency the spinal nerves; the lower are affected more often than the upper extremities and, like diabetic neuralgia, bilateral involvement of the crural or sciatic nerves is the most common type. Neuritis may be single or multiple. Its symptoms are those of neuritis or polyneuritis (*q. v.*), as paralysis, altered sensation, abolished reflexes, reaction of degeneration and trophic disturbances, as falling of the hair and nails, glossy skin and perhaps also herpes or perforating ulcer of the foot (*mal perforant*). In severe cases tabes is simulated, *pseudotabes diabetica,* first described by Fischer (1886) (see MULTIPLE NEURITIS). (*e*) The *special senses.* Retinal diseases (Jäger, 1856) occur under three forms: (i) albuminuric retinitis, accompanying contracted kidney; (ii) *retinitis centralis punctata* with characteristic small, shining central spots, usually with hemorrhagic points and always bilateral; (iii) the ordinary hemorrhagic type (see Plate V, Fig. 1). Retinitis occurred in 19 per cent. of Galezowski's 114 cases. Neuritis, neuroretinitis and retrobulbar neuritis with consecutive atrophy are progressive and occur more frequently in severe than in mild cases, sometimes as early, sometimes as late, complications. Twenty per cent. of diabetics with retinitis die within one year and 60 per cent. live over two years (Nettleship).

Amblyopia and amaurosis are complications of early diabetes and often first induce the patient to seek medical advice. Paralysis of the ocular muscles occurs in 7 per cent. of cases; accommodation paralysis is the most frequent type, and then that of the external rectus; the most probable cause is neuritis; Mauthner assumes that there is a nuclear hemorrhage. Cataract occurs in 4 per cent. of cases. Other ocular changes are rare. Aural complications include external furuncles and otitis media, which latter has a tendency to hemorrhage and diffusion, as mastoiditis.

2. *Circulatory.*—(*a*) The *blood* may become somewhat concentrated, for much water is required to eliminate the sugar; the amount of sugar (normally 0.1 to 0.15 per cent.) is increased in diabetes to 0.3 or 0.5 per cent. Fat in the blood (lipæmia) is a frequent finding; what appears to be fat is sometimes albumin (Futcher) or cholesterin in combination with fatty acids; Frazer in one case found 14 per cent. of fat. *Bremer's test* is based on changes in the hæmoglobin; thick smears of blood are made on a slide, which is heated and treated with 1 per cent. congo red solution for two minutes; diabetic blood will not stain, but the control slide of normal blood stains; this reaction, according to Bremer, often results during remissions in the glycosuria and in the preglycosuric stage; it sometimes occurs in other conditions, as Hodgkin's disease. *Williamson's test* is based on the fact that diabetic blood decolorizes a weak alkaline solution of methyl blue. It is as follows: 20 c.c. of blood, 40 c.c. of distilled water and 1 c.c. of 1 to 6,000 aqueous solution of methylene blue are added to 40 c.c. of liquor potassæ and are placed in test-tubes in a beaker of water, which is boiled for four minutes; the blue color disappears. The *alkalescence* of the blood, which is reduced in various conditions of inanition, is greatly lessened in diabetes, particularly when β-oxybutyric acid engages the ammonium and other salts and when coma develops. (*b*) The *arteries* are frequently sclerosed in diabetes. Arteriosclerosis causes many common symptoms, as cardiac hypertrophy or dilatation, angina pectoris, cardiac asthma, hemiplegia, albuminuria, senile gangrene and intermittent claudication. (*c*) The *heart* may be altered in various ways (*v. s.*). Hypertrophy occurs in about 10 per cent. and is less ominous than atrophy, which occurs in uncomplicated diabetes of the young. Heart-failure may occur after moderate exercise or may develop precipitately, ending in coma or asphyxia and correctly separated by Frerichs from genuine diabetic coma. Von Noorden observed five instances of sudden death in 140 fatal cases. "Functional heart symptoms" occur in 10 per cent. of cases. The pulse is usually accelerated and its tension is elevated. Cardiac or renal dropsy is uncommon in diabetes.

3. *Respiratory.*—(*a*) *Tuberculosis* is most frequent in the poor classes and in the young, but much less so in older, obese and gouty subjects; about 25 per cent. of diabetics contract tuberculosis. Hæmoptysis is very rare; the sputum is abundant; it is usually stated that the tubercle bacilli are scant or absent; in the secondary

necrosis, to which all diabetics are prone, the marked clinical and pathological lung-findings may sharply contrast with the small number of tubercle bacilli found in the sputum. The course is rapidly fatal. (*b*) *Gangrene* is much less common than tuberculosis. Fever, pulmonary hemorrhage and purulent sputum are common. Fœtor is less conspicuous than in other forms of pulmonary gangrene. (*c*) Intercurrent pneumonia may develop and is usually fatal.

4. *Digestive.*—(*a*) The *mouth* is generally dry and acid (from decomposition of sugar); the saliva is scanty and poor in ferment; stomatitis is common and the growth of aphthæ is promoted by the acid reaction of the mouth, though it is preventable by cleanliness. The teeth may decay from alveolar periostitis or drop out from trophoneurotic changes. (*b*) The *stomach* is affected with remarkable infrequency, considering the oral findings and the polyphagia. Marked anorexia is uncommon, though the patient may refuse fatty and nitrogenous foods. The stomach is usually dilated. (*c*) The *bowels* are generally costive. Schmitz claims that obstinate constipation predisposes to coma, though in the author's experience, most diabetics are constipated. Intestinal or gastro-intestinal catarrh sometimes seems to precipitate coma, but it may be merely an associated symptom. (*d*) The *liver* is usually enlarged (hyperæmia and fatty degeneration) and sometimes tender. Gall-stones are concomitants in 10 per cent. of cases, but Naunyn correctly denies that they have any intrinsic relationship to diabetes. French authors (Hanot and Chauffard) have described a pigmentary cirrhosis in diabetes (diabète bronzé), which has already been referred to under liver cirrhosis and chronic pancreatitis; this syndrome probably develops in the following sequence; (i) hæmolysis, (ii) pigment deposit in the liver, pancreas and skin from the altered blood (hæmochromatosis), (iii) chronic interstitial inflammation, chronic hepatic cirrhosis and (iv) chronic pancreatitis, causing diabetes. (*e*) *Pancreatic* symptoms are few, because the external pancreatic secretion is essentially normal, and only the internal glycolytic secretion is affected.

5. *Genito-urinary.*—(*a*) Albuminuria may be present (see URINE). The kidneys are frequently hypertrophied and hyperæmic; various degenerations may be noted, including Ehrlich's glycogenic degeneration. Interstitial nephritis may follow and replace diabetes, chiefly in gouty and corpulent individuals. (*b*) Cystitis is common; the urine may ferment in the bladder, just as it readily decomposes in the urinal; this may lead to pyelonephritis or pneumaturia (*v. s.*). Cystitis may cause temporary disappearance of the glycosuria. (*c*) In the female, menstruation may be disturbed or suspended. The sexual inclination is usually lessened in severe cases, but may be increased from genital pruritus (*v. i.*). Conception may occur even in advanced cases, but in 33 per cent. there is spontaneous abortion or premature delivery. In severe diabetes interruption of pregnancy is indicated; the maternal danger in uninterrupted pregnancy is great, from exacerbation of the disease, coma, collapse and infection.

Fungi develop about the labia and vagina; this is promoted by lessened general resistance and local contact with the saccharine urine. Aphthæ, leptothrix and other parasitic deposits are frequent, particularly in careless subjects; *pruritus pudendorum,* vulvitis, vaginitis, urethritis, furunculosis and even gangrene may develop. (*d*) In the male, the sexual appetite may be increased early in the disease but later is replaced by impotence, probably due to spinal degeneration. Phimosis, balanitis, small erosions near the urethra, urethritis, itching at the meatus causing tenesmus, curious nodes on the corpora cavernosa, furuncles and necrosis may first engage the physician's attention. Genital symptoms in men are less conspicuous than in women.

6. *Cutaneous.*—The skin is dry, harsh and itches when there is much polyuria and emaciation. Sugar is said to be present when the patient sweats. Itching is caused by toxæmia irritating the cutaneous nerves, as in uræmic and cholæmic pruritus. Skin eruptions include pityriasis tabescentium, urticaria, erythema, eczema, bullæ, acne, purpura and herpes. Infection is favored by reduced physiological resistance and hyperglycæmia; furunculosis (10 to 25 per cent.) is due largely to the staphylococcus and may initiate lymphangitis, phlegmon or gangrene; erysipelas is promoted by reduced general and local resistance. Gangrene is most frequent in late or middle life, and in mild cases; it usually begins on the toes and extends upward, as dry or moist gangrene; it is said to result from obliterating endarteritis, but in the writer's experience occurs three times less frequently than the ordinary plaque-like atheroma. Unfortunately gangrene is usually progressive. Wounds in diabetics heal better than in pre-antiseptic times, but delayed healing, infection and gangrene are still frequent. Enlargement of the lymphatic glands is seldom absent in severe, progressive forms with emaciation; the glands are hard, painless and probably secondary to skin infection or irritation.

Course and Prognosis.—The onset may be acute or chronic and the course may be short, covering weeks, or protracted and remittent, covering twenty years or more. Complications modify the duration and outcome. Von Noorden's summary of the *prognostic signs* covers the ground completely: *Favorable* signs are: (*a*) onset late in life; (*b*) long duration prior to the time of observation, with no grave complications or any considerable emaciation; (*c*) traumatic or (*d*) syphilitic origin; (*e*) the occurrence of mild forms of diabetes in the family of the patient; (*f*) precedent and co-existent obesity; (*g*) co-existent uric acid diathesis; (*h*) slight glycosuria and tolerance of moderate amounts of carbohydrates; (*i*) marked oscillations and increasing tolerance of carbohydrates; (*j*) circumstances which permit of dietetic and general hygienic prescriptions. *Unfavorable* prognostics are: (*a*) onset in early life, especially in childhood; (*b*) great loss of strength, despite a brief duration of the diabetes; (*c*) a history of severe forms in the family; (*d*) early appearance of

grave secondary diseases and complications; (*e*) a high degree of glycosuria, with complete intolerance of carbohydrates; (*f*) excretion of β-oxybutyric acid; coma; (*g*) circumstances rendering impossible dietetic treatment or avoidance of bodily and mental overwork.

Diagnosis.—The diagnosis is certain only when the urine in every case is examined as a routine procedure, because only then can the lighter cases, amenable to treatment, be discovered. In many cases the thirst, polyuria, emaciation, itching and the complications *per se* are suggestive. *Pentosuria;* 19 familial or hereditary cases are recorded. Pentose reduces Fehling's solution, even after fermentation of the urine. Bial's test is made as follows: a small amount of the following solution, orcin 1 gm., liq. ferri chloridi 25 drops, and 30 per cent. hydrochloric acid in 500 c.c. of water, is heated to boiling and added to the urine, which turns green if pentose is present.

Treatment.—1. DIET.—Regulation of diet, dating from Rollo (1797), is the most essential point. The carbohydrates normally supply nearly half the bodily requirements for food and energy. The diabetic loses a large part by their escape through the kidneys. Furthermore, their ingestion implies a waste of digestive energy, an irritation of the tissues by the unused sugar and a growing decrease of the tolerance of the tissues for them. A man weighing 70 kilograms (150 lbs.) requires a diet representing 2,500 calories, *i. e.,* roughly 35 calories for each kilogram. The dietary must therefore regard not merely the glycosuria, but the maintenance of body-weight and energy. It is useless to suppress the symptom, glycosuria, when the patient emaciates. The therapeutic aim is to limit the carbohydrates and replace them with albumin and fat. Naunyn and Weintraud have proven that exclusion of carbohydrates for some weeks increases the tolerance for carbohydrates taken afterward; *i. e.,* the "sugar-consuming function" is rested and in part restored.

General Outlines of Dietary.—Explicit written directions should be given to each patient. *All diabetics may eat the following foods: meat,* the muscular parts of beef, veal, mutton and game, and the tongue, lung, heart, brain, marrow and sweet-breads; also fatty liver, which contains little glycogen; meat peptones, somatose and kindred preparations, gelatin and meat jellies; *fish,* caviar, cod-liver oil, clams, oysters, lobsters and crabs; *eggs* (10 to 20 per cent. of fat); animal and vegetable *fats,* bacon, suet, olive oil, cocoa butter; butter contains 85 per cent. fat, and should be used on meats, eggs or spinach or melted in milk; rich cream; all *cheeses* (3 to 30 per cent. fat), especially Swiss varieties; *vegetables,* cress, tomatoes, cabbage, cauliflower, sprouts, artichokes, mushrooms, truffles, olives, onions, garlic, celery leaves, lettuce, cucumbers and radishes; of *fruits,* only whortleberries, young raspberries and green gooseberries should be eaten, though fruit sugar (levulose) is better tolerated than starch or cane-sugar. *Desserts* (soufflés) of eggs, gelatin and lemon; tea, coffee, diabetic cocoa (Rademann's) and von Hoevel's saccharine chocolate may also be taken.

To some cases, and in definite amounts, von Noorden allows the following (after determining the limit of tolerance, as outlined under glycosuria, *v. s.*); beans and peas (℥j); turnips, carrots, celery bulbs, wax beans (℥j); radishes (℥j); walnuts 6; hazel-nuts 10; almonds 8; (no chestnuts); apples, pears, apricots, peaches (℥j to ij); berries (℥ss to j); cooked apples, plums, pears, peaches, cherries (℥j); and milk (℥viij, of which many practitioners allow a larger measure).

Gradual withdrawal of carbohydrates is tolerated better than abrupt restriction. Glutin flours differ but little from ordinary flours in their percentage of starch. Aleuronat flour contains 7 per cent. of starch. Von Mehring believes that fat is best absorbed when some carbohydrates are given with it. If some starch can be given, Mossé prefers *potatoes,* which are more easily assimilated and contain but 16 to 24 per cent. of starch (bread contains 55 per cent.). Ingestion of more than 5 ounces of *butter* daily may increase the β-oxybutyric acid, but even more may be taken if the butter is carefully washed in cold water, which extracts the lower fatty acids. Saccharin should be used in place of sugar, gr. iss daily; it is 280 times as sweet and is antifermentative, but an excess may occasion dyspepsia. Naunyn advises an occasional *fast day* and Austin Flint kept his cases in bed and without food from Saturday night until Monday morning. As the disease is eventually progressive, a schematic diet should not be insisted upon; reasonable restriction, with *occasional dietetic treats* is less often violated by the diabetic, who is always hungry for bread and potatoes. The patient's weight, color and strength are of more significance than the urine. *Alcohol* aids in the absorption of fat, prevents tissue waste and contributes energy and heat.

Special diabetic treatment of the individual forms (according to von Noorden). (*a*) *Mild forms of glycosuria; in elderly persons* it is not necessary to determine with great accuracy the sugar tolerance. The patient may eat bread, potatoes and vegetables, according to his desire, for the quantity of sugar in the urine is usually insignificant (1 to 2 per cent.). The older the patient and the more corpulent, the more carefully should the body-weight be reduced; fatty foods should be increased, butter being used freely on bread, potatoes, vegetables and meat; and the patient should have bacon, eggs, fatty cheese, light wine, coffee, tea and carbonated water. Muscular *exercise* should be regulated according to the bodily strength and condition of the heart. A course at Carlsbad is beneficial for obesity, gout or mild circulatory weakness. In *young persons,* carbohydrates should be given with much greater caution, as carelessness in diet is more frequent and the glycosuria more often progressive. The limits of tolerance for carbohydrates should be closely watched. All carbohydrates must be forbidden excepting bread and potatoes, in addition to which the patient should have the following articles: 2 ounces of butter, with bread, cheese and potatoes (480 calories); 2 eggs (150 calories); 3 to 4 ounces of olive oil with salad, cucumbers, etc. (90 calories); 1 ounce of fatty cheese (115 calories); 1 quart of milk (590 calories); and

one ounce alcohol (210 calories); meats, *v. s.* (*b*) *Moderately severe forms* embrace cases in which the excretion of sugar is reduced to 1 to 2 per cent. only by total abstinence from carbohydrates. The rise or fall of the tolerance limit should be watched every month, and every four or six months all carbohydrates must be withdrawn for three weeks. The diet must contain much fat; butter, 3 to 4 ounces (890 calories) must be weighed out every morning and eaten on bread, vegetables, meat, fish, etc., either in solid form or melted. Olive oil, two-thirds of an ounce (186 calories), should be taken on salad (lettuce, chicory, cress, cucumbers, tomatoes, red cabbage) and the same amount as mayonnaise dressing for salad, cold meat, fish, lobster, etc. Bacon, two-thirds of an ounce (140 calories), 5 eggs (390 calories) and one ounce of brandy (280 calories) should be given. In addition the patient should have meats and some of the articles mentioned in the conditional list under general diet. (*c*) In *severe forms,* despite a continued rigid diet, sugar is constantly excreted. These cases usually occur in early life with emaciation, frequent complications and a fatal issue after a few months or years. Careful dietetic treatment may retard the progress of the disease, prevent complications and delay the fatal issue. The dietetic treatment is the same as in moderately severe cases. Carbohydrates aggravate the diabetes, but cannot be wholly interdicted. Less starch and more fats and alcohol are given than in other cases.

2. General Treatment.—Zimmer demonstrated the value of (*a*) *exercise* in lessening glycosuria, but as in obesity we have to deal with an added factor, the improved appetite. The condition of the heart must govern the amount of exercise to be taken, as Frerichs early noted that fatal syncope and diabetic coma may follow exhausting exertion. (*b*) The *mouth* and *skin* require special attention. Cleanliness averts in large part gingivitis, furunculosis and genital complications. Frequent warm (not cold) baths and change of underwear, gentle brushing of the teeth and soothing salves for eczema and similar eruptions are imperative. (*c*) Diabetics for the most part should be kept at home and at work, for they often become restless and desire to wander. A prolonged stay in a quiet place is better than travel. Sea bathing and exposure to cold and dampness are to be avoided. (*d*) *Suggestive* treatment may often be combined with the dietetic and medicinal therapy in depressed subjects, particularly in neurotics. (*e*) *Mineral springs* are suitable only for mild cases and those with obesity, gout and slight circulatory disturbance, in whom there is increased tolerance for carbohydrates. Carlsbad and other spas are contra-indicated in severe, youthful, very aged, reduced and nephritic cases. (*f*) The *mode of life* should be quiet and avoidance of excesses is indicated.

3. Treatment of Complications.—(*a*) *Tuberculosis* is the most serious complication. The liberal diet, necessitated by tuberculosis, is contra-indicated by glycosuria. A low altitude and mild climate should be selected. (*b*) *Gastro-intestinal* catarrh often precipitates

diabetic coma. As in infantile disorders, starvation for one or two days is more efficient than attempts at intestinal disinfection; only water should be given; red wine is often indicated when there is exhaustion, acting as a stimulant and astringent. (*c*) *Constipation* (*q. v.*) should be relieved by rhubarb and sodium bicarbonate; severe purges must be avoided. (*d*) *Nervous symptoms* of the neurasthenic order are observed in neurogenous diabetes; sodium bromide, opium, codeine or acetanilide may be indicated. Of all remedies, opium alone exerts an appreciable influence upon the glycosuria; it was recommended by Dobson (1776); it affects the severe more than the light type, *i. e.,* it decreases the transformation of albumins into sugar, but does not control the transformation of carbohydrates into sugar; the average dose may be gradually increased without the patient exhibiting toxic symptoms or suffering from its reduction afterward. Codeine may be given in doses of gr. j. to x, though less effective than opium; it may be combined with sodium bromide ʒss. Inunctions of mercury are seldom valuable, but are indicated when cerebral or pancreatic syphilis is suspected. (*f*) *Neuralgia* (*q. v.*) and neuritis should be treated dietetically; the salicylates may benefit mild cases as well as somewhat modify coincident gout or intestinal fermentation. (*g*) *Acidosis,* acetonæmia and *diabetic coma* are serious complications. Acidosis is said to be promoted by a rigid dietary (Naunyn, von Mehring); in pronounced acidosis even von Noorden, who opposed Naunyn's view, recommends an oatmeal diet, with butter, āā ʒviij at frequent intervals; he adds the whites of 8 eggs to this dietary which is preceded for a few days by a vegetable diet; this diet is indicated in adolescent or juvenile diabetes, particularly in severe types with acidosis and in moderately severe cases in which it helps to develop carbohydrate tolerance. Sodium bicarbonate and citrate ʒij to ʒiij, t. i. d., and calcium carbonate ʒj once or twice a day may possibly postpone threatening coma. Much water should be given by mouth, by rectum or subcutaneously with sodium carbonate ʒiss to Oij. Developed coma is practically incurable. Oxygen inhalations, alcoholic stimulation, strychnine, mild laxatives (not drastics) and subcutaneous or intravenous injections are given, but rarely with success. (*h*) *Gangrene* should be treated expectantly until there is a possible halt in the process and definite demarcation; positive rules of treatment are laid down with difficulty. If there is no acidosis diet is usually efficacious; if acidosis obtains, surgery is indicated. In personal experience, advanced diabetics stand operative shock and anæsthesia poorly. Most of them have died from alarmingly rapid syncope or from typical dyspnœic coma. Other operations may be performed, when indicated as absolutely necessary life-saving procedures and only, if possible, after strict dieting. Boils, furuncles, etc., are treated expectantly.

DIABETES INSIPIDUS.

Definition.—A chronic malady characterized by large amounts of urine of low specific gravity. It was described by Willis (1674), who first differentiated between diabetes mellitus and insipidus (without taste, *i. e.*, non-saccharine). Eichhorst observed 7 cases in 35,942 cases (0.02 per cent.), Futcher 7 in 403,535 (0.001 per cent.) and 55 occurred in 113,600 in the Berlin Charité (0.048 per cent.).

Etiology.—Two groups of cases are observed: (*a*) the *idiopathic* (nervous or vasomotor) and (*b*) the *symptomatic,* which occur in (i) *brain disease,* especially basal syphilis and less often tumors of the medulla and the floor of the fourth ventricle, cerebral traumatism, basilar meningitis, apoplexy, hydrocephalus and lesions of the middle cerebellar lobe or corp. trapezoides of the pons. It is possible that the kidneys may be at fault, losing their power to concentrate the urine. (ii) It may result from *abdominal* diseases, aneurysms, tumors or tuberculous peritonitis. (iii) Insolation, diabetic ancestry (Trousseau), malnutrition (Ralfe) with tuberculous, syphilitic or gouty antecedents, acute infections and alcoholism are numbered among its possible causes. Most cases occur in *males.* In Strauss' collection of 85 cases, 10 per cent. were under five years old, 14 per cent. under ten and 45 per cent. were between ten and twenty-five years of age; according to Jacobi, 25 per cent. occur under ten years. Weil collected 36 cases among individuals of four generations, all of whom enjoyed good health. Rare congenital cases are recorded.

Symptoms.—The onset is usually gradual, though abrupt symptoms have followed fright. (*a*) The *urine* is *greatly increased;* a secretion of 20 to 40 pints daily is not rare and even one of 90 pints is on record. More urine is passed at night than by day. It is said that less urine is excreted than water ingested. It is passed rapidly after ingestion of water (tachyuria). Its specific gravity ranges between 1,000.5 or 1,001 and 1,005. It is pale and the total solids are usually normal, though sometimes the urea, chlorides and phosphates are increased, being washed out from the tissue. Albumin, sugar and inosite are very uncommon. (*b*) *Polydipsia* is secondary, though perhaps the earliest symptom noticed. In one case, a physician with brain syphilis, 2 gallons of water were drunk each night between 10 P. M. and 7 A. M. (*c*) The *skin* is dry; carbuncles are rare. The saliva is decreased. The appetite is usually not increased, though phenomenal polyphagia marks some cases. Nervousness, headache, irritability, increased knee jerks, impotence and severe lumbar pain radiating into the legs, are quite frequent. Nutrition is often good in the idiopathic cases and no unusual metabolic findings are noted; in the symptomatic variety the nutrition depends on the causal disease; in children growth may be retarded. The temperature is low and chilling occurs readily. Aside from causal lesions, there is no constant autopsy finding except hypertrophy of the kidneys from increased activity, and sometimes dilatation of the bladder and renal pelvis.

Diagnosis.—Diagnosis is easy. Differentiation is required (*a*) from *diabetes mellitus,* with which it has no common symptoms except polyuria and polydipsia; there is no glycosuria, acidosis, complicating gangrene, carbuncles or neuritis; (*b*) from *interstitial nephritis,* in which the specific gravity is higher, the urine is less abundant, is albuminous and contains casts, and which is associated with cardiovascular, retinal and uræmic manifestations; (*c*) from *primary polydipsia* with secondary polyuria, which is exceedingly rare; and (*d*) from *transitory polyuria,* which is distinguished by its course alone, as well as by its obvious etiology, as recent fever, hysteria, resorption of massive exudates, excessive medicinal diuresis and transient urethral obstruction with sudden release of urine.

Prognosis.—The prognosis is obviously more favorable in idiopathic forms, although cerebral syphilis doubtless causes many obscure cases. Less than one-half of the patients recover, though death results less frequently from the disease itself (save cerebral or abdominal tumors) than from intercurrent infections, notably pulmonary tuberculosis.

Treatment.—Aside from antisyphilitic therapy, drugs are of little use. Iodides benefit some cases in which there is no reason to suspect syphilis other than the therapeutic results. Opium may be useful, as in the saccharine form, and large doses are often well tolerated. Valerian (℥j of the root, t. i. d.), ergot (fluidextract ♏xx, t. i. d.), sodium bromide (℥ss, t. i. d.) and acetanilide (gr. v to x, t. i. d.) are recommended. Strychnia has clearly helped some cases. Talquist considers a vegetable more efficacious than a meat diet. Posner advocates chewing gum, which apparently decreases the polyuria and polydipsia.

GOUT.

Definition.—An obscure metabolic disease, characterized by acute periodic or chronic inflammation of the joints with uratic deposits.

Mentioned by Hippocrates, gout was clearly described by Thos. Sydenham (1683), who suffered from the affection for forty years. Murray Forbes (1792) and Wallaston (1797) recognized the uratic nature of the joint deposits.

Etiology.—1. The *actual cause* is unknown. In 1848 Garrod advanced the hypothesis that uric acid accumulated periodically (before the gouty seizure) in the blood. His, Vogel and Magnus Levy and others deny Garrod's statement and maintain that uric acid in gouty subjects is increased all the time; and that the urine contains less uric acid before and more during and after the seizure. Pfeiffer holds that the uric acid is usually decreased, and that it is really less a question of its amount than of its insolubility or form. Again the idea is advanced that uric acid is not produced in excess, but is retained in the body because of periodic attacks of renal insufficiency. Whether the real metabolic change concerns the uric acid, the alloxuric or purin bodies is yet undetermined.

2. *Extrinsic, predisposing* factors. (*a*) *Heredity* is an apparent

factor in 60 per cent.; transmission by the father is more common than by the mother and the younger rather than the older children suffer from hereditary influences. (*b*) *Mode of life;* lack of exercise, over-eating, an almost exclusively meat diet and alcoholism are potent factors. The old verse read that "Wine was the father, Eating the mother, and Venus the midwife" of gout. Though often true that gout affects the well-to-do (arthritis divitum) and arthritis deformans the poor (arthritis pauperum), gout is not uncommon among the lower classes. As alcoholism is a widespread scourge, it would seem that the heavy ales and beers of England and Germany are more injurious (in this respect) than whisky and light wine. Gout may occur in temperate individuals. Obesity, diabetes, arteriosclerosis and calculous tendencies are concomitant or coördinate rather than causal conditions. (*c*) *Lead poisoning* is observed in 15 per cent. of cases, particularly in England, and its relation to gout is puzzling, though saturnine nephritis may explain it. (*d*) *Men* are particularly prone to gout (1 to 13 or 25). (*e*) *Age.* Gout begins between thirty and forty years in 38 per cent. and thirty and fifty in 60 per cent. Much less than 1 per cent. of cases begins before the seventeenth year; Gairdner observed one case in a nursling. (*f*) England leads in frequency of its occurrence. In the United States it is rare, though Futcher shows that the cases in Baltimore and London hospitals stood as 2 to 3; again, relatively few cases seek hospital treatment.

Symptoms.—These may be considered under two captions, (1) acute, and (2) irregular, chronic and visceral gout.

1. *Acute* (regular, sthenic or) *typical* gout usually appears without prodromes, though an attack is sometimes heralded by digestive disorder (pyrosis, gastralgia, constipation), "cerebral congestion" (insomnia, vertigo, tinnitus, irritability, depression), paræsthesia, cramps or pain in the muscles, dark lateritious urine or other vague symptoms. The *paroxysm is remarkably characteristic.* (*a*) *Local findings:* The patient is awakened one to three hours after midnight by an agonizing pain in the metatarsophalangeal joint of the large toe (*podagra*). The pain is described as crushing by a vise or like burning by a hot iron. Localization in the toe is so frequent (95 per cent., Garrod, or 73 per cent., Scudmore) as to be practically diagnostic of gout. The skin over it becomes hot, red, tense, slightly œdematous and exquisitely sensitive. Serum exudes into the joint. The pain abates toward morning, but the inflammation increases. Each early morning sees a repetition of the pain for three to five or less often for six to seven days; the paroxysmal pain and the local findings then gradually subside by lysis; in two-thirds of the cases, the skin desquamates over the joint. Later attacks may involve the thumb, knee and other joints, though less commonly than in chronic irregular gout (*v. i.*). The local symptoms vary in intensity; as a rule the shorter the attack the severer are the symptoms. The *pathological changes* probably develop in this sequence: (i) local stasis (inflammation), (ii) necrosis and (iii) uratic deposit; the

ordinary urates (biurate of sodium in conjunction with sodium phosphate) do not cause such local reaction, but the biurate alone, even in most dilute solutions (0.004), produces inflammation (Pfeiffer); acute residual swellings (acute gouty tophi) consist of this biurate which has been precipitated by the carbonates of the blood and tissues. Convalescence is complete in ten to fourteen days, often with greatly improved spirits and health after the gouty attack. During the seizure (*b*) *constitutional symptoms* are not absent. The temperature rises to 100.5° or 101°, rarely 102°. The pulse is usually slow and hard, sometimes arhythmic and seldom exceeds 100. The heart may palpitate. Furring of the tongue, slight leukocytosis, anorexia, nausea, vomiting, epigastric cramping and eructations are not uncommon. The urine is dark, usually decreased, acid and higher than normal in specific gravity; the difference of opinion regarding uric acid has been mentioned under etiology; Bain and Futcher find a close parallelism between the uric and phosphoric acids, both being derived from disintegration of nuclein; they both fall below normal in the quiescent period and increase during the attack. Garrod's test for urates in the blood, exudates or transudates, consists of allowing it to clot, adding 10 drops of 30 per cent. acetic acid to ʒij of the serum and obtaining the crystals after twenty-four hours.

Subsequent acute seizures may sometimes be averted by diet and exercise, but they are likely to recur, notably in the spring and fall. Excesses in alcohol or in eating may precipitate acute gout; indeed in one case it would occur within fifteen minutes from drinking a glass of champagne; trauma, exertion or excitement may favor relapse.

Retrocedent or *suppressed gout* was once thought to develop when an acute paroxysm abated "too rapidly"; many symptoms attributed to it are doubtless due to coincident arteriosclerosis or uræmia (*e. g.,* gastralgia, vomiting, cardiac pain, dyspnœa, delirium, convulsions, apoplexy, coma and death).

2. *Chronic, irregular, atypical gout* generally follows acute attacks which increase in frequency, decrease in acuity and show slight tendency toward resolution. Pfeiffer claims that gout deposits (*tophi*) cannot occur without acute attacks. In some cases it appears to begin as chronic gout, particularly in the aged, the cachectic and in women with a gouty family history, all of whom lack reactive power (atonic or asthenic gout). (*a*) *Local findings:* there are less pain, fever and redness but greater and more permanent swelling than in the acute form, until the condition eventually becomes fixed and chronic; the "attacks" may be most rudimentary and remissions may alternate with exacerbations. The *lower extremities* and hands are most often affected; it may also develop in the thumb (*chiagra*), knee (*gonagra*), elbow (*pechiagra*) and spine (*rhachisagra*), but the shoulder is very seldom and the hip almost never invaded. In some cases its moving from joint to joint (*urarthritis multiplex seu vaga*) may cause confusion with acute rheumatism. Uratic deposits (*tophi arthritici*) form in the cartilage and later in the capsule and liga-

ments, and the swelling never recedes essentially. These prominences and the attendant fibrosis lead to deformation, subluxation, crackling on movement, ankylosis and contractures. Sometimes fluctuation is noted, especially in the big toe. The proximal joints of the fingers may point to the radial and the others to the ulnar side. Tophi may rupture, evacuating yellow-white urates, though incompletely, as they are partly diffused through the tissues; these gouty ulcers heal tardily, if at all, for their basis is fibrous and but slightly granulating tissue. Tophi occur also on the ear (25 per cent.), bursæ, tendons, aponeuroses (which may explain some cases of Dupuytren's palmar contracture), and even in the skin, eyelids, nose, larynx, penis or scrotum. (*b*) *Visceral gout* includes a curious mixture of real gouty complications and incongruous doubtful symptoms ("gouty disposition," *arthritisme, herpétisme* and the so-called "uric acid diathesis").

Complications.—*Renal complications* are the most important visceral complications and sometimes dominate the clinical picture. In 1,440 cases, Garrod observed albuminuria in 26.5 per cent., more often in advanced than in early cases. Its exact relation to gout is hard to define; it may follow the metabolic changes, or, according to Ebstein, may possibly constitute a primary gout. At first albuminuria may be considered toxic, marantic, congestive or arteriosclerotic, but later the renal, cardiovascular and retinal findings of interstitial nephritis develop with all its accidents and complications; uratic deposits occur in the intertubular tissue and sometimes in the secreting tissue of the cortex and medulla, in 15 per cent. of cases (Moore). Uratic deposits (calculi) are not uncommon in the renal pelvis; hæmaturia, oxaluria, pyelitis and cystitis may also occur.

Arteriosclerosis of the ordinary type is promoted by the individual's mode of life, by metabolic changes, alcoholism, plumbism and nephritis; it develops early and, with nephritis, causes most of the so-called visceral symptoms, as the *cerebrospinal* (headache, vertigo, brain congestion, epileptiform and apoplectiform attacks, encephalomalacia), the *circulatory* (cardiac hypertrophy, myocarditis, angina pectoris, atheromatous valvular lesions, phlebosclerosis), the *respiratory* symptoms (stasis, bronchitis, asthma) and retinitis.

Eczema (18 per cent.), scleritis and episcleritis (with small circumscribed points of inflammation and sometimes semimicroscopic tophi), neuralgia or less often neuritis, lumbago, sciatica and pain in the brachial plexus, burning and psoriasis-like patches on the tongue, digestive symptoms (*v.* PRODROMES) and burning in the eyeballs are probably often gouty. On the other hand, the "gouty disposition"—in some descriptions—consists of very vague symptoms; on analysis neurotic, paroxysmal (calculous colic, migraine or asthma) and other conditions which are common in persons not suffering from gout, account for many so-called visceral gouty manifestations.

Diagnosis.—(*a*) *Acute gout* is most typical in its etiology, history and localization, with acute, circumscribed redness. In a few cases (urarthritis multiplex), a diagnosis of acute articular rheumatism

may be made; fibrous rheumatic nodes may be confused with tophi, but if the possibility of gout is recognized there is usually no error. (*b*) *Chronic gout* often has a history of acute attacks, usually involves the extremities and distal joints of the hands and feet, is marked by unequal distention and thickening of the soft parts and is often known by its association with early arteriosclerosis, contracted kidney, obesity, renal calculus and diabetes. The *x*-rays cannot determine the diagnosis (see ARTHRITIS DEFORMANS, DIAGNOSIS). Visceral gout is an entity only when it develops in a patient undoubtedly gouty.

Prognosis.—Gouty patients may live to an old age, but the recurrence every two months to two years gives a tendency to chronic gout, of which the issues are (*a*) uræmia, (*b*) arteriosclerotic complications, as coronary disease or apoplexy, (*c*) terminal infections, as pericarditis, pneumonia and pleurisy, which may be induced by the (*d*) gouty marasmus or cachexia. In general the outlook is better in frank gout than in irregular gout and better in cases which develop after forty years of age.

Treatment.—1. THE ACUTE PAROXYSM.—(*a*) Absolute *rest* is indicated. (*b*) For *pain,* the use of *colchicum* is empirical (Storck, 1763); some believe that it increases the elimination of uric acid, though the claim that it enables the tissues to break down and dispose of the uric acid seems more reasonable; nevertheless, in gout it relieves pain and inflammation and acts as a cholagogue; ʒiss of the vinum colchici radicis (not seminis) should be given daily. It must be administered with care in nephritic and marantic subjects. If long used, a tolerance to its action is acquired. It should be stopped when pain is alleviated and it does not act well when diarrhœa intervenes, which interferes with its diuretic effect. (Full therapeutic doses produce a slow pulse, nausea, vomiting, pain and purging; inversely to its action on the bowels there are diaphoresis and diuresis. It is eliminated by the kidneys, bowels and skin. After toxic doses, great nausea, retching, serous and finally hemorrhagic purging, great pain, headache, vertigo, prostration, paralysis of the peripheral sensory nerves, motor part of the cord and finally of the respiratory centre (the usual cause of death) develop; ecchymoses of the gastro-intestinal tract with swelling may be seen at the postmortem examination.)

If colchicum produces no effect, *sodium salicylate* should be given, gr. xv every two hours (ʒj to ij daily); it usually relieves the pain and swelling and probably aids in solution of the uric acid; its early effect is greatest and it usually fails after the second or third day. Finally, *acetphenetidinum* (gr. viij to x) and morphine are indicated in severe paroxysms. In prolonged attacks, potassium iodide gr. v (given with care in nephritis) and wine of colchicum ♏x are useful. (*c*) The *diet,* according to Pfeiffer, should consist of foods which "release uric acid in a free form," as meal soups, rice and other carbohydrates; meat and eggs should be withheld (though given in chronic forms and in the intervals between attacks). Plain or acid water, as Seltzer (not alkaline water as Vichy) should be given

freely; alcohol and lemonade must be forbidden. If laxatives are indicated, calomel, rhubarb or castor oil should be used. (*d*) *Local measures* should be largely avoided lest added necrosis and tophi develop, especially vesicants, the use of the ice-bag and massage; warm applications of 50 per cent. alcohol or lead water (tinctura opii, liquor plumbi subacetatis, āā 3ij and water ℥j) may be used.

2. The Interval Between Attacks.—(*a*) Regulation of diet is the cardinal indication. Moderation in the quantity is more important than the quality of the food. *Quantity:* a full diet increases the uric acid, taxes resorption which is usually impaired and favors acid fermentations which engage the diphosphates and carbonates of the food necessary for the formation of soluble uric acid salts; three medium-sized meals a day are allowed. *Quality:* (i) *alcohol* should be absolutely forbidden; (ii) *water* may be given freely, little with the meals but large amounts on an empty stomach. (iii) Moderate amounts of *fat* are allowed. (iv) *Fruits and vegetables* increase diuresis and their malates and citrates help to dissolve the uric salts. (v) *Meats* are allowed (though there are various opinions on this subject). The urea they produce seems to dissolve uric acid which the meat, if given in moderation, does not increase. Meat is essential, particularly in marantic cases. There is no difference between the effect of light and dark meats; those rich in nuclein (sweet-breads, liver and kidneys) must be avoided. (vi) *Carbohydrates* should be largely *decreased;* this rule is empirical, though it appears that following their ingestion the uric acid (as compared with the urea) increases nearly two-fold. Milk should be avoided, for its lactic acid is injurious. (*b*) *Exercise* is indispensable, but prolonged or fatiguing effort, hurry and worry are to be avoided. (*c*) *Medicinal treatment* is rather secondary. Alkalies produce an increased output of uric acid, but they operate only by preventing decomposition of the neutral (bi-) sodium phosphate (the chief uric solvent). The kind of alkali used is not essential; lithium carbonate is inferior to *sodium carbonate* or *bicarbonate,* which acts first by promoting the formation of bisodium phosphate and second by neutralizing the acid formed by fermentation; it may be given (in doses of gr. x upward) for a long period of time, and has no effect on the blood; but the twenty-four-hour collection of urine should not become alkaline (lest alkaline deposits develop in the renal pelvis). Mineral waters merely operate in the same way, as those of Saratoga and Bedford Springs in this country, Bath and Buxton in England, Aix-les-Baines and Contrexville in France, and Fachingen, Carlsbad, Homburg, Wildbad and Teplitz. Luff advises the use of guaiacum resin, gr. v to x, t. i. d. Piperazin, lysidin, urotropin, urosin and sidonal are much inferior to the simple sodium salt.

3. Chronic Gout.—This is difficult to treat. For general dietetic, hygienic and medicinal measures, see above. Colchicum wine, ♏x, and potassium iodide, gr. v, may relieve the joints, the gouty asthma, neuralgia and eczema. Tonics, principally arsenic, are indicated in

marantic forms. Bier's stasis may benefit. Tophi should not be operated on; local measures are useless, save gentle massage, dry heat, moist applications or salicylate salves (ac. salicylic. 4, lanolin 40, and oleum terebinthinæ 1 part), though all treatment generally fails.

ARTHRITIS DEFORMANS AND CHRONIC RHEUMATISM.

Excluding gouty, tuberculous, syphilitic, gonorrhœal and hæmophilic affections, there remains a group of articular affections of which the chief are arthritis deformans and the so-called chronic rheumatism. Several totally different affections are gathered under these names. Their classification is most unsatisfactory.

Arthritis deformans is a progressive or at least chronic affection, marked by changes in the synovial sac, cartilages and bone, causing functional disturbance and usually deformity in the articulations, usually symmetrical and polyarticular.

Etiology.—(*a*) The predisposing factors are (i) *heredity,* which is causative in 13 per cent. (Garrod); (ii) *age;* 55 per cent. of cases occur between twenty and fifty years of age; 5 per cent. under twenty years and 40 per cent. over fifty years of age (Garrod); (iii) *sex;* though Stewart and McCrae consider that the sexes are equally involved, Garrod observed 82 per cent. in women; (iv) fatigue, cold, dampness, poor hygiene or trauma may be predisposing factors. (*b*) *The immediate cause* is unknown. There are many theories: (*a*) the abandoned *neuropathic* theory (Mitchell) is that it results from a cord lesion; (*b*) the *infective* theory is based upon finding bacteria in the joints, enlarged spleen and lymph glands and its occasional development after infections; (*c*) the *rheumatic* theory, according to which it is a chronic rheumatism; Garrod and Bannatyne find few heart lesions; Pribrâm finds one-fifth as many as in rheumatism.

Pathology.—Whether the synovia or cartilages are first involved is undetermined; the *x*-rays do not solve the question; practitioners, who see cases early, hold that the serous sac is first affected and surgeons find the cartilage most often diseased. The synovial villi hypertrophy and the cartilage slightly fibrillates, cleaves and finally atrophies. The bone becomes thin in some places and tuberous in others from periosteal proliferation (Haygarth's nodosities); the joint surfaces become eroded and in part grow together.

1. Diffuse Progressive Type.—(*a*) The *acute form* is less common. There is some fever, rise in the pulse rate and depression and the joints become red and swollen, resembling in some ways acute articular rheumatism, but differing from it in that arthritis deformans is less fugitive, involves the small joints more frequently, is attended by less sweating and seldom by heart complications, does not react to salicylates and runs a longer course. In some cases it has even resembled typhoid, because the fever and splenic enlargement

lasted for some time before the joints became affected. (*b*) The more frequent *chronic form* develops insidiously, with uncomfortable sensations in the joints, pain or tearing nocturnal pains about the joints and vasomotor skin symptoms. It begins most often in the small joints of the hands, with or without swelling, and remits and recurs. The hands often sweat. The fingers become fusiform from joint changes, capsular thickening and collateral œdema which separate the fingers from each other. The œdema often disappears, showing more clearly the bone and the capsule thickening. The cartilages become uneven and crackle on movement, loosened villi may become free bodies and the adjacent bursæ and tendons may become swollen. The fingers deviate and become angular from flattening of the saddle-shaped epiphyses and muscular contractures (now extensor and again flexor in type). Extension to other joints occurs; the elbow becomes flexed, the shoulder often crackles and becomes fixed; the large toe is bent out and lies under the other toes, which are often extended at their first and flexed at their two distal articulations; the ankle is

FIG. 48.

Rheumatoid arthritis. (Musser.)

often abducted; the knee is swollen, shapeless and flexed from effusion or contracture; in a few cases the hip, jaw and spine are involved.

Motion is limited by the swelling, muscular contractures, articular fixation by interlocking extracapsular nodosities and capsular induration. The contiguous muscles atrophy from disuse, local inflammatory invasion or reflex influence on the cord. The skin may become puffy, glossy or atrophic. The reflexes are increased. Neuritis is not uncommon. When the vertebræ were involved, Braun found nervous symptoms in 88 per cent., as weak muscles, occipital neuralgia, involvement of the brachial plexus and anæsthesia. The heart was not affected in any of Garrod's 500 cases. Anæmia, episcleritis and other complications are uncommon. The majority of patients do not improve or recover and recurrence of the disease is very common. In some cases the process is arrested. Aside from atheroma and nephritis few intercurrent diseases develop.

2. Partial or Monarticular Type.—Pathologically this type resembles the form described, but differs from it in that it presents more profound changes and is limited to one or few articulations. There is the same capsular thickening and cartilaginous fibrillation; the cartilage often softens, ossifies or hypertrophies; the bone rarefies or becomes cystic and soft in some places, with flattening of the bone heads and widening of the sockets, particularly in the shoulder and hip; in other places bony overgrowths form, known as Haygarth's nodes or a new socket may develop; the bone may eburnate and the joint may be either smooth or ridged. As a rule the atrophic prevail over the hypertrophic changes. They resemble the extreme senile change (*morbus coxæ senilis*); trauma or a nervous basis is considered causal; it occurs in old persons and largely in males. Clinically, compared with the general progressive type, it is single or at least rarely symmetrical, occurs in the proximal large joints (hip and shoulder), and is less often attended by subluxation. The elbow, spine and smaller joints are less involved.

3. Vertebral Type.—Strümpell (1885) named this type *chronic ankylosing spondylitis;* vertebral arthritis deformans occurs with ankylosis of the spine, shoulders and hips. Marie (1898) made it a separate type (*spondylose rhizomyélique*), characterized by complete vertebral ankylosis, scoliosis, ankylosis of shoulders and hips, exemption of all other joints, incidence almost exclusively in males, and its appearance in the young immediately after bony development has ceased. Von Bechterew (1892) described a similar type due to heredity, trauma or meningeal inflammation and characterized by paresis of the muscles of the neck, trunk and extremities, some atrophy of the back and shoulder, involvement of the spinal nerves (hyperæsthesia, pain and paræsthesia) and an ascending degeneration in the cord. These are probably but subtypes of arthritis deformans.

4. Juvenile Type of Still.—(*a*) The ordinary progressive (adult) form may be observed, running an unfavorable course, and often with almost universal ankylosis; in some cases the great toes are lengthened. (*b*) *"Still's type"* occurs in poor children largely; 70 per cent. of cases are girls; 30 per cent. occur between the second and seventh years, 32 per cent. between the seventh and thirteenth years, and 20 per cent. between the thirteenth and seventeenth years. The onset is often subacute. The multiple arthritis affects chiefly the extracapsular tissues and there is seldom crepitus. The spleen is always enlarged and the lymph glands near the affected joints are hard and large. Anæmia and retarded development are common.

5. Heberden's Nodes.—Bony nodes develop on the sides of the end phalanges near the joints. They begin between thirty and forty years, but are seen chiefly at about sixty years of age. At first soft and sometimes painful and red, they become hard, indolent and as large as a pea. Their growth is more often intermittent than continuous. Disturbance in function is not uncommon, but only in extreme cases is deformity observed, as flexion of the end phalanges into

the palm or deviation toward the radial side. Involvement of the large joints is rather uncommon, whence the prognosis is favorable. Some consider them gouty, though Heberden dissented strongly.

Diagnosis.—(*a*) In *gout,* the history, localization, fine (not coarse) crackling in the knee-joint and radial articulation of the elbow, greater spontaneous pain, infrequent generalization and symmetry,

FIG. 49.

Heberden's nodes. (Musser.)

the uratic deposits and tophi in the ear are characteristic. (*b*) *Acute rheumatism* (*v. s.* ACUTE FORM). (*c*) *Multiple tuberculous arthritis* may cause diagnostic difficulty, particularly when it occurs in children, for pulmonary tuberculosis may complicate arthritis deformans; but the tuberculous hip and knee involvement, the fusiform fingers, the hectic fever, involvement of the glands and serous membranes are generally distinctive. Poncet has recently called attention to an ankylosing tuberculous pseudorheumatism, which may cause confusion (v. TUBERCULOSIS). (*d*) *Syphilis* (*q. v.*) of the joints occurs particularly in persons between four and fifteen years of age and in about 40 per cent. of hereditary forms; the epiphyses are irregularly deformed. (*e*) *Hæmophilic* arthritis is distinguished by the unmistakable family history and its three stages, hæmarthros, panarthritis and adhesions, erosions, deformity and ankylosis. (*f*) *Chronic articular infections* (chronic pseudorheumatism), especially subchronic gonorrhœal arthritis (*q. v.*), begin acutely, usually affect the knees first, then possibly after months the ankles, hands (in women particularly) and sternoclavicular and other joints; similar chronic arthritides may follow scarlatina, diphtheria, pneumonia and grippe. (*g*) *Nervous arthropathies* present much the same pathological findings; (i) the arthropathies of *tabes* (*q. v.*) occur rapidly and early, without pain, tenderness or other inflammatory evidence; are attended by much effusion, are atrophic or flail-like rather than hypertrophic, in 80 per cent. involve the lower extremities and are accompanied by other signs of tabes. (ii) In *syringomyelia* (*q. v.*) the effusions are great, painless, in 80 per cent. involve the upper

extremities and are associated with the syringomyelic dissociated anæsthesia and panaritia. (*h*) *"Chronic rheumatism"* may rarely follow acute rheumatism, in which the changes involve less the bone, cartilage and serous sacs than the capsule which becomes fibrous. The cartilage may become somewhat fibrillated and fibrous, but is seldom hypertrophied or destroyed. The vascular bands become callous (Jaccoud's fibrous rheumatism) and ankylosis may result. Some maintain that ankylosis by connective tissue and the joint obliteration differentiate fibrous rheumatism from arthritis deformans, but clinical differentiation is often impossible.

Treatment.—Treatment is notoriously futile and arrest of the process results rather from spontaneous exhaustion of the disease than from therapeutic efforts. (*a*) *"Rheumatic" influences,* as exposure to cold and dampness, overheating while at work and resting in damp garments are to be carefully avoided. Woolen underwear should be worn. General hydrotherapeutic measures are illy borne. A warm climate benefits especially those cases resembling (or including) chronic fibrous rheumatism, as Mexico, West Virginia and Arkansas. (*b*) *Diet* is not of such importance as in gout; indeed, liberal feeding is strongly indicated. (*c*) In *acute exacerbations* rest and coal-tar derivatives are indicated; salicylates are generally useless save in really secondary rheumatism. (*d*) *Passive movements* and *massage* relieve stiffness and muscular contractures. Massage reduces the œdema and adhesions. In some cases forcible pressure, as with the thumb, will cause absorption of the fresh osteoplastic nodes; it may be easily overdone, and once every day or two on the same area is sufficient. Cautious active exercise is also essential. (*e*) *Extension* by pulley weights sometimes produces wonderful results, according to Hoffa, in lessening pain and exostosis, though the author has never observed any essential results. (*f*) *Local measures* may be of some use. The *x-rays* and strong electric lights may afford temporary alleviation from pain but do not seem to affect the basic change; fibrous rheumatism is more benefited than arthritis deformans. The same may be said of local applications of warm water. *Hot air* causes hyperæmia of the parts and is sometimes of real benefit; cages of wire, asbestos and felt are devised to enclose the joint, leaving around it an air space; the temperature is gradually raised by a lamp placed under a connecting elbow to 200 or 250° F. and is gradually lowered. Bier's method of *venous hyperæmia,* used in tuberculous joints, is also advocated; the Esmarch constrictor above the joint should produce swelling but not pain. *"Firing"* the skin with the Paquelin is beneficial in lighter grades of deforming arthritis and in fibrous rheumatism. Encouraged by the results in one case in which injections of iodoform and olive oil were given, the author used it in six other cases, and either without benefit or with great increase of pain and swelling. *Operation;* resection may be considered in nonprogressive, monarticular forms, but the general poor health and age of the patient rather contra-indicate it. (*g*) *Internal medication,* as cod-liver oil,

arsenic, thyroid extract and syrup of iodide of iron may help a few cases. Luff uses guaiacol carbonate; the initial dose of five grains, t. i. d., is increased two grains every week till a single dose of twenty grains is given.

RICKETS.

Definition.—A metabolic affection of early infancy, characterized chiefly by hyperæmia and deficient calcification of the growing bones. The disease was first well described by Glisson (1650). The name is derived from an old English word meaning "twisted," and Glisson suggested the Greek "rhachitis" (vertebral inflammation) because the spine was often involved, and the term sounded like "rickets."

Etiology.—Its etiology is obscure. All theories advanced, such as deficient absorption of lime salts, infection or reduced activity of the thymus, are insufficient. (*a*) The *diet* is of prime importance; proprietary foods, condensed milk, cow's milk, excess of carbohydrates, prolonged lactation or nursing during pregnancy are probable factors. Cheadle considered that a deficiency in animal fats and proteids was causative; after 20 litters of lion cubs had been lost from rickets in the London Zoological Gardens, Bland Sutton found that a diet of milk, powdered bone, meat and cod-liver oil prevented rickets. (*b*) *Unsanitary surroundings* may be causal, as lack of fresh air and sunlight, crowded quarters and kindred factors observed largely among the poor classes in large cities, particularly in Europe. Among nurslings Kassowitz (Vienna) observed it in 89 per cent. and Joucousky (St. Petersburg) in 90 per cent.; in children's clinics abroad, Marfan (Paris) found rickets in 50 per cent.; in Holland, London, Christiania, Naples and Rio Janeiro about the same figures prevail (50 to 80 per cent.); in the United States it is most frequent among negroes and Italians. Hereditary syphilis is a predisposing factor. (*c*) *Age.* In more than 600 cases Baginsky did not observe it in children under three months of age; his figures show 7 per cent. in children between three and six months, 28 per cent. between six and twelve months, 35 per cent. between one and one and a half years, and 18 per cent. between one and a half and two years (*e. g.*, 88 per cent. of cases occurred between three months and two years of age). The occurrence of congenital rickets (apart from achondroplasia, see DIAGNOSIS OF BARLOW'S DISEASE) is usually denied but there is some authoritative testimony for it. Rickets which develops later, up to puberty, is called *rhachitis tarda.*

Symptoms.—An experienced observer may diagnosticate the gradual onset from the prodromes, as the irregular or tardy dentition; restlessness, irritability, insomnia and throwing off of the bedclothes; sweating about the head and neck; bronchitis; digestive disturbances, coated tongue, anorexia, malnutrition, meteorism or diarrhœa; immobility, the child crying when moved or touched and general hyperæsthesia. However, a positive diagnosis is not justifiable until the bone symptoms develop.

1. Bone Symptoms.—(*a*) The *skull* usually shows the earliest changes, generally in the first year of life; the bone changes (*v. i.*) are most marked in the skull and thorax. The *face appears small* and the *head large.* Nodes of bone appear on the frontal and parietal regions, which with the occipital flattening produce the caput quadratum (*tête carée*); the head may be dolichocephalic. The anterior *fontanelle increases* forward. The fontanelle and sutures (which normally close about the fifteenth or eighteenth month) remain open, even until the fourth year, *i. e.,* there is lack of normal involution. The occiput softens (*craniotabes*) in 33 to 50 per cent., near the lambdoid suture; the occipital protuberance remains hard. Craniotabes may less frequently occur elsewhere, as in the frontal bones; besides the actual bone changes (*v. i.*), the pressure of the pillow and of the brain further its development. In extreme cases the peri- and endocranium actually touch, and in moderate degrees the skull feels "parchment-like." Though it is not absolutely pathognomonic of rickets, and though it occurs also in syphilis, craniotabes is rather characteristic when there are also the tender quadrate head, head sweating, thin occipital hair and blue scalp veins. Craniotabes may be an element in the general and local spasms which occur in rickets. The frequent *systolic murmur* heard over the greater fontanelle is also heard in other affections or in health; it seldom endures beyond the fifth year and is said to occur only in conjunction with a similar bruit in the carotids. The *upper jaw* becomes long and narrow and the lower jaw becomes hexagonal, probably from muscular traction on the soft bones. The eruption of the teeth is delayed until the second or even the third year; the second teeth are crowded, carious, curved and eroded. (*b*) In the *thorax,* the rhachitic "*rosary*" appears toward the end of the first or early in the second year; it is so-called from the bead-like succession of enlarged chondrocostal joints; they are enlarged epiphyses. The *chest* is altered toward the end of the second year; this is due to the softness of its bones, the traction exerted by the diaphragm, atmospheric pressure and sometimes by external influences, as holding of the child with a hand on the chest or coincident respiratory inflammation. Louis' angle is often prominent, and the sternum juts forward, causing "*chicken breast*" (pectus carinatum). There is also a depression outside of the costochondral junctions which extends outward and downward, and is accentuated by some eversion of the costal arch. The thoracic deformity promotes respiratory complications and dyspnœa, which in turn aggravate it. With the deformity of the chest we may group lumbar *kyphosis* which is arcuate and not angular, the thickened or bent clavicle and the thickening of the spine or edges of the scapula. (*c*) In the *extremities,* the *epiphyses,* particularly of the ulna, radius, tibia and fibula, *are enlarged;* this occurs about the end of the first year, at the same time as the rosary, craniotabes and wide fontanelle. The epiphyses, like the ribs and skull, are tender and painful. It may look as though there were "*double joints.*" Deformities may

develop from muscular traction or from the weight of the body; the arms may incline outward and forward; the forearms may become convex toward their extensor surfaces. The thighs sometimes become convex anteriorly and outwardly; the legs bow outward or less often forward, especially when early walking is encouraged. Spiral or other extreme deformities are less frequent. The gait is waddling. Greenstick or actual fractures sometimes occur. (*d*) The *flat rhachitic pelvis* develops by the weight of the body pushing the sacrum into the pelvis and the counter-pressure of the thighs narrowing the anterior pelvis, so that the pelvic aperture may become heart-shaped; the acetabula lie more anteriorly and the iliac crests flare outward. (*e*) *Pathology.* Normal bone grows in length as follows: there are two zones in the epiphyseal surface facing the shaft; (i) the *first zone* is nearest the shaft, is faintly yellow and measures ½ mm.; it is the zone of preliminary calcification, into which normally the bloodvessels grow and form medullary canals by absorbing small areas of bone, and in which osteoblasts appear and calcification develops. (In rickets the calcification is imperfect and irregular in this zone and the bloodvessels grow not merely into these calcified areas but beyond them, even into the cartilage area, and absorb what little new bone is formed.) (ii) The *second zone* lies outside the one described, is sharply demarked from it, is normally bluish, measures 1 to 2 mm. in thickness and is known as the hyperplastic zone, in which the cartilage cells divide and proliferate. (In rickets this zone becomes larger, proliferates irregularly and is invaded by bloodvessels and irregular foci of abortive calcification; the demarkation between the first and second zones is ill-defined.) Normal bones grow in thickness by bony increase beneath the periosteum. (In rickets this layer becomes several millimeters thicker, irregular and nodose.) Normal bone develops in the skull and face in the connective tissue. (In rickets the lime salts are either not deposited, are insufficiently deposited (craniotabes) or over-deposited as bosses (osteosclerosis).) The essential element of these rhachitic changes is *undue hyperæmia* and *irregular, deficient calcification;* which one is primary remains a disputed point; the bones may contain but 20 or 30 per cent. of the normal amount of lime salts.

2. General Symptoms.—(*a*) *Muscular weakness* is pronounced, as evidenced by impaired breathing, the tardy efforts to hold up the head and disinclination to walk. (*b*) *Nutrition* is usually *poor;* there is anæmia, sometimes with leukocytosis and often with emaciation; the irritability and sweats have been mentioned under prodromes. (*c*) *Nervous system.* Mentality is unimpaired. Rhachitic patients frequently grind their teeth. Spasmodic conditions are not infrequent, as generalized *convulsions, tetany* and *spasm* of the *glottis,* all of which will receive separate consideration. (*d*) Temperature is not symptomatic of rickets and always denotes complications. (*e*) *Digestive disturbances* are common; there may be anorexia, diarrhœa without pain or tenesmus, more commonly constipa-

tion and *distention* of the *abdomen* which can occur without any digestive disorder, from enlargement of the liver and spleen, and possibly from the thoracic changes. The amount of chalk in the fæces may be increased (Baginsky). The *spleen* is *enlarged* in 70 per cent., and, less often and less markedly, the liver. (*f*) Rickety children *catch cold easily* and bronchitis frequently leads to atelectasis and bronchopneumonia. (*g*) The heart may be luxated from the altered thoracic conformation, which also frequently causes hypertrophy of the right ventricle. (*h*) The urine remains normal. (*i*) Eczema is common.

Course, Complications and Prognosis.—(*a*) *The onset* is insidious and the clinical *course covers a year* or more. (*b*) *Complications* include but few of immediate danger, as bronchitis, pneumonia and spasm of the glottis which may result fatally. Later complications include the contracted rhachitic pelvis of obstetrical importance, kyphosis and pulmonary tuberculosis, which is found in 36 per cent. of the fatal cases, sometimes with abdominal complications (tabes mesaraica); its early diagnosis depends almost entirely upon auscultation, for percussion is peculiarly deceptive when the chest is deformed. (*c*) The prognosis is, with these exceptions, excellent as to life, and with care, as to deformity, for many protuberances and deformities may regress.

Diagnosis.—The diagnosis is based chiefly,—and then with certainty—upon the changes in the skull and skeleton. Errors are easily possible from precipitate diagnosis, based on the purely prodromal phenomena. (*a*) *Barlow's disease* (*q. v.*), which is misnamed acute rickets, has no connection with it. (*b*) *Syphilis* predisposes to rickets but is otherwise distinct and separate; it usually develops at a time (first to sixth month) when rickets is most uncommon and is usually stamped by other syphilitic stigmata, as cutaneous and mucous syphilides; when it invades the epiphyses, the femur is usually its seat, and there may be, between the cartilage and bone, a wall-like, very tender, painful and usually doughy swelling which also is rather fusiform, compared with the rounder rhachitic nodes; epiphyseolysis with great dislocation of the fragments, may occur in syphilis, but never in rickets; in doubtful cases an *ex juvantibus* diagnosis may be made. (*c*) *Vertebral tuberculosis* (*q. v.*) is easily distinguished; the rhachitic kyphosis is arcuate, not angular; other symptoms of either disease are almost invariably present. (*d*) *Hydrocephalus* (*q. v.*).

Treatment.—1. Hygiene.—Fresh air and sunlight produce wonderful results, particularly in the country, mountains or at the sea-side.

2. Dietetic.—In nurslings (who seldom develop the disease) or in quite young babies, the use of a wet-nurse may entirely cure the disease. In older children it may be less a question of food than of digestion, which may require pepsin, taka-diastase, nux vomica or hypophosphites.

3. Medicinal.—No specific remedy is known, but the results produced by *phosphorus* make it more important than any other drug.

Under its use the child becomes easier and brighter; the nervous symptoms abate, as the insomnia, convulsions, tetany or laryngospasm; the osseous symptoms are improved, bone is deposited and dentition occurs, though less obviously in clinical experience than in animal experiments in which it seems certain that small doses stimulate bone formation directly and rapidly. Phosphorus itself is given in doses of gr. $\frac{1}{200}$ to $\frac{1}{100}$ in pill form or as the ol. phosphoratum (1 per cent. solution) ♏j, t. i. d.; the oil or emulsion should be freshly prepared, for both deteriorate readily on exposure to light. (Its toxic symptoms are malaise, fever, thirst, anorexia and weakness; vomiting of food, mucus, bile, later blood and sometimes phosphorescent material, may occur; there may be abdominal pain, diarrhœa or constipation, phosphorescent, clay-colored or bloody fæces; jaundice may develop; the liver at first may be large, but becomes small; there may be cholæmia; albumin, bile, casts, leucin and tyrosin in the urine and hemorrhages from the uterus, abortion or miscarriage and bleeding from other surfaces.) *Cod-liver oil* was recommended by Bretonneau in 1827; it carries out Cheadle's theory that animal fats and proteids are essential and conforms with Sutton's experience (*v. s.*). If it is well tolerated by the stomach, the general level of health may be elevated by its use; if not, cream and butter should be given. *Iron* (syr. ferri iodid.) is a good tonic. Calcium salts are now less used than formerly, as it has been proven that a sufficient quantity is present in the food and is absorbed.

4. Orthopedic.—During the florid stage the child should be kept quiet upon a firm mattress and off his feet. Later, corrective measures are indicated for the kyphosis and deformity of the limbs. Mikulicz holds that the apparently monosymptomatic genu valgum adolescentium is only a rhachitis tarda, and that genu valgum and varum should always be treated with rest, cod-liver oil and phosphorus.

OBESITY.

Etiology.—Obesity (adiposity, corpulence or polysarcia adiposa) is favored (*a*) by *heredity;* it frequently occurs in entire families; congenital cases are known; (*b*) by *mode of life;* lack of exercise, over-eating and alcoholic indulgence are potent factors. (*c*) The *menopause* is frequently attended by increase in weight. (*d*) *Various diseases* may cause it, as those of the nervous or osseous system, *making exercise impossible;* and gout, diabetes, various anæmias, chronic valvular disease and emphysema, which act rather by preventing adequate exercise than by deficient oxygenation or from the condition of the blood. Adipose tissue may develop from tissue albumin.

Symptoms.—The general *habitus* requires no description. Fat develops in the subcutaneous tissues, in parts where it is normally found, as in the omentum and around the kidney and it may cause fatty infiltration and degeneration of all internal organs excepting the brain and spleen; even the blood may contain fat (lipæmia).

1. Cardiac Symptoms.—The heart is embarrassed by increased resistance to the capillary flow, by the fatty infiltration and degeneration of the myocardium, and by fat accumulation in the chest wall, omentum and mediastinum. The pulse is more often frequent than slow, though cases of bradycardia are noticed (see Fatty Heart). Cardiac hypertrophy may occur. Œdema is not uncommon.

2. Respiratory Symptoms.—The breath sounds and heart tones are faint and thoracic percussion is difficult because of the subcutaneous fat. *Bronchitis* is common and probably results from the ready sweats and consequent chilling. Dyspnœa may occur on exertion only, or may develop from pulmonary œdema.

3. Digestive Symptoms.—Digestive disturbances may develop. The large liver is less frequently fatty than congested and hypertrophic. Constipation and hemorrhoids are frequent.

4. The Urine.—The urine may be scant in comparison with the fluids ingested; coincident oxaluria, lipuria, lithuria, albuminuria or glycosuria may be noticed. Actual impotence is not uncommon, as well as mechanical obstacles to coitus.

Two other groups of symptoms mark extreme obesity, *viz.:* (5) *lessened physiological resistance* to infections, as typhoid or pneumonia and to surgical measures or phlebotomy, and (6) such *associated affections* as diabetes, gout, calculous disease and less often contracted kidney, arteriosclerosis and apoplexy. Somnolence may be marked.

Treatment.—Treatment is (*a*) that of the fundamental condition, as gout or diabetes, and (*b*) *dietetic.* Various "cures" have been formulated which are enumerated because they possess more than historical interest.—(i) *Banting's cure.* Banting, a merchant, was treated so successfully by Wm. Harvey for obesity that he addressed a "Letter to the Public on Obesity" (1863). This diet was largely *albuminous* and consisted of fish, meat, eggs, tea without sugar or milk, vegetables, fruit, some red wine and toast without butter. This diet may cause indigestion, nervousness, insomnia, palpitation or gout. (ii) *Ebstein,* himself a sufferer, proposed (1872) a diet with larger proportions of *fat;* his diet consisted of meat, fish, eggs, tea, leguminous vegetables, fruit, fats and butter, but restricted the carbohydrates; digestive disorders may also occur under this system. (*c*) *Oertel* allowed *more carbohydrates,* limited the fats and fluids and insisted upon sweats and graduated exercises. From the following it will be seen that all these methods are "starvation cures," with restriction of water in Oertel's method. The average man requires 118 gm. (4$\frac{3}{3}$) of nitrogen, 56 gm. (2$\frac{3}{3}$) of fat and 500 gm. (17$\frac{3}{3}$) of carbohydrates (Voigt and Pettenkofer), equaling 3,054 calories.

The diet of Banting contains 172 gm. (5.7$\frac{3}{3}$) of nitrogen, 8 gm. (2$\frac{3}{3}$) of fat, and 81 gm. (2.7$\frac{3}{3}$) of carbohydrates, equaling 1,112 calories.

The diet of Ebstein contains 102 gm. (2.3$\frac{3}{3}$) of nitrogen, 85 gm. (2$\frac{3}{3}$) of fat, and 47 gm. (1.6$\frac{3}{3}$) of carbohydrates, equaling 1,401 calories.

The diet of Oertel contains 183 gm. (6℥) of nitrogen, 38 gm. (1.3℥) of fat, and 143 gm. (4.8℥) of carbohydrates, equaling 1,690 calories. (A calorie is the temperature required to raise 1,000 gm. of water, 1° C.; a gram of albumin equal 3.2 calories, of fat, 8.4, and of carbohydrates 3.8 calories.)

Banting's method supplies to the body about 33 per cent. of the heat and energy normally required; Ebstein's less and Oertel's slightly more than 50 per cent. The lesson taught then is *moderation in eating* (*v.* Gout). In any reduction-cure the patient may suffer from breaking down of the body proteids, which causes the weakness that is experienced. In all cases the total amount of nitrogen in the urine, which constitutes 90 per cent. of the nitrogen excreted, should be watched; if more is excreted than ingested the nitrogenous food should be increased to obviate weakness, both muscular and cardiac. The diet then should consist, as in gout, of *moderation in all foods,* with *restriction of the carbohydrates,* whose easy reduction into water and carbonic acid spares the tissue waste. The *fluid* ingested should be measured and with due individualization *reduced* to 2 or 3 pints daily. *Alcohol* should be *forbidden*, as it is a carbohydrate. Beer contains not only 3 to 5 per cent. of alcohol, but sometimes double this percentage of soluble starch and dextrine.

(*c*) *Active and graduated exercise* is indispensable, except in marked cardiac disease. It should be regular, induce sweating and bring all muscles into play, but should not cause exhaustion. Passive movements (*v.* Fatty Heart, Myocarditis and Dilatation), and massage are indicated when active exercise is contra-indicated.

(*d*) The patient should *sleep not more than seven hours* and should bathe daily to avoid skin complications and to promote perspiration. Thyroid extract is not to be taken, except perhaps under special indications, for it breaks down the albumins rather than the fats, often excites the heart, sometimes induces glycosuria and is wholly superfluous (with proper dietetics and exercise).

ADIPOSIS DOLOROSA OR DERCUM'S DISEASE.

This dystrophy was described by Dercum (1892), as a "disorder characterized by unequal symmetrical deposits of fatty masses in various parts of the body, preceded or attended by pain." There were only 47 reported cases up to 1907, most of which were females. It usually develops in middle life, chiefly after the fortieth year, though the author has a case under treatment, aged thirty-five years.

Pathology.—Its pathology is not yet developed, as but five autopsies have been made (1903). In the cases of Dercum and Burr an interstitial neuritis was found, which is likewise noted in the fatty tumors which have been removed during life. It is interesting to note that symmetrical lipomatosis has been observed in multiple neuritis. In Dercum's and Burr's cases the thyroid gland was atrophied. Adenocarcinoma and gliosis of the hypophysis have been reported.

Symptoms.—*Two cardinal clinical features* are observed, as the term adiposis dolorosa indicates; (1) the *obesity* is either diffuse or may be localized fatty tumors or sometimes both in combination; lipomata occur on the trunk and proximal parts of the extremities, but never on the face, forearms, hands, legs or feet. Indeed the thinness of the wrists and legs contrasts sharply with the massive body, thighs and upper arms. The fatty tumors range from the size of a pea to large dimensions. The pendulous abdominal fat may hang down like an apron. In one of the author's cases the localized abdominal fat was disposed in the shape of a circular life-buoy, sagging down over the thighs. In another it almost resembled œdema or myxœdema, save for its circumscribed distribution and facial exemption.

2. The *pains* may be neuralgic or paræsthetic. The trunk is often tender.

Subcutaneous hemorrhages, hæmatemesis, epistaxis and metrorrhagia are not uncommon. The patients are often nervous, perhaps hysterical. Asthenia is common and subjects often lack initiative and readily tire on exertion. In the later stages epilepsy has developed and death from asthenia, psychosis, nephritis or intercurrent affections is recorded. A number of cases, including one of the author, have improved under administration of thyroid extract.

SECTION IX.

DISEASES OF THE NERVOUS SYSTEM.

DISEASES OF THE BRAIN.

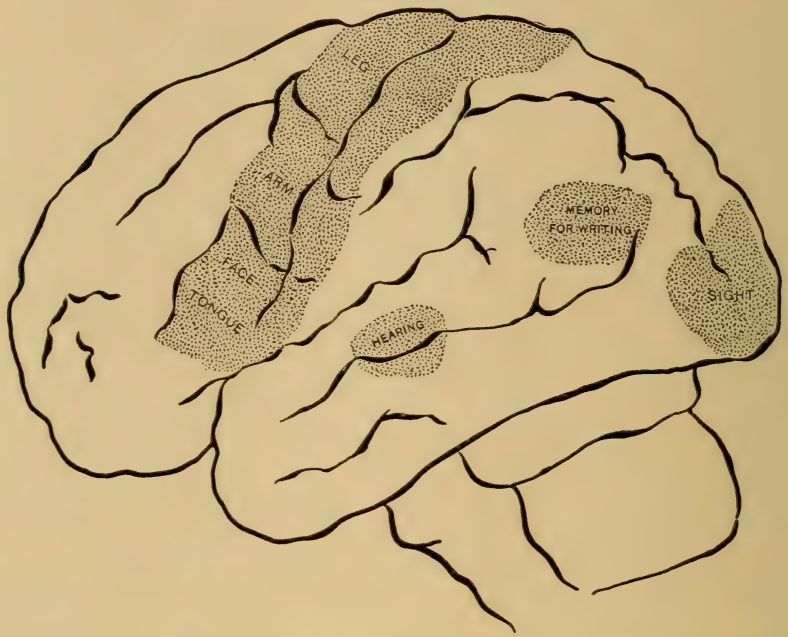
CEREBRAL LOCALIZATION.

The anatomy and physiology of the brain are most important in topical diagnosis. Autopsy findings in carefully studied cases, animal experimentation and the constantly increasing surgical intervention have developed not merely the *nature* of lesions, but also important results regarding their *localization.* The old doctrine of Flourens that all parts of the brain were equally important met its first contradiction in Broca's discovery (1861) that motor aphasia was due to disease of the third left frontal convolution. In 1870 Fritsch and Hitzig discovered that stimulation of certain areas, now known as the motor cortex, invariably resulted in contraction of definite muscles of the opposite side of the body. Since then Bevor, Horsley, Meynert, Jackson, Flechsig, Ferrier, Munk, Glotz, Nothnagel, Charcot, Wernicke and others have contributed to the development of brain localization, although much still remains unclear and tentative.

I. The Motor Cortex.—This embraces chiefly the central convolutions on either side of the fissure of Rolando (the ascending frontal and ascending parietal lobes and anterior two-thirds of the superior parietal lobe), the foot of the three frontal convolutions, especially the lower, and on the inner surface of the hemisphere, the paracentral lobule. In this area lie the large pyramidal cells which Betz discovered were the origin of the motor or pyramidal tracts. The centres are not sharply marked, but run somewhat into adjacent areas, especially those with physiological associations. The *leg centre* occupies the upper quarter of the ascending frontal and ascending parietal convolutions and part of the superior parietal lobe (all adjacent to the longitudinal fissure) and passes over to the inner aspect of the hemisphere to the paracentral lobule, which is the centre for the thigh, pelvis and gluteal muscles (Fig. 51). A lesion in the falx cerebri, or dura near the longitudinal fissure, may, in rare cases, involve both paracentral leg centres and produce cerebral paraplegia, although most paraplegias are spinal in origin. The *arm centre* is the largest, covers the middle two quarters of the central convolutions, lies below and probably somewhat overlaps the leg centre. In Fig. 52 it is seen that there are certain general as well as separate centres

for the thumb and fingers. The *head centre* is below that of the arm, covering the lowest quarter of the central convolutions (*i. e.,* the entire operculum), the upper part of the Sylvian fissure and the posterior part of the lowest frontal convolution (and possibly the first and second frontal gyri). The *face centre* lies directly beneath the arm centre, and is mostly located on the anterior ascending convolution, the upper facial component lying higher than the lower facial centres. The eye centre of the facial is bilateral and probably con-

FIG. 50.



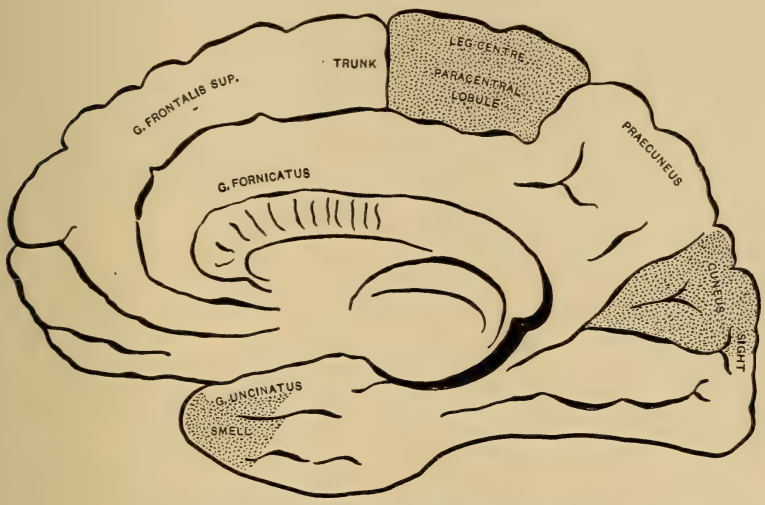
General distribution of centres in cerebral localization; outer surface of brain.

sists of several foci. The mouth apparently has several centres. The *motor speech area* is principally in the lower left frontal gyrus and is probably the chief centre for chewing. The *trunk centre* is less clearly defined; it probably lies in the posterior part of the first frontal convolution and also on its median aspect, just anterior to the paracentral lobule. The more complicated the function, the larger is the centre (as for the tongue, fingers, mouth) and the more unilateral the localization in the brain. Muscles used rhythmically or in pairs are supplied to some extent from both hemispheres, as the muscles of the forehead and of respiration, swallowing, chewing, vocal cords and to some degree of the legs and trunk. Protrusion of the tongue to one side has a monolateral centre, though its protrusion in the median line and its withdrawal depend on both hemispheres.

Total lasting hemiplegia of the opposite side of the body, with sensory disturbance, aphasia and intellectual disturbance, results

from complete destruction of the entire motor cortex of one side. Cortical hemiplegia is less common than *monoplegia,* which results from partial cortical foci. Monoplegia (paralysis of one member,

FIG. 51.

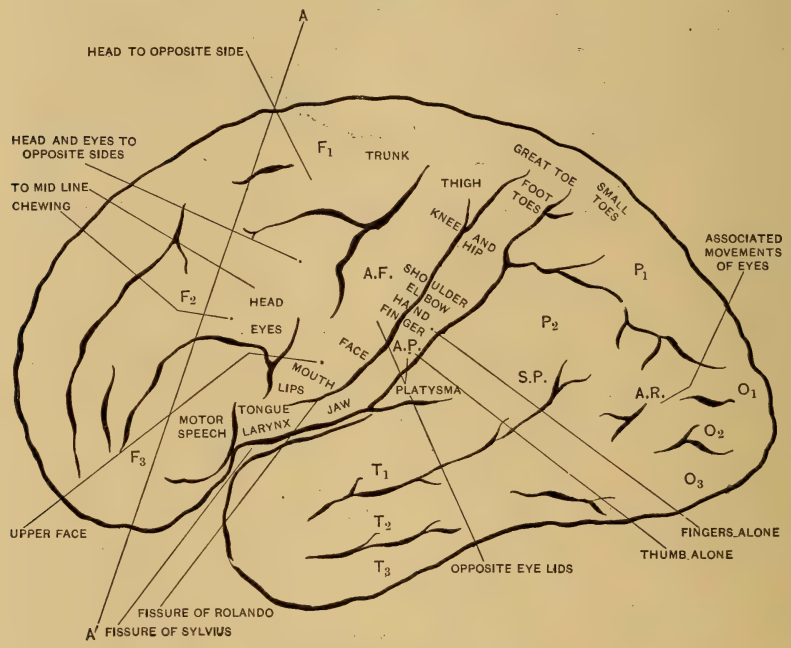


Cerebral localization; inner surface of hemisphere.

as of the arm alone) always suggests a cortical lesion. The paralyzed limbs are flaccid and later undergo contractures. *Pure monoplegias* of cortical origin are rare, although the arm or leg may be paralyzed alone from a small softening, hemorrhage, cyst or tumor, or from traumatism. Crural (leg) monoplegia is rare (few instances being reported by French writers) and is due largely to vascular lesions in the paracentral lobule (anterior cerebral artery). Some sensory disturbance may be noted. Pure brachial (arm) monoplegia may result from vascular lesions, small tumors or trauma; complex movements, as writing, are most involved. The finest differentiation or selection, as of the thumb alone, fingers or wrist, is noted in this form. Sensory disturbance is common (*v. i.*). Pure facial monoplegia is unknown, although a faciolingual form with motor aphasia is observed. *Associated monoplegia* is more usual. It consists most commonly of paralysis or paresis (partial paralysis) of the arm and face (brachiofacial monoplegia, passing under this name, although strictly not a monoplegia). Next in frequency is the arm-and-leg type (brachiocrural) in which two contiguous centres are involved. A leg-and-face type never occurs, because their centres lie too far apart. A lesion involving one cortical area after another speaks for cortical localization. Cortical paralyses are not attended by the reaction of degeneration. In cortical lesions with paralysis, twitchings or convulsive movements are common and, from their resemblance to epileptic convulsions, are called "cortical, partial or

Jacksonian epilepsy"; 50 per cent. of cases are small cortical tumors or cysts; the lesion partly destroys the motor cortex (paralysis) and at the same time causes irritation (convulsions) precisely as contractions are produced in animals by electrical stimulation of the cortex; they are mainly (*a*) mechanical, as irritation or increased intracranial tension (rapidly growing tumors), or (*b*) due to a vascular affection (hemorrhage, embolism or inflammation). Stationary lesions rarely produce Jacksonian epilepsy unless they are located in the cortex. The spasms may involve but one part, as the arm (*monospasm*), which is exclusively a sign of cortical irritation, or they may pass from one centre to others. They always commence in one centre *locally* and extend in a *fixed order*. If the arm-region is the seat of

FIG. 52.



Cerebral localization in detail.

F_1, F_2, F_3, first, second and third frontal convolutions. AF, ascending frontal convolution and AP, ascending parietal convolution. SP, supramarginal and AR, angular convolution. O_1, O_2, O_3, first, second and third occipital convolutions. T_1, T_2, T_3, first, second and third temporal convolutions. The explanation of line AA is given under INTERNAL CAPSULE.

a tumor the Jacksonian epilepsy flexes the fingers, moves the thumb, then the forearm, arm and shoulder and then passes down the cortex region (see Fig. 50 for leg, arm, face centres), so that it involves the face, mouth and finally the leg. If the lesion is in the face area, the various facial muscles contract and the eye deviates, then the next area above the arm centre participates, and finally the higher leg centre is implicated. That is, the spasm never skips from the face

to the leg centre, but it involves the arm first. If the leg area is involved, the spasms pass consecutively down to the arm and then to the face, *i. e.,* they never skip the arm, nor involve the face before the arm is convulsed.

The spasms are often preceded by a tingling in the part, are mostly clonic, or clonic broken by tonic spasms, and *consciousness is very often maintained.* Unconsciousness may intervene when the fits reach the other side of the body, when they are very frequent or when the lesion is deep and below the cortex. The opposite side is involved through the commissural fibers of the corpus callosum; "bilateral representation in both hemispheres, which saves certain groups of muscles in hemiplegia, necessitates their involvement in severe spasms" (Gowers). Jacksonian differs from regular epilepsy in that no cry is observed, the commencement is local and more gradual and consciousness, if lost, disappears gradually, not precipitately. The convulsions may precede or accompany the paralysis. The seizure lasts several seconds to a minute or two and is followed by some vertigo, nausea, stupor or disturbance of speech; if spasms occur without paralysis, paresis in the involved parts follows, due to transient exhaustion of the cortex. Jacksonian epilepsy in its lightest forms may be sensory.

Regarding *sensory localization* in the cortex opinions vary and our knowledge is far less exact. Munk holds that the sensory and motor areas are the same, while Charcot, Nothnagel and Ferrier hold that the sensory areas are separate (*v. i.*). Some *vasomotor disturbance* is common in cortical lesions, and Landois and Eulenberg have demonstrated a vasomotor centre here.

Sequences of disease in the motor centres depend on the nature and extent of the lesion. After total lesions of the central convolutions, the hands improve very little in adults; other members improve, to some extent at least, through assumption of function by the sound side of the brain; in young individuals the sound pyramidal tracts in the cord may hypertrophy to even double their original size. The motor fibers running from the pyramidal cells of the cortex to the anterior horns of the spinal cord, constituting the *upper motor neuron,* undergo descending degeneration, that is, degeneration in the direction of the motor impulse.

II. Cortex of the Parietal Lobes.—Our knowledge of the function of this cortex is less definite. (*a*) Lesions of the *first lobe* (P_1 Fig. 52, the præcuneus in Fig. 51), supplied by the anterior cerebral artery, cause disturbance in motility of the leg, with altered muscle sense, "mind blindness," inability to read (alexia) and, according to some authorities, hemianopsia. (*b*) Disease of the *supramarginal convolution* (S.P. Fig. 52), also involving the motor cortex, produces greater disturbance of sensation than when the lesion is confined to the motor centres; muscular sense is disturbed, usually with some motor paralysis or paresis. Three cases are reported of disturbance of muscle sense without paresis. Muscle sense is disturbed in 23 per

cent. of all cases of cerebral hemiplegia, though in more than 50 per cent. of cerebral paralyses, no special sensory disturbance is noted. Disturbance in muscle sense indicates that a cerebral lesion is probably located in the cortex, if indirect pressure can be excluded. There may be loss of the stereognostic sense, whereby the patient fails, when his eyes are closed, to recognize the geometric form of solid objects, as a key or ball. Cortical incoördination (ataxia) sometimes results, probably on account of disturbance in the association fibers. It differs from the tabetic and cerebellar ataxias in that the muscular strength is usually diminished and the finer movements are impossible, though the coarser movements may sometimes be carried out. Cortical hemianæsthesia is not frequent. (*c*) Ptosis and paralysis of the orbicularis palpebrarum are said to result from lesions of the *gyrus angularis* (A.R. Fig. 52) and, according to Monakow, inability to read (alexia), optic aphasia and "mind blindness." Wernicke correctly diagnosticated softening in this lobe by conjugate deviation of the eyes and head as the only symptom. Disease of the posterior inferior part probably causes crossed amblyopia.

III. The Cortex of the Occipital Lobes.—This is undoubtedly the *visual centre.* Its exact seat is somewhat disputed, but the weight of evidence shows that it is in the cuneus (Fig. 52) and the first occipital lobe. A lesion of the occipital cortex produces (*a*) bilateral *homonymous hemiopia* (hemianopsia) (for details of which and the following see AFFECTIONS OF THE OPTIC NERVE). (*b*) *Hemichromatopsia*, an homonymous color blindness, especially for green and red, in which the color perceptions are confused rather than lost; the lesion in such cases is superficial and anterior; (*c*) *total cortical blindness,* if the lesion is bilateral, or both optic radiations are destroyed. (*d*) *"Mind blindness"* results from a lesion in the anterior outer surface of the *left* occipital region, especially the second occipital lobe or from disease of the gyrus angularis. The visual memory is lost; things actually seen are misinterpreted and the patient confuses persons and things. (*e*) *Alexia*, inability to read, also called "word blindness," wherein the patient can read but fails to understand the letters or words he sees and reads. His memory for words is lost. It is due to destruction of the subcortical association fibers in the second occipital lobe (von Monakow) and the gyrus angularis; it most commonly follows vascular disease. (*f*) *Optic aphasia* results from foci in the left occipital lobes (and also in the angular and supramarginal convolutions) and consists of misinterpretation of visual impressions, so that the patient miswrites and misnames objects seen, though able to name them when he feels them. (*g*) *Crossed amblyopia* sometimes occurs.

IV. The Cortex of the Temporal Lobes (T_1, T_2, T_3, Fig. 52).—The outer surface of the posterior half of the first (and second) temporal convolution is the *auditory centre;* it is said to be larger on the left side; disease in this cortex produces (*a*) *deafness* in the opposite ear; atrophy of this lobe is encountered in congenital deafness. Deaf-

ness is usually transitory, because fibers seemingly pass to both ears from each centre. If this lobe is destroyed on both sides, total deafness results. (*b*) *Conjugate deviation of the eyes* has been observed and is said to be a reflex from auditory impressions. (*c*) Disease of the first temporal lobe produces "*word deafness,*" also known as "mind deafness"; it is a loss of the comprehension of speech. Somewhat in front of this lobe it is supposed that musical memories are stored. In the *uncinate gyrus* (Fig. 51) the special sense of smell probably has its centre. Ferrier refers the general sensory centre to this lobe. The gyrus fornicatus (Fig. 51) is said to be the centre for *taste* and possibly for the *sense of pain.*

V. The Frontal Cortex and Aphasia.—Regarding the frontal lobes anterior to their participation in the motor cortex we have little definite knowledge. Profound disease may develop without clinical symptoms. They have long been thought to be the residence of (*a*) the *psychical functions,* especially for ethical and abstract thought (Meynert and Flechsig). "Defect in character" followed the passage of a large crowbar, 1½ inches thick and 3½ feet long, through both frontal hemispheres, in the famous crowbar case of Harlow. Psychical functions probably concern the frontal lobes, although the relative integrity of the entire brain is necessary for them. (*b*) Flechsig considers the frontal lobe the anterior *association centre,* the middle one being the cortex of the island of Reil and the posterior one the large area posterior to the motor cortex. (*c*) Bruns has observed ataxia similar to the cerebellar type, but this probably results either from pressure transmitted to the cerebellum or from involvement of the trunk centre. (*d*) *The motor speech area lies in the lower frontal convolution.* Its function and relations can be well considered in connection with *motor* and other varieties of *aphasia.* The history of aphasia marks the beginning of cerebral localization and three French writers have determined its motor centre. Bouillaud, in 1825, referred aphasia to the frontal lobes; Dax, in 1836, narrowed the localization to the left frontal region and the *motor centre* was finally confined to the posterior part of the *lower left frontal convolution* in 1861 by Broca, whose name is given to this convolution. The sensory element, necessary to all voluntary movements, was unknown until later, when Meynert and Wernicke discovered the auditory centre in the first temporal convolution; Wernicke localized "*word deafness*" in the same convolution of the left side and Naunyn and others emphasized the importance of the *gyrus angularis* and adjacent parts of the *occipital* lobes in cases of disturbance of writing, visual aphasia, etc.

Motor Aphasia.—The *motor speech* centre lies in Broca's lobe, the posterior part of the left third frontal convolution; it controls the motor centres next described and retains the impressions or memories necessary to their exercise. The corresponding lobe on the right side is the actual centre in left-handed persons and often assumes the functions of the left speech centres after disease of the latter, espe-

cially in children. Articulate speech depends upon the integrity of the lower part of the ascending frontal convolution, in which lie the centres for the mouth, lips, tongue, jaw and larynx. (*a*) These constitute the *cerebral mechanism* of speech, whose disturbance is designated motor aphasia. Motor fibers run from the cortex through the centrum ovale, internal capsule (lying in front of the pyramidal tracts), crus (inside the pyramidal tracts) to their nuclei in the pons and medulla, whence the peripheral bulbar nerves to the tongue and larynx take their origin. (*b*) These lower tracts constitute the *bulbar mechanism* of speech, disease in the components of which causes abolition of articulation (anarthria) or more often imperfect articulation (dysarthria). The elements of speech are correct in dysarthria, but articulation is imperfect, the labials, palatals or linguals being imperfectly delivered or consonants run together. To speak, the individual must have ideas, conceptions. To illustrate, a cortical lesion in Broca's convolution, as small as a hazel-nut, may produce motor aphasia, either absolute or partial; the patient may pronounce correctly some few words, possibly only under emotion or in certain relations, or he may pronounce words wrongly, although he recognizes his mistake. He comprehends what is said to him, for the lesion involves no special sense element. He can read writing and print, especially if educated, because "sight may stimulate images without intervention of the motor centre," but he cannot speak voluntarily nor repeat what is said to him; he cannot read aloud; as a rule he cannot write (agraphia), because impulses cannot pass to the hand centre. He may be able to copy (*v. i.* TABLE). The cortical is the usual form of motor aphasia.

A *subcortical lesion* in the centrum ovale may sever the fibers between Broca's convolution and the motor cortex for the tongue, lips, etc. The patient cannot speak, but the speech centre is normal. It is a rare lesion, known as pure motor aphasia (Lichtheim). The patient can write. It involves the commissural fibers of the corpus callosum to the right side and usually produces permanent aphasia, because the possibility of compensation by the right hemisphere is precluded; if the lesion is in the internal capsule the aphasia is transient, since compensation through the corpus callosum is established.

Sensory Aphasia.—In speech, as in every voluntary motion, sensation and sensory memories are necessary. The sensory relations of speech are *hearing, sight, taste* and, in the blind, *touch.*

Auditory Aphasia.—Hearing is most important and develops in the child long before speech. When a child begins to talk, or when one who has lost speech relearns it, the brain cortex, through an association of centres, learns to connect certain sounds or words with definite objects or conditions. The child hears through the auditory nerves which run from each side to both *auditory centres* in the *first temporal convolution* of either side. He hears mechanically, just as we may hear without understanding a foreign language, and learns to associate the sounds with objects, conditions or ideas, for which an *auditory*

speech centre in the *left first temporal convolution* (in right-handed persons) is developed. Here sounds coming from the two centres of hearing are remembered, associated and controlled. Remembering a sound he has heard but perhaps has never spoken, the child calls into play Broca's convolution, the cortical centres for the lips, tongue, etc., and speaks. Hearing is necessary to articulate speech, unless, as in deaf mutes, education replaces, by sight and touch, the associations usually acquired by hearing. Destruction of the auditory speech centre profoundly disturbs speech. The patient then hears by the auditory nerves and centres or reads by the visual apparatus, but does not understand what he hears or reads; this is known as *sensory (auditory) aphasia* or *word deafness.* It is rarely absolute and recovery is more common than in motor aphasia. The subject in speaking transposes words (paraphasia) but does not realize his mistakes, an important distinction from motor aphasia. Often he later becomes speechless. He cannot repeat words nor write from dictation. Voluntary revival of words is lost (Gowers). Voluntary writing is impossible, but copying of words is sometimes maintained.

Subcortical auditory aphasia or pure word deafness (the rarer form), results from disease *between* the auditory speech centres and the two centres for hearing. It must therefore be below the cortex in the white matter, hence called subcortical. Auditory impressions cannot reach the auditory speech centres of the patient, who cannot understand what is said, cannot repeat or write from dictation. As the auditory speech centre is normal, memory for words is retained and the patient can read, speak and write.

"Amnestic" aphasia may be a simple difficulty in recalling certain words in speaking or writing, or it may be very pronounced (word deafness). It is also poorly called conduction aphasia (Wernicke) and is due to interruption, probably in the island of Reil, of the fibers between the motor and sensory speech centres. Gowers states that it may result from partial recovery from motor or sensory aphasia.

Visual Aphasia.—*Sight* is very important in sensory speech. The child who has learned sounds (words) and is able to speak them, reads by associating the sounds heard and spoken, with words, letters or symbols seen by him. Memory of these *visual concepts* is stored in the *supramarginal* and *angular gyri.* Reading music, etc., is learned in the same way. While in most individuals mental processes are carried on by the memory of words, in rare instances visual memory may be more important. Destruction of the *angular* and *supramarginal* convolutions by causing a loss of the *visual memories* produces *alexia,* word blindness, an inability to read. In some cases the patient can read, but without understanding. Isolated alexia is always due to lesions in these convolutions. He usually cannot write (agraphia) and cannot copy, but can speak fairly well and understand what is spoken. If the optic centre in the occipital lobes is intact he cannot name objects after seeing them, since the visual memory centre is destroyed, but may recognize a watch by hearing it tick or

TABLE OF SIMPLE APHASIAS. (Church.)

VARIETY.	LESION LOCATED.	PRINCIPAL FEATURE.	UNDERSTANDS SPEECH.		EXPRESSES SPEECH.		REPEATS DICTATION.	COPIES WRITING.	WRITES FROM DICTATION.	INCIDENTAL CONDITIONS.
			Spoken.	Written.	Spoken.	Written.				
AUDITORY APHASIA.	Posterior thirds of the first and second left temporal gyri.	Word-deafness.	No.	Yes.	Paraphasia—jargon.	Paragraphia or agraphia.	No.	Yes.	No.	Amusia.
MOTOR APHASIA.	Foot of third left frontal gyre.	Dumbness.	Yes.	Partly.	Articulation impossible.	Exceptionally.	No.	Yes.	May do so.	Amimia or paramimia.
VISUAL APHASIA.	Angular gyre of left parietal lobe.	Word-blindness.	Yes.	No.	Yes.	No.	Yes.	Yes.	Partly, but can not then read it.	Homonymous hemianopsia, when the lesion is in the optic radiation; mind blindness, if the lesion is bilateral.
CONDUCTION APHASIA (Wernicke).	Island of Reil usually; must cut off connecting tract between auditory and motor word memory centres.	Paraphasia. Paragraphia.	Yes.	Yes.	Paraphasia.	Paragraphia.	Paraphasia.	Yes.	Paragraphia.	Mental confusion.

by feeling it. Lesions just below the visual memory centre, involving the afferent fibers, produce *pure subcortical word blindness, or alexia,* that is, inability to read, with hemianopsia and preservation of voluntary speech and writing. Word deafness and word blindness often co-exist. Sixteen cases of congenital word blindness were collected by Stephanson (1904).

Pure motor and sensory aphasias are less common than complicated forms. The diagrams employed to illustrate the mechanism of aphasia are hypothetical. The anatomical lesions, mostly arterial, are largely the result of softening, the entire sphere of speech being supplied by the artery of the fossa of Sylvius. Motor aphasia is likely to occur with disease of the motor cortex or cerebral pyramidal tracts, and the sensory form with disease of the optic fibers. Apoplexy, trauma and in children tuberculosis of the brain or meninges are also causative. Lasting aphasia in children is usually due to mental causes, since in early life the right frontal area usually takes up the function of Broca's convolution. Congenital aphasia may follow bilateral meningeal hemorrhage. Aphasia also occurs in idiocy in adults, in melancholia, migraine, convulsions and is especially apt to occur from functional causes in children, as from fever, fright, worms, etc. In adults the outlook is less favorable than in children. Partial recovery is possible in cases of hemiplegia and sometimes with care, reëducation may yield fair results. The table on page 908 is borrowed from Church's excellent work on *Nervous Diseases.*

VI. Centrum Ovale.—Focal symptoms may be entirely absent, if a lesion occur between the associative, commissural, motor or sensory fibers (corona radiata). When present, symptoms are usually those of (*a*) a cortical, or (*b*) an internal capsule lesion. A lesion must be (*a*) very near the cortex to produce either Jacksonian or general convulsions. A monoplegia, hemianopsia and subcortical aphasia may occur in this location. Aphasia from a lesion in the island of Reil may produce conduction aphasia. Psychical symptoms may result from interruption of the association fibers which run from one part to another of the same hemisphere. (Commissural fibers are those running from one hemisphere to the other, as through the corpus callosum, disease of which presents no pathognomonic sign.) (*b*) Internal capsule symptoms result in lesions low in the centrum ovale.

VII. The Internal Capsule.—It is compacted and consists of nearly all the motor, sensory and special sense fibers from the brain to the lower parts. The capsule is divided into an anterior limb, the knee and a posterior limb.

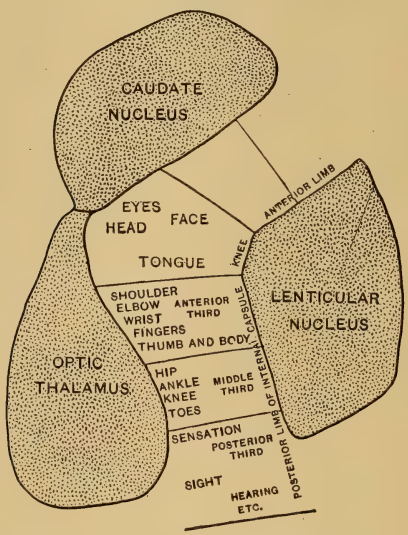
Anterior Limb.—Its function is not clear, although it contains fibers from the frontal cortex to the optic thalamus and fibers which, in lower horizontal sections, run into the knee and are found in the basal median bundle of the crus, whence they run to the pons nuclei for the eye, head, neck and probably also for the larynx, tongue and mouth. Lesions here are very rare.

Knee.—The knee (see Fig. 53) contains from before backward

fibers for opening of the eyes, associated movements of the eyes and head, the tongue and mouth; Horsley and Beevor have been able to stimulate them electrically, one by one. This segment contains the phonation fibers for speech, probably including those of the larynx.

Posterior Limb.—The posterior limb contains in its anterior third the fibers for the shoulder, elbow, wrist, fingers, thumb and body; in its middle third those for the hip, ankle, knee and toes; in its posterior third (Charcot's "sensory crossway"), those for sensation and the special senses. The number of fibers to a part depends less on the size of a muscle than on its development in fineness of movement; for instance, the fingers receive a relatively large number of fibers. If a ruler be placed anterior to the line (*AA*) in Fig. 52 and slowly moved to the right, preserving its parallelism, the cortical motor areas touched will approximately correspond with their order in the internal capsule. The most common sequence of lesions of the internal capsule is *cerebral hemiplegia* of the opposite side of the body. Though hemiplegia may result from lesions elsewhere in the motor tract, in the vast majority of cases, hemiplegia is the result of dis-

FIG. 53.



Localization in internal capsule.

ease of the internal capsule. If the internal capsule is destroyed, *total hemiplegia* results, paralysis of the arm, leg and lower part of the face (the upper part of which and the head escape because they have a bilateral supply from both hemispheres). The hypoglossus is but moderately involved and aphasia is usually temporary. With the hemiplegia are also associated hemianæsthesia, hemianopsia and unilateral disturbance of smell, taste and hearing, by lesion of the fibers ascending from the tegmentum; with the hemiplegia are observed

contractures, descending degeneration, decrease or absence of the skin reflexes, especially the cremasteric, and increase of the tendon reflexes. Monoplegia is rare, because the tracts are so compact that the face, for example, is rarely alone involved, though Duplay and Diday record isolated cases. It is possible that a *linear* lesion may involve the outer part of the capsule's knee, that is, it may involve the face and also the leg, leaving the arm free (Wernicke, Charcot, Kolisko). Hemianæsthesia (skin sensation and muscle sense) rarely exists without some paresis of the leg. Some tingling or, in incomplete lesions, some pain may result, even when the leg-arm areas are alone involved, for some sensory fibers course with the motor fibers. Hemi-tremor, -athetosis and -chorea result from lesions of the sensory area, usually associated with disease in or just below the optic thalamus. (The course of the sensory paths will be considered under LOCALIZATION IN THE SPINAL CORD.) Back of the general sensory tracts disease may injure the optic radiation (running between the occipital sight centre and the lateral geniculate body) or interrupt the auditory and other special sense fibers. Psychical disturbance, aphasia, alexia, agraphia and involvement of any cranial nerve are not symptomatic of disease of the internal capsule.

VIII. The Corpus Striatum.—This is composed of the caudate and lenticular nuclei. It is doubtful whether it is connected with the cortex. The lenticular nucleus is extensively connected with the superior peduncle of the cerebellum and the tegmentum of the crus. The caudate nucleus is connected with the internal capsule and with the crus; these fibers seem to end in the pons and cerebellum. An apoplexy in the corpus striatum probably causes rise of temperature; Nothnagel's law still holds that hemiplegia is permanent only when the internal capsule is directly injured, although indirect transient hemiplegia from simple pressure on the capsule may result from disease of the corpus striatum Landouzy reports a case of contralateral hemiathetosis from isolated disease of the lenticular nucleus.

IX. The Optic Thalamus.—This is surrounded by gray matter, continuous with that surrounding the third ventricle and central canal of the cord and is connected by fibers with all parts of the cerebral cortex, the tegmentum and superior cerebellar peduncle. Nothnagel (1879) was the first to describe its topical symptomatology. Sometimes disease in this locality is latent; again, the following findings are observed, alone or in combination: (*a*) *Contralateral hemianopsia* may develop from a lesion in the posterior part of the thalamus, the pulvinar (Gowers) or a lesion which also invades the lateral geniculate bodies (von Monakow). These areas are connected by the optic radiation with the optic centre. This hemianopsia is distinguished from that caused by a cortical lesion by the hemianopsic pupillary reaction and the absence of the marginal field of vision. (*b*) *Disturbance of the mimetic movements of expression* may develop (Nothnagel, Bechterew). The optic thalamus is the reflex centre of the involuntary movements of expression,

as weeping or laughing. A patient with hemiplegia from destruction of the left *internal capsule* cannot voluntarily move the lower right part of the face, but the paralyzed portion of the face moves when the patient laughs, for the centre is in the posterior part of the thalamus. On the other hand, in isolated disease of the optic thalamus the opposite side—indeed both sides of the face—can be voluntarily moved, since the facial nerve runs in the internal capsule, but the patient cannot laugh or weep. (*c*) *Sensory disturbances* may occur, such as hemianæsthesia, hemichorea, hemitremor, pain in the opposite arm (Edinger) or hemiataxia, but it is not absolutely certain in these cases that the sensory portion of the internal capsule was not involved. There may be vasomotor changes, atrophy and disturbance in the evacuation of the bladder and rectum. Von Monakow thinks that all the symptoms referred to the thalamus are probably referable to disease exterior to it.

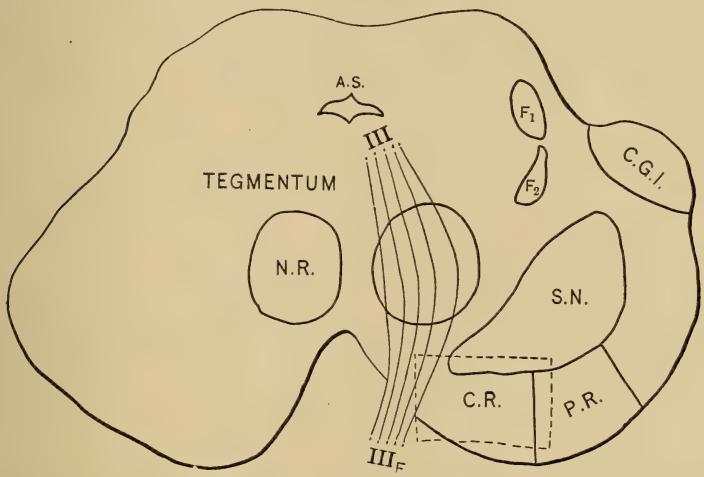
X. The Corpora Quadrigemina.—These are masses of gray matter whose fibers lie over the aqueduct of Sylvius and the tegmentum. There is a variance of opinion regarding their function. They are connected with the third nerve nucleus and some hold that they contain the centres for contraction of the heart and stomach.

Disease in the *anterior corpora quadrigemina* produces (*a*) *moderate involvement of sight;* slight disturbance has a greater localizing significance if there is no coincident optic neuritis. These bodies are connected with the external geniculate bodies and, by way of the internal capsule, with the optic radiation. (*b*) In acute lesions, narrowing of the *pupil* is suggestive. In old lesions, one or both pupils are wide and without reaction to light or accommodation. (*c*) *The eye muscles* may be partially paralyzed; this results in ptosis and upward movements of the eyes; these symptoms are not positively indicative of this localization, because the lesion is always so near the nucleus of the third nerve. Disease in the *posterior corpora quadrigemina* may result (*a*) in *ataxia,* according to Nothnagel, though others refer it to actual cerebellar disease, to connections with the cerebellum through the nucleus ruber or to disease in the fillet of the tegmentum; (*b*) in *trochlearis paralysis,* in which the eyes cannot be turned inward or outward; sometimes it results in abducens paralysis; (*c*) in *difficult mastication* from involvement of the descending branch of the quintus; (*d*) in contralateral *disturbance of hearing,* since the posterior corpora quadrigemina are connected with the fillet of the tegmentum and, by means of the "sensory crossway" of the internal capsule, with the hearing centre in the first temporal lobe.

XI. The Crus (Cerebral Peduncle).—Focal symptoms are motor or sensory. The *motor* fibers to the cranial nerves decussate higher than the pyramidal tracts which decussate in the medulla; the fibers to the third nerve cross in the upper crus, although some enter the nucleus of the same side. *Crossed paralysis* (hemiplegia alternans oculomotoria superior) is common in crus lesions. It consists of a

hemiplegia of the side of the body opposite to the lesion and a *paralysis of the third nerve on the same side* as the lesion, often known as Weber's paralysis, though first described by Gendrin. From Fig. 54 it is seen that a lesion involving the right pyramidal fibers may also catch those of the third nerve on the same side, giving a left-

FIG. 54.



Lesion in crus (shown by dotted lines), producing a superior alternating or crossed hemiplegia; the lesion involves the pyramidal tract (PR), the cranial nerves (tongue and face, CR) and the oculomotor nerve (III, F) which is paralyzed on the same side as the lesion, while PR and CR are paralyzed on the opposite side. AS, Aqueduct of Sylvius; III, nucleus of third nerve; CGI, Corp. geniculatum int.; F_1, upper part and F_2, lower part of fillet; NR, nucleus ruber; SN, substantia nigra.

sided hemiplegia (face, arm and leg) and right-sided oculomotor paralysis, which is peripheral and usually complete, with strabismus, ptosis, inability to look up or down and a wide reactionless pupil. The crossed or alternating hemiplegia is called superior because inferior alternating hemiplegia may occur in lesions of the pons. One must be sure that one lesion only exists. In tumors near the internal capsule which press on the third nerve, the same symptom complex may occur, recognized by noting whether the hemiplegia and paralysis of the third nerve occurred together (indicating a crus lesion) or whether the third nerve paralysis followed the hemiplegia (indicating a lesion near the internal capsule pressing secondarily on the oculomotorius).

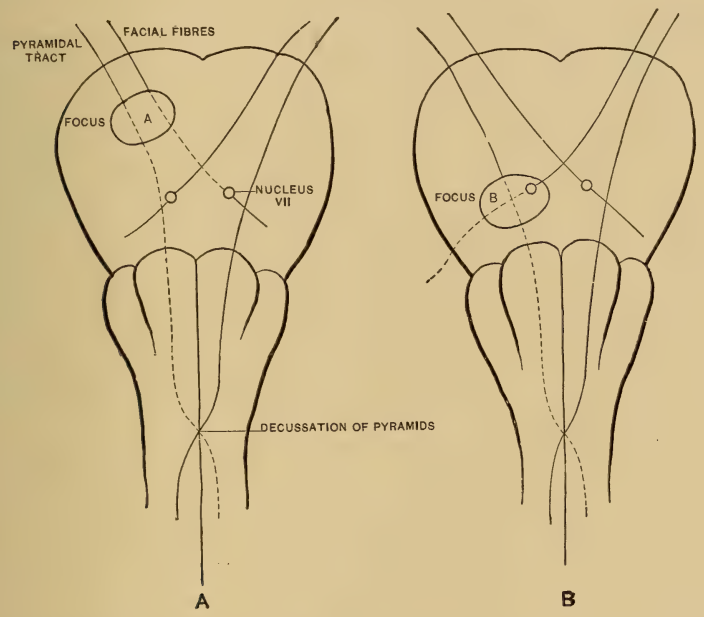
A lesion in the tegmentum of the crus may cause (*a*) *sensory disturbance,* as hemianæsthesia, which involves skin sensation and muscle sense, and of which over thirty cases are recorded; such a lesion is in the fillet; hypæsthesia (reduced sensation) is more common. (*b*) The *special senses* are sometimes involved, producing hemianopsia or poor hearing in the opposite ear. (*c*) *Ataxia* has been seen alone (6 cases) or together with other symptoms. It differs from the cerebellar type in that it is manifest only on motion.

Involvement of the red nucleus (which is connected with the cerebellum or with its arm) produces cerebellar ataxia. (*d*) *Oculomotor paresis* on the side of lesion is characteristic when combined with crossed hemiataxia, hemiplegia or hemianæsthesia. Fig. 54 shows how a tegmental focus can rarely miss the diffuse nuclei of the third nerve. If the lesion is in the lower part of the crus, the oculomotor paralysis is usually complete, but if in the tegmentum it is always partial, electing the pupil, the lid or the superior or internal rectus.

XII. The Pons.—Its structures are so closely arranged that sometimes both sides are implicated, thus producing bilateral hemiplegia, though a small lesion, as a long thin tumor, may give no localizing symptoms. Lesions of the pons (commonly tumors, hemorrhage, softening or abscess) usually produce a typical picture. From a lesion of the *upper pons, i. e.,* above the facial nucleus, a hemiplegia results like that produced by lesions of the internal capsule, which affects the face, arm and leg, all on the same side of the body. (See Fig. 55, focus A.) The facial paralysis is supranuclear, partial and with no reaction of degeneration. *In a focus in the lower pons* (focus B, Fig. 55), where the facial fibers have already crossed, the tongue, arm and leg are hemiplegic on the side opposite to the focus and the facial paralysis is on the side of the lesion; this crossed or alternating paralysis (hemiplegia alternans facialis) was first described by Millard (1856) and Gubler (1859). The facial paralysis is peripheral and nuclear, involves all branches and gives the reaction of degeneration. Sometimes there is also some slight paralysis of the face on the hemiplegic side from involvement of some fibers from the cortex. Other alternating paralyses are also observed, as *hemiplegia with crossed paralysis of the third, fourth, fifth and sixth nerves,* the paralysis of which is usually both central (nuclear) and peripheral. Hemiplegia may occur with conjugate paralysis or paresis of the lateral eye muscles, as the external rectus (abducens nerve) of one side, and the internal rectus (third nerve) of the other, due to a lesion just in front of the abducens centre, involving its fibers, or sometimes also including its nucleus. In the conjugate paralysis, the antagonist muscles pull the eyes to the other side and the *eyes look away from the side of the lesion* toward the paralyzed side. This is not to be confused with the "forced position" of the eyes due to irritation, known as conjugate deviation, in which the eyes look to the sound side of the body. In this form even the paralyzed internal rectus will functionate and converge when one eye is tested at a time. In some cases both internal recti and one abducens may be paralyzed, resulting in fixture of the eye in the median position; accommodation and convergence are preserved. Involvement of the *motor branch* of the fifth nerve produces difficult mastication. While alternating paralysis is the usual pons lesion, sometimes double hemiplegia occurs, or again, involvement of the third to seventh cranial nerves without hemiplegia. The *auditory nerve* is rarely involved, save in acute or very extensive lesions affecting the lateral lemniscus. Im-

plication of the cortical fibers of the hypoglossus produces impaired movement of the tongue (dysarthria), nasal speech and poor enunciation of consonants, though the vocal cords are normal. *Dysarthria and dysphagia,* due to disease of the cortical motor paths of the hypoglossus and vagus, are less common in lesions of the pons than are affections of their nuclei in diseases of the medulla oblongata. *Ataxia* may result from involvement of the fillet and possibly of the longitudinal bundle of the formatio reticularis and produces incoördina-

FIG. 55.



A illustrates a lesion in the upper pons, producing the usual hemiplegia (face, arm and leg paralyzed on the opposite side). B illustrates a lesion in the lower pons, producing alternating paralysis; all branches of the face (peripheral paralysis) and tongue on the side of lesion are paralyzed with paralysis of the contralateral arm and leg.

tion on voluntary movement only. *An alternating sensory paralysis* may occur, as (*a*) hemianæsthesia involving the muscle sense and cutaneous sensation of the extremities opposite the lesion, caused by disease of the tegmentum, and (*b*) an anæsthesia of the fifth nerve on the side of the lesion. *Irritative* symptoms sometimes develop, as pain, paræsthesia, painful convulsions, which usually occur in acute cases and are peculiar in their involvement of both arms or both legs, tonic and clonic spasms in the opposite arm and leg, contractures, paroxysms of coughing and trismus. The patient may "look toward the lesion" when the eye muscles are irritated. A tendency to fall toward the side on which the lesion is located is indicative of disease of the middle peduncle of the cerebellum. It is sometimes difficult to decide whether the focus is located in the *pons or in the medulla.*

Though the topical symptoms of the medulla oblongata are described under its individual diseases, it may be stated that *an alternating or crossed paralysis,* involving the third to seventh nerves inclusive, *indicates disease in the pons,* and that *aphonia, dysarthria and dysphagia* are symptomatic of *localization in the medulla,* especially when the hypoglossus is paralyzed, shows the reaction of degeneration and produces atrophy of the tongue. Glycosuria, albuminuria, disturbed respiration or heart action (by involvement of the vagus) and vomiting are other bulbar symptoms. Disease in the medulla may cause paralysis of both arms and legs or rarely of one arm and the opposite leg (hemiplegia cruciata).

XIII. The Cerebellum.—Physiology.—The functions of this organ are not fully known. Certain physiologists admit the possibility of some psychical function. Its *sensory relations* are important, because of its reception of numerous fibers from the spinal cord, including the direct cerebellar tract and the posterior median column of Goll. It is an "end organ" (Luciani) for these tracts, through which and its relations to the motor tract and the special senses, it is supposed to coördinate and *harmonize the movements of the body, to maintain equilibrium, conserve muscular strength* and *inhibit the contralateral cerebral motor cortex.* In other words, it associates (*a*) the sensory impressions ascending from the spinal cord with (*b*) those gained by hearing (the auditory nerve having connections with the middle cerebellar lobe) and (*c*) probably with those impressions resulting from the eye movements.

General Pathology.—Disease of a lateral lobe affects the same side of the body, but lesions of the middle (vermiform) lobe influence both sides. Results of extirpation in animals vary somewhat and will be considered only from their clinical aspect. Krauss found, in 100 cases of cerebellar disease, tumors in nearly 80 per cent., abscess in 10 per cent., cysts in 7 per cent. and softening and hemorrhage constituted the balance. Agenesis, sclerosis and gliomatosis are also occasionally found. Disease of one hemisphere, or indeed extensive lesions of both hemispheres may be fully compensated by the cerebrum. Acute disease of the cerebellum produces symptoms more frequently than does chronic disease, in which latter the cerebellum has opportunity for accommodative compensation. Rapidly growing tumors, abscess and sometimes acute vascular disease often produce the following symptoms, in part typical and in part secondary:

Typical Symptoms.—1. *Cerebellar ataxia* is almost always present and occurs when the patient stands; equilibrium is disturbed and the body sways (static ataxia). Ataxia also occurs when the patient moves (dynamic ataxia) and concerns especially the trunk and legs. The incoördination is due to disease of the middle lobe or vermis and the consequent withdrawal from the cerebrum of the cerebellar coördination. The gait is that of an inebriate (*démarche d'ivresse*); the body sways and the individual stumbles a zig-zag course, sometimes toward the side of the lesion. The arm and hand movements

are usually normal. Ataxia may also result from lesions of the nerves, spine or brain (cerebral cortex, internal capsule and probably tegmentum). In the ataxia resulting from other cerebral localization the incoördination affects the finer movements, is associated with disturbed muscle and stereognostic sense and very often with paresis. Bouillaud first described cerebellar ataxia and differentiated it from the tabetic type. The essential differences are (*a*) that in the cerebellar form the ataxia disappears when the patient is lying down and (*b*) that closing the eyes does not increase it (indeed it may often disappear).

2. *Vertigo* is very frequent and usually occurs rather when the patient sits than when he lies down. He thinks that objects are turning around him or he may feel himself being turned. The vertigo is either constant or paroxysmal and is due to lesion of the vestibular nerve which connects the semilunar canals with the cerebellum by means of the corpus restiforme.

Secondary Symptoms.—The following secondary symptoms are important but less significant than ataxia and vertigo: (*a*) *Choked disk* is rarely absent in cerebellar tumors and *amaurosis* often results from increased pressure on the third ventricle, leading to hydrocephalus internus and then to pressure on the chiasm or optic nerves. (*b*) *"Forced" postures* and movements are generally considered due to disease of the middle peduncle (*crus cerebelli ad pontem*). Forced postures of the head and trunk are observed; one eye looks upward and inward, while the other is directed downward and outward. Forced movements consist of the turning of the patient constantly to one side, movement in a circle and a tendency to fall toward the side of lesion and sometimes backward. (*c*) *Muscular weakness* is frequent in animals (Luciani) and occasional in man. A hemiparesis occurs on the side of lesion. The patient may not be able to rotate the trunk, straighten the body after bending or get up after falling. (*d*) The *patellar reflexes* are usually normal or increased. Sometimes they are lost from pressure on distant structures or from spinal disease. (*e*) *Vomiting* and *pain* in the head or nape of the neck are frequent and of some localizing value. (*f*) Occasionally the following symptoms develop; nystagmus, rhythmical contractions of the head, convulsions, paralysis of the cerebral nerves from pressure on the midbrain, disturbed hearing, intention tremor, muscular rigidity, irritability and weak memory.

XIV. Medulla.—(See Diseases of Medulla Oblongata.)

CIRCULATORY DISEASES OF THE BRAIN.

I. Anæmia of the Brain.—This term has been much abused. Differentiation from hyperæmia is sometimes impossible.

Etiology.—(*a*) General systemic anæmia may result from cachexia and hemorrhages of medical, surgical or obstetrical nature. (*b*) *Decreased blood supply,* due to cardiac insufficiency, valvular sten-

osis, stenosis of the vessels by aneurysm or tumor, evacuation of an enormously distended bladder and tapping of pleurisy or ascites leading to paresis of the thoracic or abdominal vessels may be causal. Fasting seems a cause in individuals who faint when the stomach is long empty. (*c*) *Capillary compression* by hydrocephalus or tumors, but especially by cerebral hemorrhage, may produce anæmia. Venous hyperæmia may act in the same way. (*d*) *Local causes* in the vessels will be considered under softening. Ligature or compression of one carotid produces symptoms only when the other vessel is atheromatous. Gerhardt induced convulsions by pressure on one carotid and considered them indicative of atheroma of the cerebral vessels.

Pathology.—The meninges and brain are pale, the brain firm and dry and *puncta vasculosa* fewer. The fluid in the ventricles is often increased. Geigel terms the slow capillary flow " diämorrhysis."

Symptoms.—Local anæmia of the brain leads to imperfect function, then loss of function and finally to necrosis, as in embolism or thrombosis (*q. v.*). In *sudden general anæmia* after hemorrhage or fainting, the subject becomes drowsy, syncope occurs and after return to consciousness amaurosis is frequent. There is ringing in the ears and the pupils at first contract from irritation of the third nerve and later dilate from its paralysis, a sign of danger. The skin is pale and clammy, the muscles lax and nausea frequent. Epileptiform convulsions often occur when much blood is lost. Anæmia of the medulla causes the slow, sighing respiration and yawning. Death after syncope was called nervous apoplexy by the old writers. *In gradually developing anæmia,* as pernicious anæmia, the brain power is lessened, and irritability, delirium, insomnia, headache, vertigo, weak muscles, spots before the eyes, impaired sight, ringing in the ears and sometimes deafness may result. These symptoms are aggravated by sitting or standing and are relieved on lying down. Palpitation and thoracic oppression are common symptoms. The pulse varies in rate and rhythm. Increased reflexes, paræsthesia and jerking of the muscles are frequent.

Marshall Hall has described *hydrencephaloid,* a variety of cerebral anæmia, seen in children suffering with acute diarrhœa, and characterized by contracted and later dilated pupils, depressed fontanelles, sunken face, pallor, early irritation followed by stupor or even coma. It may resemble meningitis from the strabismus, rigidity of the neck and convulsions. It is usually amenable to treatment.

Treatment.—Treatment, as well as diagnosis and prognosis, is chiefly that of the cause. The *anæmia* is treated by putting the patient in the recumbent posture and instituting rest in bed, iron, arsenic, a full diet and sometimes small doses of opiates. Acute anæmia necessitates absolute rest, free stimulation, coffee by rectum and enemata or subcutaneous infusions of salt solution. Firm bandaging of the extremities, sometimes called autotransfusion, is valuable. For *hydrencephaloid,* warmth, stimulation and fluids are necessary. Nitroglycerine is valuable in cases possibly due to vasomotor spasm.

II. Hyperæmia of the Brain.—Congestion of the brain is an obscure lesion. Andral (1836) described 8 varieties, and when Trousseau, in 1861, attempted to refer some varieties to epilepsy and Ménière's disease, he drew down upon him the wrath of the entire Academy. The hyperæmia in nephritis is usually uræmic and the arterial fluxion once attributed to erysipelas is entirely toxæmic. Geigel has proposed the term "endiämorrhysis" for the normal flushing of the brain by its capillaries. The essential element is the rapidity of the capillary flow. When the vessels dilate from paralysis of the sympathetic system the rapidity decreases, whence there is no hyperæmia but a lessened capillary flow, *adiämorrhysis.* When the vessels contract from sympathetic irritation, the brain is not anæmic, but the capillary flow is accelerated, *hyperdiämorrhysis.* General arterial tension is not important, but the tension of the vessel wall is the essential element (Geigel).

Etiology.—Hyperæmia may result from active or passive congestion. The prevailing description may be adhered to provisionally.

Active congestion results (*a*) from cardiac overaction; (*b*) from overfilling of the brain vessels, as in stenosis of the isthmus of the aorta; (*c*) from contraction of the arterioles in other parts; (*d*) from dilatation of the brain vessels by alcohol, nitroglycerine, coffee, exophthalmic goitre, excessive brain work, neurotic states and insolation. (*e*) It may occur as the first stage of inflammation. (*f*) In plethoric men with florid faces, thick necks and short thoraces, active congestion may cause certain passing brain symptoms.

Passive congestion results (*a*) from general venous stasis, as in valvular heart lesions; and (*b*) from local venous stasis, as sinus thrombosis; Geigel held that venous stasis leads to capillary anæmia.

Pathology.—The pathological findings are few, for congestion disappears after death; the brain may appear anæmic after death by strangulation and the head may become congested after death because of its posture alone. The sinuses and veins are usually turgid and the color of the brain is darker. Microscopic examination shows distention of the capillaries, at times increased blood pigment or actual rupture into the perivascular lymphatic sheaths or into the brain substance.

Symptoms.—No symptoms are characteristic. In *active congestion* the symptoms vary with the cause; chronic cause fewer symptoms than acute factors. Whether toxæmic symptoms of the acute infections are due to hyperæmia is an open question; the headache in early typhoid has often responded to ergot in personal experience. Nothnagel and Gowers report cases with paroxysmal throbbing of the vessels, flushing, headache, delirium, sometimes nausea and fever, relieved by nosebleed and venesection. Irritability, insomnia, spots before the eyes, ringing in the ears and increased pulse are said to occur. In progressive paralysis the transient pyrexia, heat in the head or coma is referred to congestion of the brain. In middle and advanced life the so-called congestive apoplexy has been noted, which consists of

coma, sometimes with transient hemiplegia, though most cases are due to small hemorrhages, softening or disease of the vessels.

Passive congestion, if gradual in onset, may be well tolerated or may produce stupor, dulness or even delirium. If intermittent, as from coughing, the symptoms are a sense of fulness in the head, headache and sometimes convulsions; these are increased by exertion, by the dorsal decubitus and constipation.

Diagnosis.—Focal symptoms, as hemiplegia, indicate organic disease. Such symptoms, as flushing, heat or pressure in the head, cannot be used for diagnosis, because they are often due to neurasthenia or to hypochondriasis. Marie and Leube have never made the diagnosis of brain hyperæmia. The great similarity between the symptoms of anæmia and hyperæmia of the brain has been noted.

Treatment.—Active congestion is treated by slight elevation of the head; by *venesection* in extreme or very plethoric cases in adults or in children by application of *leeches* over the mastoid bone, the veins of which connect with those of the brain; by *free purgation,* by 2 drops of croton oil, followed by mercurials and concentrated salines, to draw the blood to the large abdominal vessels; by *hot baths,* to draw the blood to the periphery; bromides and *vasoconstricting* measures, especially digitalis. Alcohol and opiates (save in small doses) should be avoided; ice may be applied to the head and carotids; a *fluid diet* should be given.

III. Œdema of the Brain.—**Etiology.**—Œdema of the brain results from the same causes as general œdema: (*a*) circulatory disturbances, as venous congestion in cardiac disease, emphysema and the acute infections; and (*b*) the *marantic group,* including cachexia and nephritis. Collateral and inflammatory œdema may develop around brain tumor, abscess or hemorrhage. In some instances brain œdema occurs in the death agony. It may also develop when the brain shrinks, *hydrops ex vacuo.*

Pathology.—The brain is pale, moist and glistening on section, the so-called "wet brain." The affection may be general or local; the ventricular and subarachnoid fluid is increased. The volume of the brain may be increased in the generalized form.

Symptoms.—The symptomatology is indefinite and a certain diagnosis cannot be made between œdema and lessening of the capillary velocity. When the œdema is localized, transient focal symptoms may develop, as unilateral convulsions or hemiplegia; even crossed paralysis was observed personally in cases of nephritis. Traube held that brain œdema causes the uræmic manifestations of nephritis, a view vigorously opposed by Cohnheim. Great variability in the symptomatology of organic brain disease may be due to associated œdema.

IV. Cerebral Hemorrhage.—This most common and important of all brain lesions involves an enormous part of brain pathology. It is usually hemorrhage into the brain substance and is spontaneous.

Although apoplexy strictly refers to a "stroke," which is symptomatic of various other lesions, it is used to indicate hemorrhage into the brain (Rochoux).

Etiology.—(*a*) *Miliary aneurysms* are the chief or sole cause of cerebral hemorrhage; the leading issue at present relates to their pathogenesis. According to many authors, the causes are (*b*) those of *arteriosclerosis* (alcoholism, plumbism, syphilis, hard physical work, contracted kidney, gout, diabetes and over-eating), but others maintain that atheroma does not *per se* predispose to hemorrhage. Eighty per cent. of hemorrhages occur *after the fortieth year* of life. It may occur in early life from glioma, whooping-cough or trauma. More cases occur *in men* (80 per cent.) than in women. *Heredity* is not an uncommon factor, but relates rather to renal and arterial lesions than to an actual heredity of hemorrhage; this is especially emphasized by English writers and by Dieulafoy. Hemorrhage is most common among civilized races and in temperate zones, because of the strenuous habits of life. For years the importance of the *apoplectic habitus* (plethora, ruddy face, short, thick neck, wide shoulders and chest, round, small body and large muscles) has been emphasized, although Gowers finds most cases in thin individuals. *Contracted kidneys* are found in 33 per cent. of cases of cerebral hemorrhage and are among the chief causes of atheroma and miliary aneurysm. (*c*) *Increased arterial tension* is usually considered an important etiological factor. Hypertension alone is less important than its relation to arteriosclerosis and miliary aneurysms, which occur where the blood pressure is greatest, as where the vessels branch. Rupture is also seen in cardiac hypertrophy from stenosis at the isthmus of the aorta. Syphilis is usually considered a factor, but it more frequently produces softening than hemorrhage; it is a causal factor of aneurysms of the large vessels of the brain. Violent exertion, alcoholic excesses, excitement, coitus, straining, overeating, vomiting, coughing or convulsions may rupture an already-diseased vessel or a miliary aneurysm. A healthy vessel almost never bursts, however high the blood pressure. Embolism from heart disease may produce the "embolic aneurysm." Sometimes trauma is a factor, usually, however, with the causes mentioned. (*d*) *Permeability of the blood vessels* causes a few cases, as in nephritis, purpura, anæmias and infections, by hemorrhage by diapedesis through the arterial wall.

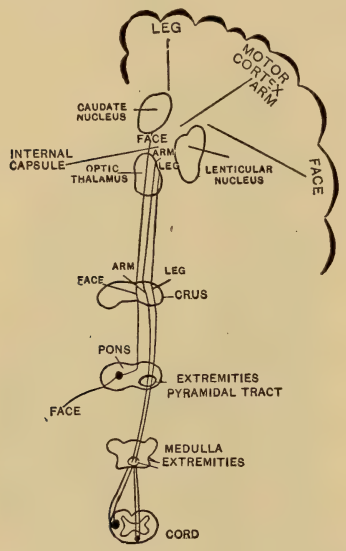
Pathology.—Hemorrhage most often occurs in the branches of the arteria fossæ Sylvii. Among its branches the lenticulostriate artery of Duret, called by Charcot *the artery of hemorrhage,* is the seat of hemorrhage in 75 per cent. of cases. Though the vessel is small, it is likely to rupture because (*a*) it is so near to the large internal carotid and its course is very direct; (*b*) the blood pressure is high; (*c*) the vessel has no special external support; and (*d*) there is no collateral circulation, hence no relief to pressure, rendering the vessel tortuous. Rupture occurs very often in the long direction of the nerve fibers of the internal capsule. The veins stagnate very easily

in this locality. Hemorrhage is almost always arterial. *Miliary aneurysms* were described by Brunner in 1700, but Charcot and Bouchard (1866) emphasized their frequent relation to brain hemorrhage. They are fusiform, more rarely sacciform, enlargements of the arterioles, sometimes multiple, like a bunch of grapes, and measuring $\frac{1}{100}$ to $\frac{1}{25}$ of an inch; they may be seen with the naked eye, but are best observed by careful washing or macerating in water, and examination on a slide under a low power. They are found in persons over forty years of age and in the locations where hemorrhages are most common, *i. e.,* in the central ganglia. Their *pathogenesis* is obscure. Charcot and Bouchard described a change in the outer tunic (periarteritis) but Eichler and Zeigler held that intimal changes were primary. Roth finds that miliary aneurysm is due to atrophy of the muscular coat; this is at present the generally accepted theory. They are the chief or sole cause of spontaneous hemorrhage. Large aneurysms will be considered separately.

Localization.—Hemorrhage is most common about the lateral ventricle, *i. e.,* in the caudate and lenticular nuclei, the internal capsule and the adjacent centrum ovale, extending to the optic thalamus, but rarely beginning in it. Hemorrhage in the white matter has usually an elongated form. It is much less common in the cortex, pons, crus, cerebellum or medulla. Though it occurs mostly in the substance of the brain, hemorrhage may either rupture on the surface or break into the ventricles, causing sudden death; this is the usual autopsy finding in recent apoplexy. Hemorrhage is usually single. The multiple form is observed in anæmia. When the skull is removed after large hemorrhages the dura is tense, the convolutions flattened and pale, the falx pushed toward the other side and the fissures indistinct. Fluctuation may be felt in rare cases and on section the focus is seen; it sometimes involves the entire hemisphere or may be no larger than a pea, but usually the size of a hazel-nut or walnut. The brain around the focus is softened from pressure, œdematous and in the focus torn and pulpy. The dark clot later becomes chocolate-colored, then yellow-red and finally yellow-white. The wall of the focus is irregular in the gray nuclei and regular in the white matter and threads of bloodvessels run through it. Microscopically, leukocytes laden with fat drops, hæmatoidin and disorganized tissue are found. As the blood absorbs, the cavity becomes more regular and encapsulated by inflammation and contains a serous or milky fluid; this is the *apoplectic cyst* of Virchow. Final replacement of the focus by an *apoplectic scar* is much less common. *Traumatic hemorrhage* may occur infrequently near the point of injury or *contre coup* on the opposite side; it sometimes takes place after a week or more; this is the late traumatic hemorrhage of Bollinger, which occurs near the fourth ventricle and aqueduct of Sylvius, and probably in areas of traumatic softening. Brain hemorrhage may indirectly press on other parts, may press on their arteries and veins or directly sever the internal capsule.

Secondary degeneration occurs in the *pyramidal tracts,* mostly as a result of lesions of the internal capsule. It is usually partial, and sound and diseased fibers intermingle; it descends through the crus, pons, medulla and cord, in the latter of which it involves the inner part of the anterior columns of the same side (the direct anterior

FIG. 56.

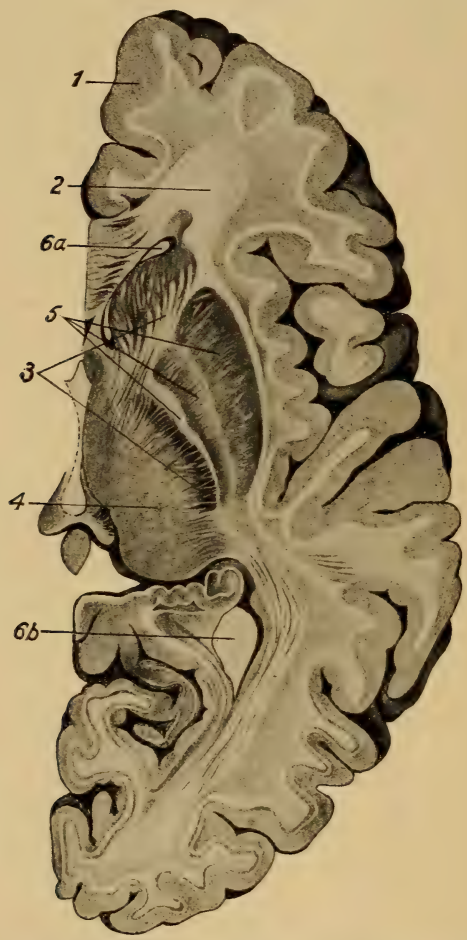


Showing course of individual fibers of motor tracts.

pyramidal tract) and part of the lateral columns of the opposite side (the crossed lateral tract). These areas become progressively smaller and cease just above the conus medullaris. In children a cerebral lesion may cause degeneration in the anterior horns of the cord and their fibers. An ascending degeneration occurs only when the large pyramidal tracts of the cortex are diseased, except in the young, in whom degeneration of all neurones is prone to follow the destruction of one neurone. (See figures under LOCALIZATION IN THE SPINAL CORD.) Other degenerations: (*a*) The *optic radiation* degenerates when a lesion occurs in the cortex of the cuneus and also degenerative changes in the external geniculate body and pulvinar. After years it may reach the optic nerve. (*b*) Hemorrhage in the second and third frontal lobes leads to degeneration in the *anterior limb of the capsule,* median basal fibers of the crus and the fibers connecting the optic thalamus with the cortex. (*c*) Lesions in the temporal lobes cause degeneration in the *lateral part of the crus.* (*d*) Degeneration in the median lemniscus follows diseases of the cortex, the subthalamic region and the upper part of the pons; it crosses to the opposite nuclei of the medulla and after years destroys the ganglion cells in the posterior columns of the cord. Cerebellar hemorrhage may rarely cause degeneration of the arm of the pons and of the corpus restiforme.

Symptoms.—*Prodromes* are most rare. Miliary aneurysms produce no symptoms until they rupture and premonitory symptoms are due to *atheroma.* None of them are pathognomonic. They may be (*a*) general, as described under congestion and softening (*v. i.*); (*b*) cardiorenal, as hypertrophied heart, high arterial tension and the urinary findings of contracted kidney; or (*c*) sometimes focal, as diplopia, alexia, facial paresis, dysarthria or preparalytic chorea.

FIG. 57.



Horizontal section through the right hemisphere; 1, Cortex; 2, white substance; 3, internal capsule; 4, optic thalamus; 5, lenticular nucleus; 6*a*, anterior horn; and 6*b*, posterior horn of the lateral ventricle. (After Déjérine.)

Possibly in some cases prodromal symptoms are due to an initial small hemorrhage or to hemorrhage developing very slowly.

1. THE INSULT OR STROKE AND ITS PRIMARY SYMPTOMS.—The "stroke" or apoplexy is usually *sudden;* its most common general symptom is *coma,* which may be absent in small lesions and its most

common focal symptom is *hemiplegia.* It may develop during effort or excitement, or again without cause, as during sleep, when the head is low. In *ingravescent apoplexy* the onset may be gradual, with delirium, convulsions and finally coma; it is due to hemorrhage external to the lenticular nucleus, which later involves the internal capsule (hemiplegia). Death may, in rare instances, occur in five minutes (Abercrombie) from rupture into the ventricles or from direct pressure on the medulla (*fulminating apoplexy*).

The insult, or stroke, is due chiefly to *anæmia of the cortex* and to some extent to direct brain trauma, pressure by the hemorrhage on other vessels, serous infiltration and alterations in the cerebrospinal fluid (in which lumbar puncture sometimes shows blood). Its *cardinal typical symptoms* are *coma,* which is greater the nearer the lesion is to the optic thalamus and third ventricle; *flushed face,* or sometimes pallor or cyanosis; usually a full, tense, regular and *slow pulse;* long, deep, *stertorous breathing* (due in part to palate paralysis); the *cheeks puff out,* but are drawn in on inspiration, as a result of paralysis of the buccinator muscle; inspiration is less irregular than it is usually said to be; yawning is frequent (Todd); the *temperature* usually suffers an *initial fall,* but rises when reaction sets in, though lesions in the pons, medulla or central ganglia may cause an initial rise. The *pupils* are variable, usually wide, from paralysis of the third nuclei or from sympathetic stimulation; they are sometimes unequal and in deep coma *reactionless.* They may be narrow from irritation of the oculomotor nucleus in lesions of the pons, or when the ventricles are involved. Convulsions are infrequent. The head and eyes look toward the side of the brain lesion ("conjugate deviation"), *a flaccid hemiplegia* occurs, in which the limbs fall limp when they are lifted; the paralyzed muscles appear semi-fluid and lax; the *reflexes* are *gone,* which symptoms will be described in detail below. There are *involuntary evacuations* of urine and fæces. Ollivier described an increase of the urine with a low specific gravity (1,004) and transitory *albuminuria* and *glycosuria,* usually due to a large lesion producing indirect pressure on the medulla or to a lesion in the medulla itself.

2. The permanent damage can only be estimated when the reaction is passed. The *direct focal symptoms* are those of the hemorrhage itself and the subsequent cyst or scar; they are permanent if essential structures are involved, as the internal capsule. *Indirect focal symptoms* are those of other structures disturbed by pressure, œdema or inflammatory reaction. Time is required to separate these two classes of symptoms. The pulse and respiratory symptoms, conjugate deviation, albuminuria and temperature are usually indirect symptoms.

3. Details of Important Symptoms.—1. *Convulsions* are not common; they indicate involvement of the pons, ventricles, cortex or corpus striatum.

2. *Conjugate deviation,* described by Vulpian, but especially by

Prévost, is a transitory phenomenon in which the *lateral muscles of the head and eyes are paralyzed;* their fellows on the sound side contract, so that "the patient *looks at his lesion*" (Prévost); that is, in left hemiplegia the eyes and head look toward the right side of the brain, where the lesion is located. The centres paralyzed are in the lower parietal lobe (gyrus angularis and gyrus supramarginalis, Landouzy). It disappears in a few days because the sound side of the brain compensates or because it is a distant pressure symptom, or at the most only a moderate paresis remains, shown by nystagmus. Conjugate deviation may also be due to *irritation* or convulsions (Jacksonian epilepsy), when the patient "looks away from his lesion" (Landouzy), *i. e.,* toward the side affected with convulsions. These differ from the deviation in localization in the pons (*q. v.*).

3. The *hemiplegia* (see LOCALIZATION, INTERNAL CAPSULE) is due to a lesion of the internal capsule (75 per cent. of cases). During deep coma it is often difficult to determine which side is paralyzed. The affected side is usually flaccid and the mouth shows greater puffing on the hemiplegic half. If the coma is not profound the sound arm and leg may move. Sometimes, especially with hemorrhage into the lateral ventricle, the paralyzed side is rigid (early rigidity or contracture). The temperature is at first lower on the hemiplegic side from paralysis of a cortical centre. Later, during reaction, it is higher on the side of paralysis and may occur with œdema in this arm and leg. After consciousness returns the contralateral hemiplegia is more clearly seen. The lower two-thirds of the face (nose, mouth, cheek) are paralyzed, but the upper third (eye and forehead) is usually quite free; in very rare cases only, a total facial paralysis is seen. The lids can be moved, but the nasolabial fold is obliterated, the lips cannot be puckered as though to whistle, the mouth is oblique or a little triangular when the teeth are shown. The upper part is so often unaffected because it is innervated from both hemispheres or because it has two distinct centres and paths, whereas in peripheral facial paralysis all branches are involved and atrophy and the reactions of degeneration occur. If the optic thalamus (*q. v.*) is intact, the paralysis is not apparent on emotional movements, as laughing. The *tongue* is slightly involved; when protruded it deviates toward the diseased side (Schiff, Heidenhain) because the sound genioglossus muscle pulls the tongue forward and over toward the hemiplegic side; the base of the tongue is higher on the paralyzed side which may lead to some difficulty in the external speech mechanism (dysarthria), while internal speech (*i. e.*, aphasia) is not often disturbed unless there is psychical alteration. The *palate* may hang lower on the paralyzed side, but this is of little importance because it varies physiologically. Deglutition, mastication and phonation are but little or only transitorily involved; marked difficulty in mastication is usually associated with total hemianæsthesia. In the *trunk* paralysis of the trapezius and levator anguli scapulæ causes sagging of the shoulder, while other muscles of the neck are relatively free.

(Wernicke thinks certain cerebral fibers of the spinal accessory are involved.) The *accessory muscles of respiration,* as the scaleni, are involved on deep inspiration, and ordinary respiration is somewhat less complete, to which some would refer certain lung complications, as atelectasis. Hughlings-Jackson finds respiratory movement greater on the paralyzed side in automatic breathing and greater on the sound side in forced breathing. Early, the paralysis of the *arm and leg* is often absolute, sometimes partial (hemiparesis). The arm is usually more involved than the leg. In rare cases the arm may be free in linear lesions; again the leg may be unaffected or the face remains free (see INTERNAL CAPSULE LOCALIZATION); monoplegia is much less common than in cortical and subcortical disease and when one member is more affected than the others there is still partial or "dissociated" hemiplegia. In the *typical case* the arm is most involved, though all parts on one side usually are somewhat paralyzed. The arm abductors, hand flexors and finger extensors are most affected. In the leg, the extensors of the leg, dorsal foot flexors and knee flexors are most implicated. The *paralysis is flaccid.* Considerable improvement may follow (see SECONDARY SYMPTOMS). The hemiplegia is almost invariably contralateral, *i. e.,* opposite to the side of the lesion. (In the older literature hemiplegia on the same side, or collateral hemiplegia, was described; Lederhose collected 45 cases, due (*a*) to more than one lesion, (*b*) to a focus pressing on the other side of the brain, or (*c*) to mistaking the early rigidity of the paralyzed side for the normal side.

4. *Sensory disturbance* has been considered in the topography of the internal capsule. It is observed mostly during the insult or reaction, is less important than hemiplegia, less frequent, less distinct, less complete and less stable. Some hemianæsthesia is frequently found in early cases after careful examination, as disturbed tactile sensation, stereognostic sense and sometimes muscle sense, pains and hyperæsthesia or paræsthesia. A large lesion only can cause a complete hemianæsthesia, which very seldom occurs without some paresis. Its affects the skin and mucosa of the nose, mouth, palate and eyes, but leaves the cornea intact (Grasset), which is supplied by the ophthalmic ganglion (see DIAGNOSIS). Cortical hemianæsthesia is usually partial and most often associated with a monoplegia; lesions a little above the capsule, may produce hemianæsthesia, involving the fibers from the thalamus to the cortex. Hemianæsthesia also occurs in lesions of the crus, pons, medulla and cord (*q. v.*).

5. *Special senses.* Hemianopsia, due to pressure on the optic thalamus, is frequent, usually occurs with conjugate deviation, is temporary and is often overlooked.

6. The *reflexes* of the skin, cornea, pupils, mucosæ and tendons are suspended when there is complete coma, in which the central nervous system is almost wholly paretic. After the stroke they return slowly, first on the sound side. On the *hemiplegic side* they return more slowly, the periosteal, tendon and patellar reflexes often become exag-

gerated from interrupted inhibition; the patellar reflexes may return only after a considerable time. The skin reflexes return slowly, especially the epigastric, cremasteric, scrotal and mammillary; this is a point of early diagnostic value; they probably have a higher centre than the tendon reflexes, although the explanation of their conduct is not clear; it has also been suggested that there is a cerebral centre controlling Setchenow's inhibitory reflex centre in the medulla; when this higher centre is affected the medulla centre wholly inhibits the reflexes.

The *Babinski sign* (toe phenomenon) indicates organic pyramidal disease and may be found very early; when a sharp object is slowly drawn over the outer part of the sound sole there is plantar flexion of the large and sometimes of the other toes; when drawn over the diseased sole there is great dorsal flexion (extension) of the great toe. After the primary insult, coma and reaction are over, the chronic symptoms can be studied to the best advantage.

4. The Chronic or Secondary Symptoms.—The indirect symptoms, the inflammatory reaction and collateral œdema disappear; this is usually followed by considerable improvement.

1. If the *hemiplegia* is indirect, from a lesion without the capsule, marked improvement or rarely total recovery ensues. Complete lasting hemiplegia follows total division of the pyramidal tracts, due to hemorrhage from the anterior choroid artery (Kolisko). The face may recover rapidly; certain muscles are entirely unaffected or are involved for a short time only; this is explained by *Broadbent's theory* that *muscles used in pairs* are little involved or escape entirely; with some modification of his theory, we find that: the *intercostals, masseters* and trunk muscles used together, innervated from both hemispheres and having little differentiation in function, escape permanent paralysis. The functions of the *arm* and *hand* are especially complicated, are more often used alone, are innervated almost wholly from one hemisphere and therefore suffer the greatest permanent damage. In adults the hand is usually disabled for life. The *legs* are much used together, have some innervation from both hemispheres and often show improvement after paralysis. Just how compensation occurs is undetermined. Broadbent thinks that the bilateral spinal and bulbar nuclei enable "paired muscles" to be innervated from the sound hemisphere. Possibly uncrossed fibers in the anterior (and lateral) columns may explain the phenomenon. Compensation is not an absolute advantage, for Brown-Séquard found the sound side reduced in strength in 50 per cent. of his cases. The face often improves after an attack. In the *arm* the residual paralysis affects functionally associated muscle groups rather than individual muscles, as the muscles supinating the forearm, those abducting and lifting the arm and shoulder, those moving the elbow, opening the hand and apposing the thumb (Wernicke and Mann). The shoulder improves a little, the elbows considerably, the hands very little. In the leg the flexors, dorsal flexors of the foot which "shorten" the leg in walking,

abductors and internal rotators of the hip are most involved. The hemiplegic gait is awkward, the foot is swung out and around in a half circle and scrapes the ground. The power of the paralyzed leg is greater when it is used with the sound leg, as in walking, than when it is used by itself.

2. The *contracture,* the important posthemiplegic manifestation, is the transitory or permanent fixation of a joint in an abnormal posture from nutritive or functional factors. *Passive contractures* are mechanical, as from muscular shortening, while *active* contractures result from disease of the central nervous system. Early contractures are often regarded as tonic spasm (myotatic irritability). The late contracture develops in two to four weeks and is quite typical; the shoulder is lifted, the arm adducted to the chest, the elbow rigid, the forearm pronated, the wrist flexed and the fingers flexed even violently into the palm. When the wrist is passively flexed the fingers can sometimes be extended. In general the contracture involves both extensors and flexors, but more often the latter. The contractures occur in the muscles which are least paralyzed. In the lower extremity, the contracture is not marked (in the thigh there is often only a trace), the knee is extended, plantar flexion of the foot occurs (*pes equino-varus*), the great toe is dorsally flexed. Locomotion is difficult, for there is bending of the trunk to the sound side, lifting of the pelvis because of the adduction paralysis and swinging of the foot over the sound foot. There may be pain in walking and the sound leg also suffers some slight contracture. Contracture may occur in the face, platysma and sternomastoid, but the trunk is rarely affected. Hitzig regards them as involuntary associated movements. In rare cases, and largely in children, the paralyzed side remains lax (*hémiplégie flasque* of Bouchard).

3. *Associated movements* are variously explained; by Westphal as due to lessening of reflex inhibition, by Hitzig as movements attempted in paralyzed muscles but reflected to their associates and by Leube as due to bilateral representation in the hemispheres; an effort to grasp with the sound hand produces a similar movement in the one paralyzed; movement of the diseased side occurs after passive movement of the sound one; movement of the paralyzed foot results when the patient attempts to flex the hip.

4. *Hemitremor* is uncommon. The muscles are stimulated, but not enough vibrations occur for actual involuntary movement.

5. *Atrophy* may occur in both spastic and flaccid cerebral forms, though more in the latter; Steinert concluded that atrophy is as frequent in cerebral as in peripheral paralysis. Charcot referred it to degeneration of the anterior horns of the cord, while Quincke and Senator assume an injury to some trophic paths. It develops in a month or two, has no relation to the degree of paralysis, is often associated with disturbed sensation and muscle sense, affects principally the arm and occurs in large lesions which are either cortical in the region of the third branch of the artery of Sylvius or central in the

distribution of the lenticulothalamic artery. The electrical irritability of the muscles is normal or somewhat depressed. The skin may atrophy.

6. *Posthemiplegic chorea* (Wier Mitchell, Charcot, Raymond) results in 80 per cent. of cases from lesions in the internal capsule or near it, in the optic thalamus, the lenticular or caudate nucleus, from which irritation is reflected to the brain cortex. It develops months or years after *small lesions,* when the hemiplegia and rigidity are largely gone. It occurs especially in the arm, causes movements similar to but coarser than those of chorea, sometimes resembles the tremor of multiple sclerosis or paralysis agitans and is often associated with pain and sensory disturbance (see Localization, Internal Capsule). It sometimes is preparalytic. In *posthemiplegic athetosis* (Hammond) the fingers and toes are incessantly moved, athetosis meaning "without fixed position." It is a "mobile spasm" consisting of somewhat spasmodic, slow, coördinated movements in paralyzed members; it is frequent in partial paralysis acquired early in life, is slow in development, appears after the paralysis has regressed and has the same localization as hemichorea. The following differentiation by Greidenberg and v. Monakow gives their main characteristics:

Hemichorea.	*vs.*	Hemiathetosis.
1. Face, trunk, whole extremity involved; may pass to all extremities.		1. Chiefly or alone in forearm and hand (fingers) and in leg and foot (toes); simultaneous innervation of antagonists; other parts paretic and stiff.
2. *Excursion* active, manifold, tremor-like, purposeless, increased by exertion, tension lost by irritation.		2. Slow rhythmic hyperextension, elastic resistance, seems purposive, joints fixed.
3. Contracture little or none; limbs relaxed; no great deformity.		3. Contracture frequent, but spasmus mobilis often great.
4. Hemianæsthesia frequent.		4. Hemianæsthesia rare.
5. Quiet in sleep.		5. Not always quiet in sleep.
6. Increased by movements and attempts to suppress them.		6. Lessened to some extent by movement.

7. *In trophic and vasomotor disturbance* (*v. s.,* Muscular Atrophy) the paralyzed members are usually cool, moist and sometimes œdematous. The skin is glossy, even livid and fissured. Nothnagel observed in some cases paralysis of the cervical sympathetic causing increased temperature, redness, narrowing of the eye fissure, drooping of the upper lid, retraction of the eyeball, lachrymation and narrow pupils. Sometimes there is overgrowth of the hair. Bedsores were considered trophic by Charcot, but can usually be avoided by care regarding pressure or heat and by cleanliness. Swelling of the joints and hypertrophic neuritis (Charcot) on the hemiplegic side are rare,

8. *Psychic disturbance* is not symptomatic of brain hemorrhage and is chiefly referable to arteriosclerosis. It is evidenced by self-concentration, weak memory, insomnia and irritability. Carpani found more mental alteration when the left half of the brain was diseased. Laughing and weeping are referable to disease of the optic thalamus (*q. v.*) or to disease of the median fibers of the pons (Brissaud).

Diagnosis of Brain Hemorrhage.—Two cardinal features, *coma* and *hemiplegia,* together with the *typical insult,* facilitate diagnosis. In cases with slow onset, differentiation from thrombosis (*q. v.*) is difficult.

1. Coma.—In *syncope* the pulse is fast, weak and often irregular; in "cardiac apoplexy" an insult follows heart rupture, myocarditis and various forms of heart failure, and differentiation from rapidly fatal brain hemorrhage may be impossible. It is always dangerous to make a diagnosis of *alcoholic coma;* an intoxicated individual may suffer injury, basal fracture or actual apoplexy; an opinion should be withheld until such complications can be excluded, though the alcoholic coma is usually incomplete, the vomitus and breath smell of alcohol, violent struggling is frequent and early convulsions and focal symptoms are rare. *Opium coma* is characterized at first by slow onset, slow respiration, slow, hard pulse, flushed skin, great contraction of the pupils, opium in the stomach washings and absence of convulsions and focal symptoms; later there are Cheyne-Stokes' respiration, rapid pulse, clammy cyanotic skin and dilated pupils. In opium and similar intoxications the focal symptoms and reflexes of apoplexy are lacking. Hemorrhage in the pons produces narrow pupils, which are often unequal. While in general the diagnosis of apoplexy during the coma is precarious, *uræmic coma* rarely occurs without premonitory symptoms, as *headache,* drowsiness, delirium, *restlessness, vomiting,* myosis, *retinitis* or less often convulsions; the coma is often less profound than in cerebral hemorrhage. The renal, cardiac and arterial findings of nephritis may also occur in apoplexy, of which nephritis is a frequent cause (33 per cent.) and it must be remembered that focal symptoms sometimes occur in uræmia; the author has repeatedly observed hemiplegia and even crossed paralyses. In deep coma rigidity may be observed in both uræmia and apoplexy, but in uræmia it is often variable. In *diabetic coma* the fruity, acetone breath, acetone in the urine, headache, unrest, Kussmaul's breathing and absence of focal symptoms are characteristic. The coma may be sudden and granular casts are frequent; the diagnosis is suggested by a clear history of glycosuria. *Symptomatic apoplexy* is of more theoretical than practical importance; transient coma and hemiplegia sometimes occur in brain œdema, congestion, progressive paralysis, multiple sclerosis and more rarely in epilepsy and tabes.

2. Hemiplegia.—This is a prominent finding in embolism and thrombosis (*q. v.,* for Differentiation); an exact diagnosis is often impossible. Kolisko states that most cases with the clinical diagnosis

of embolism prove to be hemorrhage and *vice versa.* Hemiplegia may occur in brain abscess and meningitis. When it occurs in meningitis with coma, there are headache, generalized convulsions, rigidity, hyperæsthesia, optic neuritis, eye-paralyses and fever. In extradural hæmatoma and pachymeningitis the Jacksonian epilepsy followed by cortical paralysis is distinctive, *i. e.,* the focal symptoms are prominent compared with the general symptoms of early apoplexy. Hemiplegia may also occur in hysteria (*q. v.*), syphilis, brain tumor, trauma, chorea or paralysis agitans. Hemiplegia, not resulting from any lesion, may occur in alcoholism, plumbism, gout, uræmia, diabetes, pneumonia, etc.

3. Other Locations of Hemorrhage.—(*a*) Hemorrhage may occur in the meninges (see Diseases of Meninges). (*b*) It may occur, though rarely, in the *cortex;* Jacksonian epilepsy in subcortical lesions, convulsions and rigidity may be severe and lasting (Mills). (*c*) *Ventricular hemorrhage* is usually secondary to hemorrhage in the corpus striatum or optic thalamus and usually reaches all the ventricles. It may in rare cases be primary from aneurysms or ruptured veins in the choroid plexus (Dana). One-third of all cases are in persons under forty years of age. It occurs usually with violent symptoms, convulsions (33 per cent.), early and sometimes bilateral rigidity. The conjugate deviation changes to the other side, the pulse is slowed, the temperature falls, the reflexes are suspended. Cheyne-Stokes' breathing, glycosuria and polyuria develop and death usually results within a day; the onset is sometimes slow. (*d*) Hemorrhage in the *crus* (*q. v.,* Localization) is usually descending, from bleeding near the internal capsule. Crossed hemiplegia oculomotoria (Weber's type), crossed ptosis or diplegia may be observed. (*e*) Apoplexy in the *pons* (*q. v.*) occurs in 10 per cent. of cases, often in the median line and frequently with convulsions. Paralysis and convulsions may occur in the arms or legs only; the trunk musculature is involved more frequently than in higher hemorrhages. The eyes look away from the side of the lesion. The pupils may be large from paralysis or small from stimulation of the third nerve; in the latter instance opium poisoning must be differentiated. Respiration is affected early, vomiting is frequent, there is often early high fever and death is usual within twenty-four hours; it has occurred in seven minutes. (*f*) Hemorrhage in the *medulla* will be considered later. Early or instantaneous death is frequent, but a gradual onset is known. It is rarely diagnosticated. It may very rarely induce gastro-intestinal hemorrhage. (*g*) *Cerebellar apoplexy* occurs in 5 to 8 per cent. of cases. Vomiting is very frequent (50 per cent.). The insult is sometimes less marked than in the usual type and "the absence of hemiplegia is more significant than its presence" (Gowers). Hemiplegia is due to indirect pressure or secondary ventricular hemorrhage. Cerebellar symptoms, as ataxia, may result.

Prognosis.—The prognosis concerns (*a*) the immediate danger and (*b*) the ultimate outcome. (*a*) The *immediate prognosis* is always

serious. In hospital cases 75 per cent. die but in general practice 66 per cent. survive the first stroke, 33 per cent. the second and very few the third. When death occurs it is usual in one-half to two days, but the outcome is uncertain for a week, because light cases, even rare forms without coma, may rupture into the ventricle with rapidly fatal result. Coma lasting more than twenty-four to thirty hours is dangerous. Early low fall of temperature or its rapid early rise is unfavorable, indicating a large focus. For the same reason, conjugate deviation, relaxed sphincters, impaired breathing, tracheal rattling, bilateral or ventricular symptoms, marked glycosuria or albuminuria, decubitus in the first week, convulsions and rapid pulse are unfavorable. The mode of death is as follows: the breathing becomes more rapid, irregular, of the ominous Cheyne-Stokes' type, and rattling from accumulated secretion in the air-passages; the slow and regular pulse becomes rapid, small, irregular, often with a rise of temperature and finally ceases, but respiration for a time continues; death occurs in total coma.

(*b*) *Ultimate Outcome.*—The symptoms of shock disappear and the patient becomes partly conscious or delirious; the *period of reaction* begins usually within forty-eight hours after the onset. The pulse becomes faster, the temperature rises, pain develops in the head and limbs and rarely the paralyzed limbs become rigid. Life still hangs in the balance and the patient may die from the reaction, hypostatic pneumonia (called trophic by some writers), pulmonary œdema or hemorrhage, acute decubitus or from a second hemorrhage. Reaction is over in one to six weeks. If the patient survives there is improvement, depending on the extent and localization of the injury, determined only after weeks or months. If movement appears in three weeks the outlook is good; if it does not return in three or four months it is not likely to develop later. Disappearance of indirect pressure leads to bettering (i) of damage to the sensory nerves and those of special sense, (ii) of motility, first of the trunk and face, then of the leg and less of the arm. Absolute recovery is improbable. Hemichorea and hemiathetosis are incurable. Broken neurones never unite and secondary degeneration, contractures and increased reflexes result. Exaggerated reflexes, developing a few weeks after the injury, are forerunners of contractures and ankle clonus, which preclude full regression. The outlook is fairly good in children when the hemorrhage is not due to blood affections, because compensation is likely to occur. Recurrence is possible in decrepid and aged subjects, although less common than usually believed; the average duration of life is five years (Dana).

Treatment.—1. Prophylaxis.—Prophylaxis regarding worry, hard work, syphilis, the use of alcohol, eating and excesses is necessary when there is hereditary tendency toward hemorrhage or when one hemorrhage has already occurred.

2. The Insult.—(*a*) Absolute *quiet* is indicated, and any movement, transportation or extended examination should be avoided.

(*b*) The head should be slightly elevated, but flexion of the neck, tight clothes or anything hindering the return venous flow should be avoided. Bowles thinks that stertorous breathing is due to the position of the paralyzed palate and that turning the patient on his side relieves it more effectively than does venesection. (*c*) *Venesection* was discountenanced by Trousseau and Traube. It is certainly indicated only in robust individuals with strong hearts and tense vessels; about 12 ounces of blood should be withdrawn; the chief objection to venesection is the difficulty in diagnosis of cerebral hemorrhage. Any physician will admit that he has occasionally bled patients with thrombosis, embolism, syphilis or tumor. In thrombosis and embolism venesection increases the anæmia of the brain, and Cushing has shown that the high tension is a vicarious effort to keep the medulla supplied with blood. The same objection holds to compression of the common carotid. (*d*) *Leeches and sinapisms* are useless. (*e*) The *ice-cap* may be used, but cannot influence the deep circulation in the brain. (*f*) Evacuation of the *bladder* and *bowels* is indicated. Two drops of croton oil will act in an hour or two and in case of failure the dose may be repeated. Croton oil was Hughlings Jackson's only remedy. (*g*) The *diet* should be fluid and administered by the nasal catheter or by rectum. (*h*) The *skin:* decubitus is prevented in practically all cases by care regarding posture and cleanliness. Heat and sinapisms must be used with greatest circumspection because the hemiplegic side is prone to necrosis and inflammation. (*i*) *Symptomatic* treatment of cardiac and of respiratory failure is by champagne or ammonia, but this should not be given until necessary; treatment of restlessness or convulsions is by chloral per rectum or by morphine which theoretically congests the brain but quiets it without detriment.

3. The Reaction.—During the *inflammatory reaction* the treatment is expectant and if possible should be conducted without drugs.

4. The Chronic Stage.—(*a*) Gentle *massage* of the muscles, movement of the joints and alcohol rubs are instituted as soon as the coma subsides. (*b*) *Potassium iodide* is indicated when the reaction subsides. (*c*) As soon as the leg improves, in four to eight weeks, the patient should be encouraged to sit up and walk. (*d*) The *faradic current* is valuable after four weeks and, supplemented with massage of the paralyzed groups and their antagonists, minimizes the contractures. (*e*) *Hydrotherapy:* cool baths or rubs are efficacious except in anæmic or poorly nourished individuals. Warm baths are to be avoided in old people and strychnia in all cases.

V. Cerebral Embolism.—Embolism and thrombosis of a brain artery result in *brain softening* (encephalomalacia), which is next after hemorrhage in importance and frequency and is one of the best known subjects in brain pathology. Brain softening from arterial occlusion is not the "softening of the brain" known to the laity, who give that name to progressive paralysis of the insane. *Encephalo-*

malacia must not be confused with inflammatory softening (*encephalitis*). Softening was first recognized by Abercrombie (1818) and Rostan (1820), while those coming after them usually considered it inflammatory. Virchow's researches on embolism confirmed the views of Rostan and Abercrombie, while Cohnheim elaborated the vascular relations, and Panum, Piltz, Prévost, Cotard and Cohn worked out Virchow's views.

Etiology.—In embolism, Spierling found the kidneys involved in 77 per cent., the spleen in 54 per cent. and the brain in 22 per cent. of cases. (*a*) About 90 per cent. are due to *heart disease.* Most common is *acute endocarditis* or acute exacerbations of chronic valvular lesions, in which the embolism is benign (mechanical) or malign (mechanical and septic). Calcareous fragments from the valves or parts of torn valves, may be thrown into the brain. Of valvular diseases, mitral stenosis produces embolism most frequently because of stasis in the left auricle. Less frequent are cardiac clots formed in the weak hearts of decrepid, cachectic subjects, in myocarditis or in such acute diseases as typhoid. Weakened heart action and slow circulation are never the only causes. (*b*) *Atheroma of the arch and aneurysm* are far less frequent causes, as are (*c*) *lung lesions,* as tuberculous cavities, abscess, gangrene or empyema (after irrigation), tumors, echinococcus, gumma or abscess of the heart. (*d*) In the rarest instances lung tissue (Böttcher), fat embolism from fractures, pigment embolism in malaria (Frerichs) or liver cells in acute yellow atrophy may produce cerebral embolism. (*e*) With a patent foramen ovale, thrombi may reach the brain from the venous system; it has been observed in manual expression of the placenta. (*f*) Most cases occur *in youth* (and middle age) when acute infections are most frequent, as chorea, scarlatina or endocarditis.

Pathology.—(*a*) The *embolus* in fresh cases is gray-red and later becomes paler, drier and more friable. The "riding emboli" straddle the point of arterial branching; they may in rare cases soften and disappear, or by slipping produce another embolism further on (Laborde). Secondary thrombosis may occur at the infarction. (*b*) The *artery* beyond, save where the embolus retracts or softens, becomes a thin, hard, impervious cord. Behind the plug an aneurysm may form. If the embolus is septic, inflammation intervenes in and around the vessel. (*c*) The fate of the *brain tissue* depends wholly on (i) the degree of occlusion and (ii) the collateral circulation. (i) If the stoppage is partial, only stasis, œdema and swelling occur; if total, necrosis results. (ii) The brain arteries are "end arteries," as Cohnheim pointed out, whence complete collateral circulation is unlikely, although the *necrosis* is *rarely as extensive* as the *distribution of the artery occluded,* even where, as in the basal ganglia, the anastomoses are few. There is more chance of collateral compensation at the cortex than at the base and the least collateral compensation occurs in the deep parts of the centrum ovale (Duret and Heubner). The brain tissue is *ischæmic* or its blood is venous (Mar-

chand). The collateral vessels (arteries or venules) allow regurgitation around the focus, the vessels become more pervious and some white and red cells often escape; this collateral fluxion saves the tissue more often than is usually admitted, especially if the heart is strong. With a weak heart or diseased collateral arteries, ischæmic necrosis must follow. Some pathologists hold that shutting off of a large bloodvessel raises the tension in the adjacent (collateral) vessels and thus causes the small capillary hemorrhages often observed; but a marked hemorrhagic infarct is not common, for it is prevented by swelling of the nervous tissue (Weigert). Ischæmia lasting a few hours probably produces necrosis, but microscopic changes are seen only after one or two days; the brain becomes œdematous, gelatinous, grayish, marbled, fusing with the slightly œdematous brain tissue surrounding the focus; necrosis develops rapidly, the brain becomes fluid and pulpy, although its remarkable cohesion is a matter of frequent comment. The *gray matter* contains yellow plaques (*v. i.*); certain convolutions may waste or disappear; the gray and white substance, especially after plugging of the posterior cerebral artery, may wholly disappear, as the hippocampus, lingual and fusiform lobules. The *white matter* often disappears and is replaced by lax cellular tissue, in whose meshes are cystic formations with turbid fluid and flocculi of brain tissue. The meninges are often thickened. Durand-Fardel described three forms of softening: (*a*) The *red softening* is usually fresh and is found most often in embolism; there is capillary extravasation, which lends it the red color; it is irregularly oval or wedge-shaped in basilar lesions; it usually measures but a centimeter or two; it is prominent on section from blood and serum; it is soft, but not fluid in the centre; is mostly located in the cortex or central ganglia and lasts one or two weeks. (*b*) *Yellow softening* follows the red; the yellow color is due to old blood pigment or to fatty change; it is sunken on section, from atrophy of the brain substance; is fairly demarked and may contain fluid, *hydrops ex vacuo;* it may last years and is found especially in the cortex. (*c*) *White softening* may represent (i) a *fresh lesion* about tumors or abscesses or in various cachexias, is sunken below the cut surface and occurs where few vessels are involved and little regurgitation from adjacent vessels occurs; (ii) *lesions* which are several years old, occurring when the blood and fat are absorbed, whence their snow-white appearance; demarkation is often slight, and if diffuse gives rise to the spongy *état criblé.*

Demarkation may occur after weeks, producing a "cyst" of spongy structure and milky contents. More rarely neurogliar increase may lead to shrinking and a *scar* may result. *Microscopically* myelin and fat drops, leukocytes laden with granules and fat, collapsed vessels, relics of necrotic nervous tissue and some increase in neuroglia are observed. Location of embolism is considered on page 937. *Secondary degeneration* occurs when the pyramidal tracts are involved (see page 923).

Symptoms.—As in apoplexy, we consider (1) the *insult* and (2) the

permanent symptoms, to which might be added the symptoms of the causal disease, *e. g.,* those of mitral stenosis, etc.

1. The *insult* is almost always (*a*) *sudden* and *without prodromes* (save when the embolism does not wholly occlude the vessel and symptoms appear only when secondary thrombosis develops). Embolism in a small vessel may occur without symptoms. The patient may be stricken *without coma* and sit "astonished" at the stroke. The insult is caused by *brain anæmia, negative pressure and shock* (the *étonnement cérébrale* of Trousseau and Jaccoud). In some cases (*b*) *coma* occurs, generally *less profound* and *less protracted* than in hemorrhage; irritative symptoms, as (*c*) *convulsions, tonic spasms* and conjugate deviation are *more common;* convulsions are often unilateral, corresponding with the *hemiplegia.* (*d*) All possible *intermediate types* of *focal* disease are observed; for instance, cases with vertigo; tendency to fall to one side; hemianopsia, alexia, hemianæsthesia, motor or sensory aphasia or only confusion, delirium and aphasia (Fagge). (*e*) The *pulse is full but not slow.* (*f*) The *temperature* rarely suffers initial depression and later may rise.

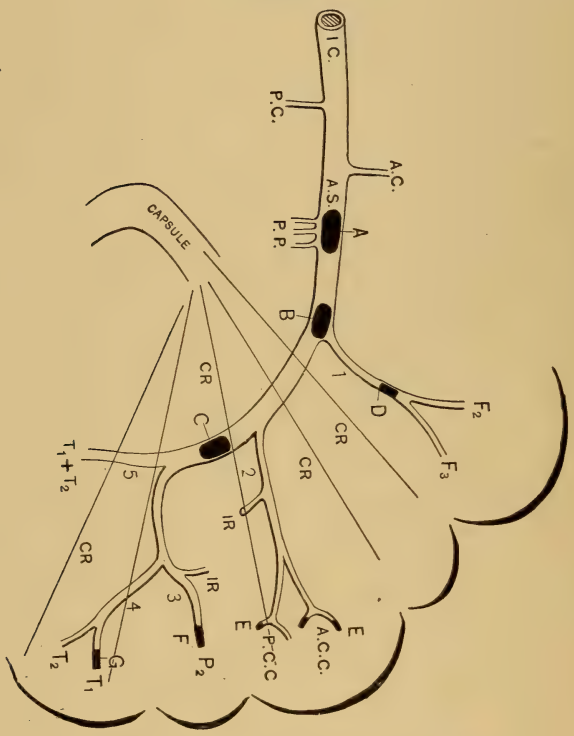
2. The *permanent symptoms* and *secondary degeneration* are the same as in hemorrhage.

Localization of Embolism.—1. The Artery of the Fossa of Sylvius.—This is most frequently involved (80 per cent.) because it is the largest and most direct branch of the middle cerebral which is practically a continuation of the internal carotid. Convulsions occur in 33 per cent., with or without loss of consciousness, and are most frequent when the smaller branches are embolized. High temperature is common. The clinical picture varies with the site of infarction; embolism at A (Fig. 58) where the perforating branches (P. P.) are given off to the internal capsule, corpus striatum and anterior part of the thalamus, must involve (*a*) these structures, and (*b*) the motor and sensory cortex, with *hemiplegia,* fairly complete *hemianæsthesia, motor aphasia* (alexia), *deafness* and *word deafness* (if it occurs on the left side). Convulsions are absent, because the injured internal capsule cannot convey the cortical irritation to the extremities; the large areas at the base and cortex thus rendered anæmic may cause *marked reaction* (Huguenin), sometimes resulting in death. In rare cases only the central perforating branches are involved and the cortex obtains the blood by anastomoses with the posterior and anterior cerebrals. The left Sylvian artery is more frequently involved, because the left carotid comes directly from the arch; 59 per cent. are left-sided (Gelpkel). If the lesion is at (B) (Fig. 58), the motor speech area, F3, which produces motor aphasia, the anterior and posterior motor convolutions (A.C.C. and A.P.C.) which produce hemiplegia or hemiparesis, the parietal convolutions, gyrus angularis and gyrus supramarginalis (P2) which produce alexia, and the temporal convolutions (T1 and T2) which produce deafness or word deafness, are involved. Somnolence and delirium are common; convulsions almost invariably occur, because the internal

capsule escapes. Convulsions and pain are most frequent in embolism of the smaller vessels. Little hemianæsthesia is observed. A lesion at (C) is the most frequent site. It affects the island of Reil (I.R.) in part; cortical convulsions, hemiplegia, monoplegia, sensory aphasia (T1, T2), alexia (P2) and sometimes sensory disturbance occur. It is easily seen how an isolated lesion at (D) could produce motor aphasia; at (E) a monoplegia; at (F) alexia; or at (G) word deafness.

2. Posterior Cerebral Artery.—Embolism of the posterior cerebral (in 2 per cent.) comes from the vertebral artery (and often occurs with embolism of the Sylvian artery); it supplies (*a*) all the

Fig. 58.



Embolism of artery of the fossa of Sylvius.

IC, internal carotid; PC, post. communicating; AC, anterior cerebral artery; AS, art. fossæ Sylvii and 1, 2, 3, 4 and 5, its main branches. CR, corona radiata from cortex to internal capsule. (See text for explanation of lesions at A, B, C, D, E, F or G.)

ventricles, the thalamus, ant. corp. quadrigemina, the geniculate body and the peduncle by its central branches, and (*b*) the mesial aspect of the occipital, part of the parietal and most of the temporosphenoidal lobes. The most common general symptoms are convulsions, coma, vertigo, conjugate deviation and nystagmus; while the most important focal symptoms are *sensory hemianæsthesia, sensory*

aphasia and bilateral homonymous *hemianopsia.* The lesions are sometimes symmetrical.

3. Embolism of the Anterior Cerebral Artery.—Few clear cases are recorded, since the artery originates at a right angle to the carotid.

4. Internal Carotid Artery.—In embolism of the internal carotid (in 4 per cent.) the plug must be large to produce much damage, for if the vessels of the circle of Willis are normal, only transient hemiplegia results. Disease of these vessels or abnormalities in size may produce permanent, alarming or fatal results. Secondary thrombosis is especially dangerous. Sepsis is a frequent factor.

5. Basilar Artery.—Embolism of the basilar artery occurs in 6 per cent.; the clot never fills the vessel, because it first passes the smaller vertebral artery; symptoms occur less frequently at the time of embolism than later when secondary thrombosis occurs. They are convulsions, trismus, paralysis of the third, fifth and seventh nerves, crossed hemiplegia, diplegia, narrow pupils and conjugate deviation. The temperature often falls low and then rapidly rises. Death may follow in two to five days from respiratory failure.

6. Vertebral Artery.—Involvement of the tongue, larynx, lips, pharynx and spinal branch of the fifth nerve result in anæsthesia, dysphagia or other signs of acute bulbar palsy. Hemianæsthesia, hemiplegia and hemiataxia may develop. It is often fatal.

7. Cerebellar Arteries.—This is the rarest of all embolisms.

Diagnosis.—The main points are (*a*) recognition of the *cause,* which is usually *cardiac;* (*b*) absence of *prodromes;* (*c*) *sudden* insult, frequent *convulsions* and infrequent *coma.* (For details and differentiation, see table on page 943.)

Prognosis.—The outlook depends (*a*) on the *primary* disease, being most favorable after acute infections and in youth; (*b*) on the *intensity of the onset* itself, during which death may occur; (*c*) on the *localization;* the initial symptoms may improve; the outlook is more favorable than in hemorrhage. Psychical symptoms are cortical in origin, while the vegetative or trophic are due to a lesion in the basal ganglia; basilar and vertebral embolisms are most unfavorable. (*d*) Recurrence is possible, although it is less likely than usually stated. (*e*) The condition of *other organs,* as the vessels of the circle of Willis, the strength of the heart muscle, etc., determine the prognosis.

Treatment.—(*a*) The *insult* is treated as in hemorrhage in regard to diet, keeping the patient quiet and general measures. (*b*) All derivative measures, as *drastics* and venesection, are *absolutely to be avoided.* (*c*) The heart must be *steadied* and *stimulated,* if hemorrhage is clearly excluded, with a view first of relieving the brain ischæmia by a strong collateral circulation and second of strengthening the heart to avoid recurrence. Digitalis acts too slowly, and camphor, strychnine and ammonia are preferable. (*d*) *Convulsions* should be treated by chloral given by rectum, avoiding alcohol and supporting the heart. The administration of more than a dram of

chloral in three hours is very dangerous; gr. x to xv should be given forty-five to sixty minutes apart. (*e*) The *after treatment* should be followed out as in the corresponding stage of apoplexy.

VI. Cerebral (Arterial) Thrombosis.—Thrombosis is more common than embolism. Once it was thought that fibrin was thrown out of the circulating blood by a ferment. Virchow held that slowing of the current was the chief cause, while Zahn, Weigert and others attributed great importance to coagulation necrosis of the leukocytes. Bizzozero, later also Ebert and Schimmelbusch held that the blood plaques played a large part in coagulation. Brücke taught that the living healthy vessel prevented coagulation.

Etiology and Pathology.—1. Changes in the Vessel Wall.—(*a*) *Arteriosclerosis* (*q. v.*) *is the chief cause of this most important group.* It affects the basal arteries especially and usually several vessels. Blood plaques are deposited on the rough intima and in the tortuous, inelastic, calcareous, fatty vessels, with their unequal calibre; layer after layer of fibrin is deposited, aided by local slowing of the blood stream and by weakening of the heart. Thrombosis may be local or extensive. Tumors, abscesses and traumatic meningitis may initiate thrombosis. Brain atheroma usually occurs with general atheroma (see Arteriosclerosis). The vessels most involved are, in order of frequency, the carotid, middle cerebral and its Sylvian branch, basilar, vertebral and posterior cerebral. (*b*) *Syphilitic endarteritis* occurs in young subjects, 84 per cent. being found between the twentieth and fortieth years. The initial proliferation may of itself, without thrombosis, obliterate the vessel lumen (see Brain Syphilis). The pathological sequence is the same as in embolism; ischæmia, necrosis (softening) and loss of function occur, save that in gradual thrombosis the focus is less homogeneous than in the sudden lesion of embolism. The foci are often multiple, for example, in the Sylvian artery and in the posterior cerebral vessel of the opposite side.

2. Changes in Heart.—Sudden cardiac insufficiency in arteriosclerosis may precipitate thrombosis, as brought out especially by Kolisko. An acute infection in the aged, low arterial tension from exhaustion, grief and similar causes may precipitate encephalomalacia.

3. Changes of the Blood.—This constitutes the smallest class of cases. Burns, poisoning by carbon monoxide and illuminating gas, gout, chlorosis, leukæmia, metallic poisons, jaundice and marasmus in very young children or in extreme old age may cause thrombosis. Mechanical factors, acute infections and heart weakness are frequent factors in this class. Malarial thrombosis might possibly be included. Thrombosis affects the smaller vessels and is usually multiple.

Symptoms.—In some cases thrombosis is found at necropsy without previous symptoms ("latency").

1. Prodromes.—These are frequent and may appear hours, days, and even weeks or months before focal symptoms develop. The most common are headache, which is severe in the syphilitic variety, ver-

tigo, tingling, numbness and weakness in a limb or in one-half of the body, disturbance in sight, mental changes, poor articulation, irritability, loss of appetite for work or change in character.

2. Onset.—The onset varies; (*a*) an *acute onset* with insult is not frequent. If consciousness is lost, the coma is less deep and protracted than in hemorrhage; a "stroke" indicates thrombosis in one large or in several vessels. Conjugate deviation, flaccidity and paralysis are frequent during the coma. Consciousness is usually preserved in the syphilitic type. With acute onset the later course is usually slow. (*b*) The *onset is usually gradual.* Layer after layer is added to the clot but strengthened heart action may for a time help the collateral circulation. When the heart weakens, the clot increases and the collateral blood supply decreases, so that, after alternating advance and regression, the final thrombosis with irreparable ischæmia and softening results. The cardiac strength and the number and size of the vessels involved determine the acuity or chronicity of the course. *Reaction* may be marked, especially in the aged. (*c*) In the *chronic* or long *latent* variety, prodromes are absent, psychic alteration is apparent and focal symptoms are usually developed.

3. Chronic Stage.—In the chronic stage focal symptoms, as monoplegia, hemiplegia, hemianopsia or aphasia, are common. Apoplectiform seizures mark the advance of the process in new vascular areas. The motor or sensory paralysis, at first transitory, is prone to recur and presents much the same picture as hemorrhage and embolism in their chronic stage.

Localization.—It is impossible to determine the localization when multiple foci exist, as in the Sylvian artery of one side and the posterior cerebral artery of the other; to the few reported cases Senator adds one in which left-sided hemiplegia was associated with aphasia. The favorite sites are the cortical branches of the Sylvian artery and the branches of the posterior cerebral vessels.

1. Sylvian Artery.—The most common cause of this localization is syphilis or atheroma. When the *main trunk* is involved, the same symptoms appear as in embolism, *viz.,* hemiplegia, aphasia, hemianæsthesia and hemianopsia with this modification; the *focal symptoms are more severe* in thrombosis, because other vessels are somewhat diseased; the *general symptoms are less severe.* Prodromes appear first, as vasomotor changes and tingling, then hemiplegia possibly with delirium or loss of consciousness; the symptoms may improve, then there may be recurrence with aphasia and coma. The course is a "step-like" progression and regression. *Monoplegia* is more common than in embolism, because thrombosis seeks the cortical vessels, whence the face and arm may be affected, aphasia may exist alone, or alexia, mind blindness or deafness. When the basal arteries are diseased, the vegetative functions are involved, that is, trophic changes with involuntary evacuations occur (*gâtisme*).

2. Anterior Cerebral Artery.—This artery is rarely totally occluded. A clot beyond the anterior communicating vessel often

produces crural monoplegia with some paresis of the arm. Very *gradual anæmia* produces *wasting* without softening; sudden anæmia produces softening. The brain may atrophy three to seven ounces, with senile dementia, mania or hypochondriasis and though not strictly softening, its close arterial connection is obvious. The frontal lobes present the "worm eaten" atrophy, with abundant *hydrops ex vacuo.*

3. Posterior Cerebral Artery.—Thrombosis in this artery is fairly common and may result in hemiplegia alternans oculomotoria, hemihypæsthesia and hemianopsia with irregular defects in the crossed fields; optic changes vary accordingly as the optic radiation, cuneus or other parts are affected.

4. Basilar Artery.—If the thrombosis is acute the symptoms are like those of embolism. If slow, ophthalmoplegia may develop which is rarely lasting, or other motor paralyses, hemiplegia with alternating palsy of the fifth, sixth and seventh nerves.

5. Vertebral Artery.—(*a*) *Acute bulbar palsy,* with stormy symptoms, cannot be distinguished from embolism; or (*b*) *subacute bulbar palsy,* with *dysphagia, paralysis of the larynx,* disturbed articulation, hemianæsthesia, *homolateral ataxia, contralateral hemianalgesia* or hemiplegia with crossed paralysis of the tongue may result from this localization.

Diagnosis and Differentiation.—The *prodromes, gradual onset* and *cortical location* are most distinctive, although the analysis may be greatly confused and differentiation of complicating conditions in the heart and kidneys may be wholly theoretical or impossible. (See Table, page 943.) Thrombosis may be confused with *brain tumor,* in which the course is slower, stabile focal symptoms less frequent, headache, vomiting and convulsions more frequent than permanent paralysis; and choked disk and Jacksonian epilepsy are frequent. In this connection, we may consider *hemiplegia in the aged* and Marie's *les lacunes de désintegration cérébrale; degeneration clefts* occur, chiefly in the large basal ganglia, sometimes in the internal capsule or pons (25 per cent.) and but seldom in the centrum ovale (13 per cent.); there are usually several foci. Marie holds that the lesion is a rarefaction (cavity formation) of the brain tissue due to chronic sclerosing *encephalitis, without vessel occlusion.* It is said to cause 90 per cent. of "insults" in the aged, which occur usually with *partial paralysis;* sometimes with aphasia, dysarthria or dysphagia; rarely with loss of consciousness; and never with sensory disturbance or contractures. The gait is the *démarche à petits pas.* The prognosis is fairly good; great improvement may occur and the patient may live eight to ten years, though hemorrhage may intervene.

Prognosis.—The prognosis depends on (*a*) the *cause;* it is unfavorable in *arteriosclerosis,* because collateral vessels are probably diseased and the cause is progressive. If the other vessels are sound, enormous disease may be compensated, as in Kussmaul's case of occlusion of both the subclavian and carotid arteries. In *recent syphilis* the prognosis is more favorable, though old specific thrombosis is

HEMORRHAGE.	THROMBOSIS.	EMBOLISM.
(1) *Predisposing etiology.* Before third year (glioma); 80 per cent. after fortieth year.	After eightieth year more frequent than hemorrhage. Twentieth to fortieth year syphilis (of which 84 per cent. occur between these years).	Youth; hemorrhage practically never in youth.
Miliary aneurysms; atheroma and cardiac hypertrophy.	Weak irregular heart; fevers, cachexia. Atheroma, especially syphilis, alcoholism, plumbism.	Endocarditis (rheumatism, sepsis), especially mitral. Weak, fatty, dilated heart. In heart disease Eichhorst found in 60 per cent. hemorrhage and in 40 per cent. embolism.
Heredity. Nephritis; cause in 33 per cent. "granular kidney."	Rare. Rare.	Rare Rare.
(2) *Attack precipitated* by heavy meal, acute alcoholism, high art. tension (excitement, effort, shock).	Low arterial tension; by sleep, grief, exhaustion, fatigue.	By exertion, dislodging clot.
(3) *Prodromes* unusual, unless from causal arteriosclerosis.	Very common, mental irritability, weak memory, headache, vertigo, numbness, tingling, weakness in extremities.	Absolutely none.
(4) *Insult:* Usually sudden; without prodromata; long duration (fatal if more than two days); Cause; positive pressure producing brain anæmia.	Rarely acute onset (unless in a large vessel); usually gradual, especially in senile softening. If an insult, focal and general symptoms usually appear during the coma.	Sudden, shorter and less severe than in hemorrhage. Cause; negative pressure. Slow insult only with secondary thrombosis.
(5) *Intra-cranial pressure:* "Pressure symptoms" present, polyuria, albuminuria, glocosuria, red face, beating arteries, Cheyne-Stokes breathing; slow, hard pulse is the fundamental difference (Brissaud); bruit de gallop speaks strongly for hemorrhage. Irregularity, if combined with strength not against hemorrhage. Pulse may be fast (Brissaud).	None. Pulse soft, weak.	None (pallor rather than congestion).
(6) *Temperature:* Rectal temperature usually depressed at first, may fall till death. May rise later (inflammatory reaction). Early rise (pons, medulla, ganglia). Sudden rise of bad omen; rises before death.	Initial depression, usually lasting. Seldom temperature unless lesion in pons. More probably early reaction. Especially in the aged.	Temperature may rise high at once without bad omen. Temperature no absolute guide; occurs especially when large trunk embolized.
(7) *Coma:* More frequent, greater in degree, longer in duration, usually complete. If lesion seems small, coma speaks for hemorrhage. Deep and prolonged speaks for hemorrhage and usually fatal if over twenty-four hours.	Less frequent and shorter. Recovery possible after several days of coma. None in syphilitic thrombosis. If lesion seems large, consciousness speaks for softening. If coma, usually focal symptoms are then seen Coma in large bilateral lesions	No coma usually.
(8) *Paralysis,* sudden, complete, hemiplegic, non-progressive after first seizure. Foot gains more rapidly than hand. Persistent aphasia exceptional. Spasmodic laughing and weeping common.	Gradual, incomplete, monoplegia, hemianopsia; repeated, often symmetrical paralysis. Converse Aphasia common Absence of focal symptoms rather more common (central ganglia). Laughing and weeping exceptional.	More like hemorrhage. Monoplegia rare. In 63 per cent it is in the left side of the brain. Fairly common.
(9) *Convulsions:* Usually generalized; very rare from cortical irritation or in corpus striatum; post-hemiplegic are rare.	Less than in embolism. More localized, Jacksonian epilepsy speaks for softening. May recur. Post-plegic frequent.	More common (33 per cent.) than in hemorrhage or thrombosis (lesion cortical).
(10) *Sensation:* Anæsthesia usually transient.	Paræsthesia persists and more marked. Cerebral pains (hyperæsthesia dolorosa).	Ditto.
Postplegic chorea, trembling, athetosis, common.	Less common.	Less common.
(11) Atheroma retinal arteries, hence retinal hemorrhage (not significant unless large). Aneurysms very rare but speak strongly for hemorrhage. Retinitis albuminurica.	Arteriosclerosis.	Optic retinitis sometimes from endocarditis (Broadbent and Mackenzie). Occasionally embolism in retinæ.
(12) *Secondary reaction;* not much.	Greater secondary reaction.	
Psychical symptoms; far less.	Often greater; intelligence, memory aphasia, word deafness, etc.	Very seldom; after attack may be some delirium.

incurable. The younger the patient the better is the outlook. (*b*) *The onset;* a brusque beginning is unfavorable because most cases of this class die. Coma indicates thrombosis in large vessels or in both hemispheres and is therefore ominous. If there is no insult the focal symptoms are usually irreparable, *i. e.,* there are no indirect symptoms. The first seizure does not usually threaten life. (*c*) The *location and extent of the lesion.* The number of vessels involved is obviously important. In Sylvian thrombosis involving the branches to the central ganglia, cachexia and more trophic disturbance occur and the patient dies in four to six weeks with involuntary evacuations and bed-sores (Marie). Cortical lesions, on the other hand, are more prone to produce psychical alteration. Basilar or vertebral thrombosis is directly dangerous. (*d*) The *focal symptoms* are likely to *persist* and *recur.* Ophthalmoplegia may regress. (*e*) The condition of other organs, above all that of the *heart,* is of prognostic import.

Treatment.—(*a*) The *general treatment* is the same as in hemorrhage and embolism. (*b*) The *causal treatment* relates to the iodides, which are of value in atheroma and are specific in early syphilis. (*c*) The *heart* must be stimulated with strychnine, digitalis and strophanthus; absolute quiet in the prone position and free administration of food and fluid by rectum and by nasal catheter are indicated. Drastics and all depleting measures must be avoided. On the heart devolves the entire possibility of collateral compensation. (*d*) In the *period of reaction* alcohol should be avoided, for it accentuates the inflammation. For delirium and headache, bromides are most useful; no cardiac depressants, chloral or coal-tar products may be used. (*e*) The *chronic stage* is treated as in hemorrhage. At all times catheterization must be conducted antiseptically and great care exercised against bed-sores.

VII. Intracranial Aneurysms.—Etiology.—Aside from miliary aneurysms, aneurysms may occur in the large brain vessels. They are seen in 0.2 to 1 per cent. of autopsies. They are more frequent in males (60 per cent.) and in middle age, although they may visit either extreme of life and a few more (53 per cent.) occur before than after the fortieth year. The causes are those of aneurysm elsewhere (*q. v.*); they are (*a*) *atheroma* in whose causation alcohol is particularly important; (*b*) *trauma,* initiating arteritis, especially in the internal carotid, as after basal fracture (Killian); (*c*) *syphilis,* especially productive of basilar aneurysm, which often occurs within a year after infection; (*d*) *embolism,* the most frequent cause in early life. *Endocarditis* is the usual cause when trauma and syphilis are excluded (Lebert and Church). The embolus produces arteritis from which the vessel dilates. The clot may later disappear.

Pathology.—Location.—The statistics of Lebert, Durand, Bartholon and Osler combined show the following frequency; middle cerebral, 29 per cent.; basilar, 26 per cent.; internal carotid, 14 per cent.;

anterior cerebral, 8 per cent.; anterior communicating, 6 per cent. They are rather more frequent on the left side, in 20 per cent. are multiple, are usually sacculated, of the size of a pea or walnut (rarely large as an egg) and are genuine aneurysms involving all coats.

Symptoms.—Symptoms may be absent until rupture occurs. They are (*a*) *general,* as *headache,* which is the most common sign, or *convulsions,* which result from aneurysm of the Sylvian branch; a systolic murmur, neuralgia or epileptiform seizures may be noted. (*b*) They may be *local;* according to their location, hemianopsia, paralysis of the ocular nerves, hemiplegia, optic neuritis (extension of inflammation from the sac), aphasia and symptoms referable to the pons or medulla may be noted. *Rupture* occurs in 75 per cent.; (i) into the meninges or (ii) into the brain substance or ventricles, from which death rapidly results; healing has been observed.

Diagnosis.—*Rupture* is often the first symptom and differentiation from hemorrhage is therefore necessary. A general cerebral hemorrhage in a young individual with a history of trauma, heart disease or syphilis is rather suggestive of aneurysm, especially when the location is basilar. If symptoms have been present they are often those of a basal tumor; syphilis (*q. v.,* Cerebral Syphilis) and trauma are causes of tumor and aneurysm, but iodides and mercury usually are not beneficial in aneurysm. The murmur is suggestive but is rare and may be found in tumors which compress vessels or in very vascular brain tumors. The location of the aneurysm is important; they are most often embolic in the Sylvian and specific in the basilar artery. Loss of sight in an eye, sometimes with impairment of smell, rather indicates aneurysm of the anterior cerebral or, if the eye muscles are paralyzed, of the internal carotid artery. Oculomotor paralysis without loss of vision suggests aneurysm of the posterior communicating artery. Some cases closely resemble arterial thrombosis.

Treatment.—The indications are the same as in aortic aneurysm (*q. v.*), *viz.,* rest and iodides. Surgical intervention is occasionally opportune, as ligation of the carotid.

VIII. Sinus Thrombosis.—The marantic form was known to the oldest authors, while Abercrombie, von Dusch and Bright described the pyæmic form. Lebert (1854) diagnosticated the first case and with Tonnelé built up its clinical recognition.

Etiology and Pathology.—There are two varieties, (1) the marantic and (2) the inflammatory types.

1. *Marantic thrombosis of the sinuses and veins* is the *primary non-inflammatory* type, due to altered blood states and weakened circulation. It occurs (*a*) in *children* mostly, especially in the first six months of life and after acute infections, particularly infective *diarrhœas;* (*b*) less frequently in the *aged* from weak heart and venous stasis; (*c*) in *cachexia,* from cancer, gout and tuberculosis; (*d*) in the anæmia of *pregnancy* and in *chlorosis;* (*e*) *compression* of veins or sinuses, as by tumor, is rare. Its most frequent *site* is the superior longitudinal sinus, especially in children, because the sinus is fixed,

its lumen is triangular and irregular, crossed by trabeculæ and compressed by the Pacchyonian granulations; the tributary veins empty against the current of the sinus, the veins ascend and their stream is very weak. Gowers believes that thrombosis in veins entering the superior longitudinal sinus may cause hemiplegia in children. Experimentation shows that simple slowing of the current is not sufficient for coagulation and that the vessel wall must be altered; in chlorosis there is fatty degeneration of the endothelium. In the aged, marantic thrombosis rather elects the cavernous or transverse sinus. The older the antemortem clot the lighter is its color and the more laminated and adherent it is to the sinus wall, wherein it differs from the reddish, loose and separable postmortem clot. The marantic clot may be total, occluding the vessel, or partial. It may be short or fill the whole sinus and enter the tributary veins which are then seen as firm cords over the hemispheres.

2. The *inflammatory thrombosis, thrombophlebitis,* is *secondary* to disease near the sinus; as (*a*) *ear disease,* chiefly chronic, sometimes acute, suppurative otitis media, *caries* of the bone, sometimes syphilis, tuberculosis or cholesteatoma. Infection from the ear reaches the sinus by (i) infecting the mastoid cells, which infect the transverse sinus; (ii) by the passage of the virus from the middle ear to the sinus by infecting small communicating veins (Politzer) or (iii) its passage along the petromastoid canal (Voltolini). (*b*) *Acute infections,* with otitis media; (*c*) *sepsis,* puerperal fever, pyæmia; (*d*) *trauma,* tuberculous *meningitis,* erysipelas, disease of the orbit, nose and face. The most common *sites* are the transverse, petrosal and cavernous sinuses, whence it may extend to their branches. The clot is puriform, fœtid, soft, discolored and contains pyogenic organisms; the process is frequently associated with meningitis and brain abscess.

In both types *brain changes may result,* as œdema, swelling or softening of the cortex. Most frequently the thrombosis is bilateral; suggillations in the pia, bloody imbibition of the brain or reddening of the cerebrospinal or ventricular fluid may occur. Pulmonary infarcts occur in 50 per cent. and are simple or septic according to the type of thrombosis.

Symptoms and Diagnosis.—Clinical signs may be absent or obscured by the primary affection. Diagnosis depends upon signs of (1) extra- and (2) intracranial venous stasis.

1. Extracranial Stasis.—(*a*) In thrombosis of the *superior longitudinal sinus,* stasis in the nasal veins is often shown by *epistaxis* (connection through the foramen cæcum); this was first observed by von Dusch. It is an effort of nature to deplete the cerebral congestion. Gerhardt observed *cyanosis* in the anterior frontal veins and *œdema,* especially in children, in the temples between the great fontanelle and ear (by way of the emissaria Santorini through the foramen parietale). Œdema is often absent in chlorotic thrombosis. (*b*) In thrombosis of the *cavernous sinus* stasis develops in its tributaries, producing *exophthalmos, œdema of the lids* and conjunctiva (vena

ophthalmica); *retinal stasis,* more rarely choked disk or retinal thrombosis, results from congestion of the vena centralis retinæ. Sometimes œdema of the forehead results from stasis in the vena facialis ant. Epistaxis is rare. (*c*) In thrombosis of the *lateral sinus* a hard, tender *œdema behind the ear* (Griesinger) and *in the*

FIG. 59.

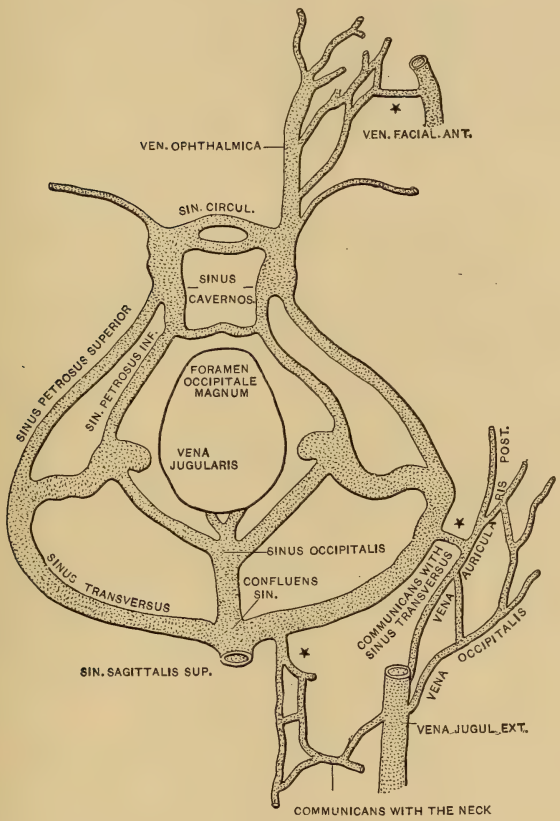


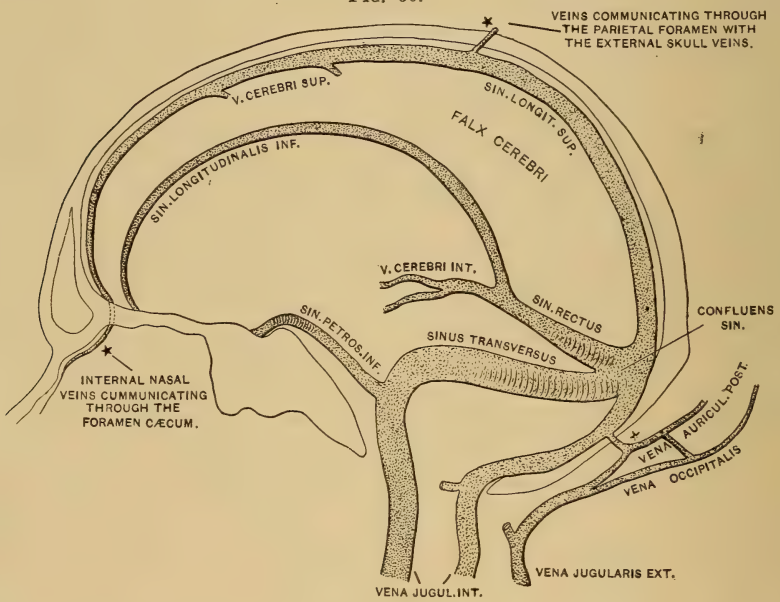
Diagram showing the connections* of the transverse and cavernous sinuses (Leube).

neck (communicating with the posterior auricular vein) occurs in over 50 per cent. of cases. Occasionally the *swollen thrombotic internal jugular vein* may be felt. The *external jugular frequently collapses,* or if the internal jugular is thrombosed it becomes overfilled (Gerhardt). In 33 per cent. the disk is hyperæmic and in another 33 per cent. there is choked disk from complicating abscess or meningitis (Jansen). (*d*) In thrombosis of the *sinus petrosus* Gowers questions whether any signs are distinctive.

2. INTRACRANIAL STASIS.—Progressive clouding of the mind, delirium in adults, convulsions in children, conjugate deviation of the eyes and head, stiff neck, vomiting, headache, irregular pupils, nys-

tagmus or strabismus are noted. Hemiplegia and sensory symptoms are occasional. Bilateral symptoms, as paraplegia, are due to the involvement of the veins in both hemispheres or more often to meningitis. Bouchut thought that antemortem convulsions in children were often thrombotic in origin. Many of these symptoms resemble the *hydrencephaloid* (Marshall Hall) of brain anæmia. The fontanelles, sunken at first in infantile diarrhœa, may later become prominent (increased cerebrospinal fluid). Sometimes in chlorotic thrombosis the process may extend to the vena Galeni magna, producing hydrocephalus internus and resulting in early stupor, coma and death. Irritation or paralysis of the third, fourth and sixth nerves may occur because of their intimate relation to the cavernous sinus; involve-

FIG. 60.



Leube's diagram showing the connections of the superior longitudinal sinus with the external veins.

ment of the first branch of the fifth nerve may result in ophthalmia neuroparalytica. Rarely is there paralysis of the hypoglossus and spinal accessory.

OTHER SYMPTOMS.—Other symptoms are those of the causal affection. The temperature is often normal in the simple variety. The pulse is slow at first and rapid later. The thrombophlebitic variety is usually attended by fever, chills, sweats, diarrhœa and other septic manifestations.

DIFFERENTIATION.—Differentiation from meningitis (*q. v.*) and brain abscess (*q. v.*) is often impossible; the cause, the extracranial stasis and evidences of sepsis are of diagnostic importance. Körner held that metastases occur in the lungs from sinus thrombosis and in the bones and joints from mastoid caries.

Prognosis.—The prognosis is almost always unfavorable; death occurs in the second week, or possibly later. A few recoveries from both types are on record. Local atrophy of the brain has resulted after recovery from the marantic type.

Treatment.—*Prophylaxis* relates chiefly to the otitic variety, because discharges from the ear are much too lightly regarded by the laity. In *marantic thrombosis* stimulation and avoidance of constriction of the neck are the chief indications. In *septic types* operation is indicated; recovery is most likely when there is early diagnosis and early operation. Opening and packing the lateral sinus was first performed by Zaufal in 1884. He also recommended ligature of the internal jugular veins, which was first performed by Thersley and Lane in 1888. Recovery occurs in 50 per cent. of operated cases and in 72 per cent. of Macewen's series. Recovery has occurred even with complicating lung abscess (Ballance). Gravitating pharyngeal abscess and extradural foci also necessitate surgical interference.

IX. Infantile Cerebral Paralysis.—Cerebral infantile palsy is classed under circulatory diseases because of the frequent etiological hemorrhage, embolism and thrombosis. Pathologically it is sometimes described under encephalitis, sclerosis or porencephalia. Clinically it is variously classed according to the dominant syndrome, hemiplegia, diplegia, Little's disease, paraplegia, hemiathetosis and athetosis. The disease is a clinical picture, but with varying etiology and pathology. Osler, Sachs, Strümpell, Freud and Rie have contributed most valuable monographs.

1. The Hemiplegic Form.—Etiology.—It is equally frequent in either sex. Most cases occur in the *first year.* Gowers finds 60 per cent. in the first two years, 75 per cent. in the first three and 88 per cent. in the first five years of life. *Infections,* as scarlatina, measles, less frequently variola, pertussis and diphtheria are apparent, and heredity, maternal syphilis and trauma are doubtful causes. The causes may operate before, during or after birth. The pathology is considered under the next type.

Symptoms.—(*a*) Initial symptoms, present in 66 per cent., are severe in character, consist of *fever,* vomiting, *convulsions,* delirium and coma, and last from a few hours to several days. Convulsions, present in 50 per cent., are often unilateral and are usually followed by coma. In 33 per cent. of cases the onset is insidious. (*b*) The *paralysis* (hemiplegia) usually develops when consciousness returns. It is more frequently right- than left-sided. The lower face is involved, but usually soon improves, the leg improves more than the arm and hand, and the paralysis, at first *flaccid,* soon becomes *spastic* and permanent. Sometimes the eye muscles are involved and hemianopsia may be noted. The tactile and stereognostic sense may be somewhat reduced, but sensation is slightly implicated, because the other hemisphere compensates. (*c*) The *residual symptoms* are the same as in adults, with some exceptions; the paralysis is

spastic (spastic infantile hemiplegia, Heine) but also *atactic* and *athetotic.* Because it occurs in the young growing body and brain, marked *trophic changes or, at puberty, retarded development* appear, *intellection is retarded or actual idiocy results* and *epileptiform seizures* are common. *Contractures* are frequent, the *reflexes* are increased and the gait is somewhat impaired, the foot dragging. In rudimentary (*formes frustes*) or benign cases the paralysis may almost disappear and, after months or years, may be so replaced by epileptic seizures, hemiathetosis, hemichorea, hemitremor (described under hemorrhage) or hemihypertonia (rigidity with but little paralysis) that the paresis readily escapes recognition. *Aphasia* is rarely lasting; (*a*) *reflex aphasia* is very common in childhood from fever or digestive difficulties; (*b*) the brain readily compensates in childhood; (*c*) permanent aphasia indicates great or total loss of intelligence. *Trophic changes* are in part a moderate atrophy and in part lack of later development, though seldom so marked as in the spinal type of paralysis. They are noted in the extremities, body, sometimes the face and in the skull, which often thickens; they are most common in porencephalia (*v. i.*). *Epilepsy* increases in frequency after the paralysis and is rated at from 13 per cent. to even 66 per cent.; it occurs (*a*) at the onset, (*b*) with the paralysis or (*c*) a year or two later; it is not commensurate with the degree of paralysis but is more marked in rudimentary palsy; it may be slight like a vertigo; it is most often like the Jacksonian type, but may end in total loss of consciousness. In infantile cerebral paralysis there are less violence, initial cry, trauma, foaming at the mouth and fewer involuntary evacuations than in ordinary epilepsy. After a decade or more it often becomes very marked, although it may disappear between the fortieth and fiftieth years.

Diagnosis.—The chief differential interest centres in its separation from the spinal type (poliomyelitis):

	Cerebral Type.	Spinal Type.
Tendon reflexes.	Increased.	Decreased or abolished.
Muscular atrophy.	Only after a long time.	Rapid.
Electrical reaction.	Normal, or little quantitative decrease.	Partial or total reaction of degeneration.
Distribution of paralysis.	Hemiplegic—diplegic.	Chiefly monoplegic.
Intelligence.	Often involved.	Usually not.
Epilepsy and hemichorea.	Common.	Rare.

2. The Double Hemiplegic or Diplegic Form.—Etiology.—(*a*) *Prenatal causes,* apparent in 20 per cent., include fright, trauma, mal-

nutrition or syphilis. These causes seem most common in cases which develop athetosis. (*b*) *Anomalies of labor,* suggested by Little and proven to be a factor by Sarah McNutt, include difficult or long labor, malpresentation, resistant maternal tissues, twin pregnancy, version, cord prolapse and instrumentation, although delivery by forceps is less important than the causes calling for their use. *Asphyxia* is an important etiological factor. Premature delivery is also causal, possibly because the soft skull is injured (Gowers) or because venous stasis occurs from precipitate delivery. (*c*) *Extra-uterine causes* include trauma and acute infections.

Pathology.—The pathology presents many obscure points. The *initial lesion* cannot always be determined. (*a*) In the *traumatic class, meningeal hemorrhage* causes 66 per cent. *of the diplegic cases;* Sarah McNutt found hemorrhage spreading over the motor cortex on both sides. It is promoted by *asphyxia* and the sliding of the skull bones of one side under or over those of the other side; hemorrhage is often basal in head presentations and on the convexity in the after-coming head. The blood is absorbed slowly, especially if the brain is lacerated, resulting in atrophy of the motor areas of both sides; hemorrhage rarely results from hereditary syphilis. (*b*) *Vessel disease* (18 per cent.) may be arterial (thrombosis, embolism or hemorrhage) or venous (in the cortical veins, Gowers). (*c*) Strümpell advanced the views of Vizioli and Marie that *acute encephalitis* in the motor cortex (poliencephalitis) causes the acute onset, an argument largely based on analogy (poliomyelitis in children), though seldom verified.

Chronic Lesions.—The most important are diffuse lobar sclerosis and porencephalia, which are peculiar to infantile brain palsies: (*a*) *Diffuse lobar sclerosis* and atrophy of the brain occur in 55 per cent. (Osler). The meninges are normal or somewhat thickened. About the atrophic areas and in the ventricles the cerebrospinal fluid is increased. One or both hemispheres may be involved but usually unequally. The lobe affected is shrunken, hardened and the white matter is decreased, while the gray seems increased. The convolutions are wasted and smooth or "worm eaten." The nervous tissue is wasted, the neuroglia increased and the bloodvessels tortuous, somewhat thickened and their perivascular lymph spaces widened five- or six-fold. The nature of the initial lesion is disputed; it is thought to be encephalitis (Bourneville), poliencephalitis (Strümpell) and *embolism.* (*b*) *Porencephalia* (Heschl, 1859) consists of loss of brain substance and cavity formation. It occurred in 26 per cent. of Osler's cases. The nature of the initial lesion causing porencephaly is a matter of doubt; it is ascribed to agenesis, encephalitis or meningo-encephalitis, trauma during labor or to the uterus during pregnancy, or *anæmic necrosis* (Kundrat); the latter is the most widely accepted view. It is most frequent in the distribution of the Sylvian or occipital vessels. Most often it is congenital and then the convolutions radiate from the defect, while in those cases resulting *post*

natum, the convolutions stop short of the defect "as though cut off." Bourneville and Sollier hold that the congenital type communicates with the lateral ventricle, while the acquired form does not. Sixty-six per cent. are bilateral and somewhat symmetrical. (*c*) The *hypertrophic nodular sclerosis* described by Bourneville consists of multiple, discrete, prominent, often umbilicated nodes the size of a quarter or half dollar. They are overgrowths of the neuroglia.

Symptoms.—Diplegic cases are half as frequent as the hemiplegic. Freud describes the following forms: (*a*) *General rigidity,* or Little's disease, 33 per cent. of which are caused by asphyxia. The main characteristics are (i) *predominance* of the *rigidity* over the paresis, and (ii) *predominance* of the *leg paresis* over that of the arms, the converse of adult cerebral paralysis (hemorrhage occurs most often over the leg centres). Convulsions often usher in the symptoms.

FIG. 61.

Spastic paraplegia, cross-legged progression. (Dercum.)

which appear after birth or a few months later. The rigidity is general but most conspicuous in the *flexors and adductors,* leading to unusual postures. The face is slightly involved, the maximum manifestation being spasm of the mouth; in rare cases the child cannot nurse or swallow. Speech is slow in development and stuttering is common. In 33 per cent. the head cannot be lifted or the trunk

flexed, due to involvement also of the uncrossed pyramidal tracts. The arms may be but slightly paretic, but the legs suffer extensor spasm on movement and the toes turn down (talipes equinus or equino-varus). The child learns to walk late, if at all, because of the adductor rigidity, which may even cause crossing of the legs. The reflexes are greatly increased. The intellect is somewhat affected in 66 per cent. Nutrition in the paretic members is usually intact. Later epilepsy is uncommon. The paresis may regress and the mind improve, but the legs remain spastic. (*b*) *Paraplegic rigidity* resembles type (*a*) except that the arms are but little or not at all affected and the legs alone are rigid. The name Little's disease is also given to this form. Strabismus is common (40 per cent.). Premature delivery is a factor in 50 per cent. of cases. (*c*) *Paraplegia* is rare and is referred to extra-uterine causes or infantile infections. The legs are rigid and paralyzed. In severe types there may be strabismus and imbecility. (*d*) In *bilateral spastic hemiplegia* (spastic diplegia) the symptoms may vary on the two sides. Mental degeneration is frequent; nystagmus, convergent strabismus (25 per cent.), bulbar symptoms and speech involvement (60 per cent.) are frequent. (*e*) *General infantile chorea* is also called "choreatic diplegia"; asphyxia is a factor in 25 per cent. Its clinical characters are (i) paralysis, (ii) rigidity, (iii) chorea-like movements which are irregular, usually slight in degree and wide in distribution. (*f*) In *bilateral athetosis* the mind is less involved than in the preceding types. The paralysis may precede it or it may be primary.

Forms resulting from premature delivery are less subject to convulsions, athetosis or mental changes and are more regressive than those due to birth trauma or later acquired lesions. (See Diseases of the Spinal Cord for the allied Family or Hereditary Forms, as Hereditary (Friedreich's) Ataxia.) In all forms *epilepsy* may (i) replace the regressing paralysis; (ii) remain so prominent that the paralysis is overlooked; (iii) paralysis may appear only after years of epilepsy, or (iv) may not appear at all, "cerebral paralysis without paralysis."

Diagnosis.—The diagnosis cannot always be made at once, although later, characteristic symptoms develop and leave little diagnostic difficulty. Separation from the family spastic paralysis (*q. v.*) may be difficult or impossible. As a rule, aside from compression of the cord, confusing spinal cord disease is very rare in children.

Treatment.—Therapy is most unsatisfactory. In the initial stage convulsions may be treated by chloral. Some regression in the paralysis is frequent, but the hemiplegia, mental changes and epilepsy are incurable. Marked improvement can be expected in the syphilitic cases alone. Orthopedic devices are valuable. The resulting epilepsy is usually intractable and bromides are of little avail. Flechsig advises their use in combination with opium. Operative interference has been suggested. Twenty-five per cent. die from the operation, but the surviving cases seem to improve, especially in regard to the convulsions.

BRAIN TUMORS.

Etiology and Pathology.—Eichhorst found brain tumors in ⅙ of 1 per cent. of his cases. Sixty-six per cent. of tumors occur in the *male sex,* especially glioma and tubercle, while sarcoma occurs equally in the two sexes. *Age.* In the first decade, 18.5 per cent. of cases are observed; in the second, 14 per cent.; third, 20 per cent.; fourth, 18.5 per cent., and in the fifth, 14 per cent. (Gowers). The causation is that of neoplasms in general, as developmental errors, granulomata, metastatic growths and parasites. Trauma is a certain but infrequent cause; it may clearly cause growths, especially glioma, acting as the *"agent provocateur"* in gumma, sarcoma, osteoma, tubercle or aneurysm. The author has seen endothelioma of the dura develop years after and directly at the seat of an unhealed skull fracture. Syphilis may induce sarcoma.

Classification.—1. The Infectious Granulomata.—(*a*) *Tubercle* constitutes 50 per cent. of cerebral tumors, 50 per cent. being found in the first and 75 per cent. in the first and second decades (Gowers). The subject is usually poorly nourished and often exhibits lung changes; 33 per cent. occur in the cerebellum, 33 per cent. in the cerebrum and the remainder in the pons, central ganglia, crus, medulla and corpora quadrigemina. With equal frequency they are single or multiple and may number even 20 or 80. They originate from the meninges or vessels of the brain substance and are firm, rather rounded tumors, with grayish-red periphery and yellow caseated centre. Tubercles range from the size of a pea or walnut to that of an egg or of the fist, from fusion of many small growths. They grow along the lymph vessels, cause vascular thrombosis, present the degenerations of tubercle of other tissues and may be shelled out of the brain tissue, which is compressed and atrophied, but neither infiltrated nor softened. (*b*) *Gummata* (see Brain Syphilis) occur more frequently than statistics indicate, for they respond to treatment and often disappear spontaneously. In the adult they are the most common type of tumor. They are most frequent in the cerebral hemispheres and pons and are rare in the cerebellum and central ganglia; they are most often the size of a pea or hazel-nut, sometimes larger or miliary in size. Their centre is yellow-gray and their periphery is reddish-gray, gelatinous, rich in cells and ultimately cicatricial. Gummata are usually multiple. The adjacent brain tissue is not infiltrated, but commonly softened and compressed. Microscopically they closely resemble tubercle, but Virchow described them as more nodose, angular, irregular, more distinctly connected with the dura even when deeply located, more gelatinous and less caseated; both have been confused with actinomycosis.

2. Neuro-epithelial Growths.—(*a*) *Glioma* constitutes 25 per cent. of brain growths; 50 per cent. occur in the hemispheres, especially the cortex; 25 per cent. in the cerebellum and the balance in the central ganglia, pons, medulla, crus and corpora quadrigemina.

They occur more frequently in the brain than in the cord or retina, to which three structures they are peculiar. Gliomata are, in 90 per cent., single and are seen most frequently in adults (first to twentieth year, 20 per cent.; twentieth to fortieth year, 50 per cent.; fortieth to sixtieth year, 25 per cent., Gowers). Gliomata originate largely in the gray substance, which they so closely resemble in tint that detection is difficult at operation or indeed at autopsy, and secondary hemorrhage or softening in them may be confused with primary hemorrhage. Cases formerly called brain hypertrophy were probably gliomata. Their size varies from that of a pea almost to that of a hemisphere. Glioma infiltrates without sharp demarkation rather than compresses the brain tissue and does not form adhesions. The cells resemble the neurogliar structure and usually have round or oval nuclei; Klebs found ganglionic cells. Their processes are fine; numerous and branched delicate band-like lines result from fiber degeneration. (*b*) The rare *cerebroma* of Hayem was held by Virchow to be heterotopic gray matter in the white substance. (*c*) *Adenoma* of the pineal gland and hypophysis may be brought under this heading, as also may (*d*) growths from the plexuses and (*e*) neuromata.

3. Connective Tissue Tumors.—(*a*) *Sarcoma* is the most frequent, and constitutes 20 per cent. of brain tumors; it may develop in the meninges, periosteum or vessels. It is most common in adults. Though it infiltrates more than tubercles or gummata, it does so less than would be expected. Many are well demarked and cause more softening than infiltration. Its structure and degenerations are those of sarcoma elsewhere. Though it is most commonly single, diffuse or multiple sarcomatosis is sometimes encountered. Endothelioma (Ziegler), cylindroma (Billroth), angiosarcoma and melanosarcoma are but varieties. The sand tumors (psammomata) of the pineal gland, Pacchyonian bodies and choroid plexus are sometimes described as angiolithic sarcomata. The "pearly tumors" (cholesteatomata) are formed of flat endothelial cells. (*b*) *Benign connective tissue growths* (lipoma, fibroma and osteoma) are very infrequent.

4. Metastatic Tumors.—Cancer is not infrequent (7 per cent. of brain growths), especially from mammary or lung carcinoma, just as lung suppuration often secondarily involves the brain. It often invades the hemispheres and basal ganglia and may attain huge dimensions. Like sarcoma, it infiltrates. Cancer is very rarely primary.

5. Cystic Tumors.—Cysts may result from hemorrhage or softening, degeneration in neoplasms, infantile inflammations or defects (porencephalia), trauma or inflammation in the choroid plexus. *Dermoids* and teratomata are most rare. *Parasitic cysts* occur largely in persons between the tenth and thirtieth years; *echinococcus* cysts may be single or multiple; they occur chiefly on the surface of the brain or in the meninges. *Cysticercus* cysts develop mostly on the pia and arachnoid over the cortex along the sulci or in the ventricles. They occur in $\frac{4}{10}$ of 1 per cent. in certain autopsies, though Diamond

(1899) could collect but 8 cases from the American literature; they vary from the size of a pea to that of a hazelnut, are usually multiple (200 in Snell's case) and contain clear fluid with black dots, the heads of the parasites. Fibrous encapsulation and calcification may occur later. The cysticercus racemosus may reach a great size.

6. Aneurysms.—Aneurysms have been considered.

Localization.—The localization in Gower's figures was: cerebrum, 42 per cent.; cerebellum, 25 per cent.; base, 11 per cent.; pons, 8 per cent.; central ganglia, 7 per cent.; medulla, 4 per cent.; corp. quadrigemina, 2 per cent.; crus, 1 per cent.

Symptoms.—In rare instances, tumor is found at autopsy without clinical symptoms (*latency*). This is due to location of the tumor in a "silent" or tolerant brain area; it may occur in tuberculous meningitis, mental disease, aneurysm, cysticercus, psammoma, cholesteatoma or meningeal tumors, which slowly compress the brain. As a rule, symptoms result from the following *mechanisms:* (*a*) *Direct pressure* exerted on the brain, which may flatten the convolutions, obliterate the sulci, force open the skull fissures in children, absorb or perforate the cranium. Pressure is conspicuous in growths of the posterior fossa closed by the rigid tentorium. (*b*) *Internal hydrocephalus,* distention of the ventricles, may follow closure of the aqueduct of Sylvius or compression of the veins, particularly of the choroid plexus and the vena Galeni magna, and especially by tumors of the cerebellum and corp. quadrigemina. (*c*) The *brain* may be softened by pressure on its vessels (Bouveret), inflamed, or hemorrhage may develop (as in glioma). (*d*) Infiltration or destruction of the brain matter may result. (*e*) *Meningitis,* local as in gumma, or general as in tubercle, may develop. (*f*) *Auto-intoxication* from the growth is possible, as suggested by Klippel. (*g*) Distortion of the cerebral or spinal nerves (Dinkler, Mayer) may cause their degeneration, or changes in the posterior columns of the cord and its posterior roots.

The symptoms produced are *general* (diffuse) and *focal* (local).

1. General Symptoms.—The general symptoms usually come first in the evolution and include the following: (*a*) *Headache* is present in almost all cases, is usually constant, with accessions (especially in the morning), is general, rarely unilateral, is severe enough to keep the patient awake at night, is increased by alcohol, exertion and coughing, and rarely corresponds with the tumor's site except when the pain is persistently local and the tumor near the surface. Headache is usually referred to irritation of the dural branches of the fifth nerve, an explanation which is not always satisfactory. It may be pulsating in aneurysm. (*b*) *Choked disk* is the most important general symptom and occurs in 85 per cent. It consists of a grayish-red swelling of the disk to even two or three times its normal dimensions, which lessens its sharpness of outline and physiological cupping; the arteries become less lustrous and smaller, the veins turgid and tortuous, extravasations of blood and white patches of

fatty degeneration or exudation appear. (See Plate VII, Fig. 3.) It is due to increased intracranial tension, which causes mechanical hydrops of the optic sheath (Schmidt-Rimpler, Matz, Schwalbe) or to inflammation from choking of the veins by toxic products (Leber and Gowers). It is bilateral (90 per cent.), although more marked on the side of the lesion, is not an early symptom and depends neither on the size of the tumor nor on its location. It is almost always present in growths of the cerebellum and corp. quadrigemina, is less frequent in cortical and meningeal localizations and is often absent or appears later in those of the pons, medulla and corp. callosum. There is often, but not always, limitation of the field of vision (scotoma for color and lessened acuity of vision). In many cases it develops into atrophy, oftener still into stasis and blindness, while in some it may regress or very rarely recur. Transitory *amaurosis* results from compression of the optic tract or chiasm by the distended third ventricle. (*c*) *Convulsions* occur in 50 per cent., are called the "signal symptoms" by Seguin and may be general; general tonic precede clonic convulsions in no special order; the pupils react poorly, the skin reflexes are increased unless deep coma develops, the tendon reflexes are not suspended, respiration is labored and coma occurs. Convulsions are more frequently late than early. Slight seizures, like the *petit mal* of epilepsy, may occur. Jacksonian epilepsy belongs to the focal symptoms. (*d*) *Vomiting* is usually an early general symptom, indicating irritation of the vomiting centre (it is sometimes a focal symptom). It is of the cerebral type, occurring without effort, nausea or pain, often on an empty stomach, as in the morning, or at once after eating, regardless of the kind of food ingested; it is initiated by sitting up, turning of the head or excitement. Later the tongue is often coated. Vomiting is closely associated with headache and is due to intracranial pressure or meningeal irritation. It is rarely absent in growths of the cerebellum or medulla, is very frequent in disease of the corp. quadrigemina and occurs in 25 per cent. of tumors in the hemispheres. (*e*) The *sensorium* is often involved. The most common change is dulness, which often deepens into stupor and coma. It is not surprising that tumors affecting the association fibers and avoiding the motor centres may cause disturbance of intelligence, judgment or memory only; they are frequently discovered at autopsy in the frontal or temporo-sphenoidal lobes in subjects committed to asylums for psychoses or general paralysis. Hysteria may occur in subjects of organic brain disease. (*f*) *Vertigo* is often an early symptom; it may be mere "giddiness" or it may affect the labyrinth, pons or cerebellum and be a sense of actual rotation. It is present in 31 per cent. (Mills and Lloyd), occurs especially in the locations mentioned and as a focal symptom when the middle cerebellar peduncle is implicated. (*g*) *Slowed pulse* may result from direct, but more often from indirect, pressure on the vagus fibers behind and lateral to the hypoglossal nucleus. The vagus fibers become accustomed to pressure of long standing and the

heart rate increases. It is often associated with yawning, hiccough, Cheyne-Stokes' breathing, glycosuria or polyuria.

2. Focal Symptoms.—Focal symptoms may be absent when the disease is limited to "silent" or "tolerant" areas, when the tumor does not destroy nerve structures and when vicarious compensation by other parts is possible. Focal symptoms of irritation or paralysis may result directly from actual lesion of a centre or its fibers or indirectly from "distance pressure" upon them. They are, therefore, most significant when no particular increase of intracranial tension exists.

(*a*) *Frontal Lobes.*—The reported changes in mentality are much discussed; early observers, as Bernhardt, claimed that mental changes were no more frequent in frontal localization than in localization elsewhere. Many cases show the symptoms indicated above. Jastrowitz describes a condition designated as moria, a dementia with characteristic irritability and excitement. A tendency to make jokes (Witzelsucht) is described by Oppenheim. Psychical changes, especially those developing early, justify a "probability" diagnosis only. Allan Starr records the first diagnosis confirmed by operation. "The two prominent characteristics of the mental state are apathy and suffering, which may be associated or alternate" (Church). Early or late mental alteration sometimes follows other localizations. Sometimes tumors in the frontal lobes are thought to be in the motor areas from indirect pressure upon the latter. The head and eyes may deviate and the neck and back are sometimes rigid. Bruns has recorded frontal tumors with cerebellar ataxia, which is caused by unequal innervation with disturbance of the sensorium (Oppenheim) or involvement of the trunk centres (Strümpell). Some would associate with frontal lesions a lack of initiative. Bradyphasia (slow speech) is probably psychical and differs from the separation of syllables sometimes seen in tumor of the pons. In "laryngeal aphasia" the patient moves his lips and attempts to speak, but finds the voice lacking (Oppenheim).

(*b*) *Motor Cortex.*—(i) *Jacksonian convulsions* are often (50 per cent.) dissociated, and follow a given sequence (see Cortex Localization); consciousness is often preserved unless the other side of the body is involved. The convulsions may be few or run into the hundreds (17,000 in eleven months, as reported by Gowers). They may be preceded by the general convulsions described above. When the conducting fibers are destroyed they cannot occur, whence they may be observed in the part nearest the seat of greatest damage. As long as convulsions are present the motor centres cannot be wholly destroyed. Tapping the tendons or the skull may initiate spasms and convulsions can sometimes be aborted by electricity, by grasping the arm, etc. (ii) *Cortical paralysis* or paresis is usually monoplegic and is associated with cortical spasm; at first a weakness from exhaustion follows the spasms; later it becomes more marked. In some cases paresis (or total hemiplegia) may be the first sign of tumor and may

be narrowly localized, as in the thumb alone, the four fingers or great toe; paralysis is accompanied by increased reflexes, rigidity and rarely by muscular atrophy. Surgical extirpation has relieved both spasms and paralysis. In infrequent cases the paralysis may be bilateral, as when a cortical tumor near the median line presses on the opposite hemisphere or when there are bilateral symmetrical growths; these, however, are still rarer. (iii) Sensory changes are less frequent, because the sensory area is larger than the motor area and extends posterior to it and it is possible that sensory representation is bilateral. (The gyrus fornicatus is thought by some writers to be the seat of tactile and muscular sensibility.) Hypæsthesia, especially for tactile and muscular sensibility and paræsthesia, are not rare. Cortical pains are rare. Vasomotor symptoms, tachycardia and salivation are observed, but are not localizing.

(*c*) *Parietal Lobes.*—Some sensory disturbance is fairly common. Incoördinate movements, ataxia and athetosis have been observed. Wernicke observed a tumor in the lower parietal lobe with conjugate eye movements and Weil noted incoördination of the extremities on the opposite, and of the eyes on the same side of the body. Sensory aphasia, hemianopsia, deafness and word deafness are also observed.

(*d*) *Temporal Lobes.*—Symptoms may be confused with those of the contiguous occipital lobes; hemianopsia, disturbance of eye movements, chiefly toward the opposite side, ptosis and agraphia are recorded.

(*e*) *Occipital Lobes.*—Hemianopsia is the most constant finding; it results from any occipital lesion large enough to compress the optic radiation in the white substance (Henschen). Optical hallucinations indicate a cortical occipital localization rather than one in the optic radiation. Alexia, optic aphasia and psychical blindness are seen.

(*f*) *Pons.*—Focal may precede the general symptoms; they have been enumerated under cerebral localization. *Hemiplegia with alternating paralysis* of some of the first eight cranial nerves is the most common type (in 50 per cent.). Foci in the substance of the pons involving the sixth nerve usually also involve the third nerve of the opposite side; foci near the surface involving the sixth nerve do not involve the third nerve. Sometimes the nerves may be involved without hemiplegia (25 per cent.), but rarely without crossed motor irritation or hemianæsthesia; hemiplegia alone occurs in 25 per cent. Unilateral tumor of the pons may also compress the opposite motor tracts (paraplegia or diplegia) and cranial nerves. Motor irritation is not uncommon, as trismus, facial spasm and sometimes trigeminal neuralgia, convulsions in the contralateral limbs, etc. Tetanic rigidity is rare. *Sensory disturbance* is usual, as a result of involvement of the lemniscus and formatio reticularis and occurs as hemianæsthesia cruciata (in arm and leg of one side and in the face of the opposite side when the focus is low in the pons), hemianæsthesia of face, arm and leg of the same side (tumor in the upper pons) and anæsthesia of one or perhaps of both sides. Dysarthria, dysphagia,

difficult urination or defecation and disturbed respiration on change of posture have been observed.

(*g*) *Medulla.*—It is remarkable that not only cystic but also solid tumors may produce no symptoms or only such that localization in the pons or cord is first suggested. In a small number of cases only are the symptoms not ambiguous. Bilateral paralysis of the ninth to twelfth nerves with degeneration and atrophy of the lips, tongue, palate, pharynx or larynx is characteristic and presents a picture like progressive bulbar palsy. Alternating hemiplegia is rare. Sensory, motor, atactic and nerve involvements are usually bilateral. Growths *outside the medulla* may cause paralysis of the neck muscles. Slowed or rapid pulse, singultus, embarrassed breathing, glycosuria and polyuria are not uncommon. Degeneration in the posterior columns of the cord from pulling of the medulla on the posterior cervical roots or from toxæmia was found by Batten and Collier (1889).

(*h*) *Cerebellum.*—Cerebellar ataxia, referred by Nothnagel and Gowers to lesions of the vermis in 85 per cent. of the cases, occurs but half as frequently in disease of the cerebellar hemispheres. Compression of the pons, medulla, corpora quadrigemina and fifth to twelfth cranial nerves is somewhat characteristic. Sachs regards involvement of the sixth nerve as almost pathognomonic. Of the general symptoms, choked disk is severe and almost invariable; occipital headache and rigid neck are very frequent. Convulsions are common and opisthotonos with tetanic rigidity of the body and extremities is most suggestive.

(*i*) *Optic Thalamus.*—In most cases spastic hemiplegia results from invasion of the internal capsule; hemichorea, hemiathetosis, hemianæsthesia and Nothnagel's mimetic paralysis are not infrequent. Less common are unilateral convulsions, forced movements or postures, contralateral hemianopsia, paralysis of the eye muscles, widening of the opposite pupil and monoplegia.

(*j*) *Corpus Callosum.*—According to Bristowe, dementia (in 90 per cent.), absence of initiative in speech and action, paraplegia, dysarthria and few general symptoms, as choked disk, are characteristic. Oppenheim holds that there are no distinctive symptoms.

(*k*) *Corpora Quadrigemina.*—Paralyses of the third nerve are common, sometimes associated as double ptosis, and bilateral paralysis of the internal recti. The pupils are usually free. Oculomotor paralysis may exist with crossed hemiataxia from tegmental involvement or cerebellar pressure. Disturbed hearing from destruction of the upper layer of the tegmentum, amblyopia and intention tremor are also observed. No paralysis, except of the third nerve, is present unless the tumor extends beyond the corp. quadrig. (Oppenheim).

(*l*) *Pineal Gland.*—The symptoms are the same as when the corp. quadrigemina are affected, save that the fourth and sixth nerves are involved more than the third and compression of the pons or medulla is more common.

(*m*) *Tumors at the Base.*—These include bone tumors, aneurysms,

hypophysis growths and those from the nerves or meninges. (i) In the *anterior fossa,* hypophysis tumors (see ACROMEGALY) disturb vision and produce bitemporal hemianopsia with amblyopia or sometimes irregular or transverse hemianopsia. In some instances there is amblyopia of one eye and hemianopsia of the other. Usually there is no choked disk, but simple optic atrophy is present. The third nerve is paralyzed in most cases and sometimes other ocular trunks are affected. Exophthalmos is occasional and the growth may perforate externally into the eye, pharynx or nose. Loss of smell (anosmia) and mental symptoms are observed in tumors of the anterior fossa. (ii) When they occur in the *middle fossa* mental symptoms and convulsions are less common. The Gasserian ganglion may be compressed and result in neuroparalytic ophthalmia. Other cerebral nerves, as the third and sixth, sometimes suffer. It must be recalled that a tumor elsewhere may indirectly compress the basal structures. Syphilis and sarcomatosis may be diffusely basal. (iii) Lesions in the *posterior fossa* may simulate bulbar palsy. The lower eight cranial nerves usually suffer more readily and earlier than the pyramidal tracts; the grouping of the cranial nerve implication is different; thus paralysis of the sixth nerve is rarely associated with the conjugate fourth nerve, as occurs when the lesion is central. The seventh and eighth nerves are often conjointly paralyzed in disease of the posterior fossa and but rarely so in central tumors. The symptoms often resemble those of lesions of the pons, medulla or cerebellum.

Diagnosis.—Diagnosis depends upon the combination of focal and general symptoms. Four general groups of cases are encountered:—(1) Absolute clinical latency; (2) general and focal symptoms, the typical case; (3) general but not focal symptoms; (4) definite symptoms of some brain disease but of equivocal significance. According to Bruns, 80 per cent. of cases can be detected.

ANALYSIS OF THE GENERAL SYMPTOMS.—(*a*) *Headache* is suggestive when it is severe, keeps the patient awake and when the skull is tender to percussion or pressure (the parietal regions are normally somewhat sensitive). Its associates, as choked disk or vomiting, are equally important. Organic irritative brain disease, as abscess or inflammation, may cause headache, but mere increase of intracranial tension, as in hydrocephalus, need not do so. Functional nervous disease may cause headache, as migraine, neurasthenia, hypochondriasis, hysteria; it may result from eye disease, morbid conditions of the blood or intoxications (uræmia, gastro-intestinal disorder and poisoning by nicotine, opium, alcohol or lead), and from arteriosclerosis. (*b*) *Choked disk* in 80 or 90 per cent. of cases indicates brain tumor (Oppenheim) and if very marked it is seldom due to other causes. It is less frequent in abscess and meningitis, in which neuritis optica is more frequent. It is most rare in embolism, softening, hemorrhage, hæmatoma duræ matris, arteriosclerosis, poliencephalitis, encephalitis, the acute fevers, lead poisoning, neuritis multi-

plex or alcoholism (in which central scotoma is present). *Uræmia* must be excluded; although the two diseases have much in common, as headache, epileptiform or apoplectiform attacks, hemiplegia, aphasia and slow pulse, neuroretinitis is more common in nephritis. The choked disk of chlorosis is rare, but responds readily to rest and treatment by iron. Optic atrophy has the same significance as choked disk. (*c*) General epileptiform *convulsions* occur in meningitis, pachymeningitis, abscess, vascular disease, syphilis, alcoholism, plumbism, multiple sclerosis, paretic dementia, uræmia and gastro-intestinal disease in children.

Epileptic Convulsions.	Convulsions Resulting from Organic Brain Disease.
More coarse; more biting of tongue; more frequent cry; more sudden and complete loss of consciousness, frequently a visceral (epigastric) aura; usually longer interval between attacks.	Convulsions more orderly, slow, clonic, broken by tonic contractions; often with gradual local commencement, without loss of consciousness, and with paresis in the Jacksonian type.

(*d*) *Vomiting* is probably cerebral and organic and due to tumor, if hysteria, migraine, anæmia, toxæmia and gastric disease can be excluded. It occurs on getting up in the morning.

Combination of General Symptoms.—Headache and choked disk occur in chlorosis (*v. s.*), in plumbism (also with delirium, convulsions and coma, but distinguished by the lead line on the gums) or in uræmia in which special examination for albumin, casts and cardiovascular changes are definitive. The same combination may also prevail in hypermetropia in anæmic girls, in amenorrhœa (Broadbent) and in injuries of the head, but care usually distinguishes them from brain tumor, which is characterized by its slow onset and usual focal signs.

Focal Symptoms and Topical Diagnosis.—*Focal symptoms usually follow the general,* whereas, in apoplexy, for example, the focal symptoms, as hemiplegia, are early. In tumor with hemiplegia, contracture or spasticity accompanies the paralysis rather than follows it, as usually occurs in hemorrhage or embolism. Hemiplegia from a tumor is less pure, less circumscribed, less systematic (Fournier). A tumor at the base may cause hemiplegia from pressure on the middle cerebral artery with encephalomalacia. Jacksonian epilepsy with paresis is, in 50 per cent. of cases, due to cortical tumors. It is also produced by hemorrhage, softening and inflammation and is then earlier, is not confined to as small an area and is not as progressive as in neoplasm. The cortical paralysis and epilepsy observed in alcoholism, paretic dementia, plumbism, uræmia and rarely in tuberculosis, cancer and diabetes (Senator) are not dissociated and are but transitory.

Nature of the Growth.—(*a*) *Location. Cortical* growths are

probably gumma or tubercle (possibly glioma, cysts, cysticercus or sarcoma); *subcortical*, glioma or sarcoma; *pons*, tubercle, gumma, glioma; *cerebellum*, tubercle or glioma; *extracerebral*, sarcoma; *multiple occurrence*, tubercles or gummata. (*b*) *Course.* A rapidly growing tumor is probably glioma or sarcoma. An apoplectic attack indicates glioma or ruptured aneurysm. Growth with remissions is characteristic of tubercle or glioma. A very slow course suggests fibroma or osteoma. Coincident meningitis indicates tubercle or syphilis.

DIFFERENTIATION.—Tumor must be distinguished from *brain abscess;* abscess and tumor have much in common, as their general symptoms; both may result from trauma, but brain abscess is almost always secondary to trauma or to a pus focus (otitic or metastatic); unilateral optic neuritis is more common than choked disk, and abscess (*q. v.*) when once "manifest" is more apt to produce fever, chills, a rapid course and apoplectic seizures than is tumor, while involvement of the cranial nerves is less frequent. It must be borne in mind that tumors may cause fever and that in abscess, for a time at least, there is a normal or subnormal temperature. Differentiation from paretic dementia, pachymeningitis hemorrhagica interna, meningitis, syphilis and hydrocephalus will be considered under each disease.

Chronic syphilitic or tuberculous meningitis or meningo-encephalitis may be confused with diffuse tumors at the base.

Multiple sclerosis. In both affections intention tremor, disturbance of speech, mental change, vertigo, nystagmus, apoplectiform or epileptiform attacks and tonic spasms of the extremities may occur, but marked brain symptoms rather speak against multiple sclerosis, at least in its earlier stages. The pressure symptoms of tumor and increase of cerebrospinal fluid are absent and in the disk there is an incomplete optic atrophy. If undue stress be placed on the tremor it may be confused with incoördination tremor of tumor in the optic thalamus, pons or cerebellum.

Course and Prognosis.—The first symptoms are usually general, followed by the focal symptoms. The disease runs a fatal course in *one-half to two years,* in rare cases four to fourteen years. A long latent tumor may appear to begin acutely. *Exacerbations* result from aneurysmal rupture, hemorrhage into a glioma, cyst formation, ventricular effusions, meningitis or local neuritis. *Remissions* are not uncommon; healing of parasitic and simple cysts, aneurysms, tubercles (in the young) and gummata is possible. It was thought that aneurysms and gliomata may be cured with iodide treatment (Wernicke). *Unfavorable* symptoms are pronounced mental change, incoërcible vomiting, irregular rapid pulse, repeated fits, apoplectiform seizures or festinating neuritis optica. The *mechanism of death* is (*a*) exhaustion from vomiting, pain or cachexia; (*b*) inhalation pneumonia; (*c*) coma with disturbed respiration, circulation or other bulbar signs; (*d*) sudden death in tumors of the base (rarely of the cortex); (*e*) meningitis or arterial disease; (*f*) intercurrent infections.

Treatment.—Medical treatment does not produce encouraging re-

sults save in cases of syphilis, but every patient should be given a course of mercury and iodide for at least three months. Tubercle may sometimes be arrested by increasing the physiological resistance by forced feeding, fresh air and administration of cod-liver oil and arsenic. The headache is often relieved by iodide, in any kind of tumor, by the ice-bag, morphine, atropine, hyoscine, the coal tar products and by decompression operations. Convulsions should be treated by chloral. Brain congestion from alcoholism, excitement, exertion, overeating or obstipation should be avoided.

Surgical Interference.—Macewen, Horsley, Bennett, Ferrier and Godle are the pioneers in brain surgery. (*a*) In *radical operation* the outlook is not very encouraging. Allan Starr estimates that but 18 per cent. of tumors are accessible, and but 7 per cent. removable; Siedel finds only 3 per cent. are operable. Thirty-four per cent. are basal. At operation the tumor is found in 64 per cent. of cases. The best results are obtained in meningeal and the next best in cortical growths, especially of the motor and occipital zones. Duret (1903) analyzed 344 operations; 18 per cent. died, 64 per cent. improved and vision was completely restored in 60 and partially in 18 per cent. In cerebellar tumors sudden death is frequent, because of manipulation compression of the medulla. In 116 operations for cerebellar growth the mortality was 38 per cent. (Frazier, 1905).

All varieties, even the syphilitic scar or tubercle, may be removed, excepting metastatic growths. Recovery from the operation is frequent, although the mortality ranges from 20 to 50 per cent. But few ultimately recover. Recurrence is common. Death occurs from hemorrhage, sepsis, dislocation of the brain, basal compression, brain œdema and shock. Horsley and Macewen have suggested that the operation be performed in two steps; first exposing the tumor and later removing it after the brain has accommodated itself to the change of relations. (*b*) *Palliative decompression* may be beneficial. Removal of a button of bone with incision of the dura often greatly relieves the headache and growing blindness, allows fluid to escape and permits the tumor to grow outward when it cannot be removed. Henschen claims that the palliative procedure hastens death twice as frequently as it relieves the symptoms. (*c*) *Ventricular drainage* has not proven helpful; from lumbar puncture 17 cases of sudden death are reported.

INFLAMMATION OF THE BRAIN.

I. Encephalitis.—"Inflammation of the brain" as understood by the laity, is meningitis. Encephalitis does not include the inflammation about tumors, hemorrhage, softening, syphilis and meningitis. It was once held by Durand-Fardel that all softening was encephalitis (cerebritis). Gluge (1837) suggested that some cases were encephalomalacia; Coswell and Hasse (1850) definitely distinguished between them; and Virchow anatomically and Hayem clinically described clear cases.

Etiology.—There are three main etiological factors: (*a*) *Intoxications,* chiefly from alcohol, and far less often poisoning by sulphuric acid, ptomaines in decayed meat and fish, nicotine and sunstroke. (*b*) *Infections,* especially influenza, then scarlatina, measles, hydrophobia and numerous others. In such cases microbes may invade the brain alone. Some relation to chlorosis, sinus thrombosis, chorea insaniens or maniacal forms of exophthalmic goitre is possible. (*c*) *Trauma,* by laceration or concussion, may produce a *locus resistentiæ minoris* for secondary microbic invasion.

Pathology.—The process begins as multiple disseminated and often symmetrical foci, principally in the gray matter; these grow and diffuse, sometimes most widely. They vary in size from microscopic dots to a diameter of several inches, but usually are the small "flea bite" areas of capillary hemorrhage, which glisten and stain the adjacent tissues, show above the cut section and later become foci of softening. Microscopically, hyperæmia, perivascular exudation of red and white cells, serous effusion and changes in the nervous elements are observed. Aggregations of mononuclear cells are seen, somewhat resembling a tubercle, and degenerated, fatty and swollen ganglion cells; the medullary sheaths and later the axis cylinders degenerate. Its issues are:—(*a*) regression (recovery); (*b*) necrobiosis with softening; (*c*) cyst formation; (*d*) cicatrix formation or (*e*) possibly diffuse brain sclerosis or multiple sclerosis. *Associated nervous changes* are optic neuritis, multiple neuritis and poliomyelitis, and *general changes* are enlarged spleen, retinal or general hemorrhages and parenchymatous degeneration in the kidney, etc.

Special Pathology and Symptomatology.—1. Poliencephalitis Acuta Superior Hemorrhagica or Ophthalmoplegica.—This was said by Wernicke (1881) to occur in *alcoholic* subjects and therefore in adults; it attacks the gray matter about the third and fourth ventricles and aqueduct of Sylvius, whence it may spread upward or downward; it results in eye paralysis and is analogous to acute poliomyelitis. The *general symptoms* are delirium tremens, or somnolence in non-alcoholics, headache, vomiting and rigid neck. The *local symptoms* consist of an early extensive ophthalmoplegia, which involves associated eye muscles, as the two internal recti, or the internal rectus of one eye and the external of the other and usually leaves the pupil and the levator palpebræ superior free. The gait is atactic from involvement of the corpora quadrigemina. Speech is frequently difficult. Neuritis is observed in the optic disk and sometimes in the peripheral nerves, as evidenced by the absent patellars, ataxia, tender muscles, etc. Sometimes there is hemiparesis and facial paresis. The temperature is usually normal or subnormal, though respiration and pulse are increased. Death occurs within two weeks after these violent symptoms. This type is most easily recognized, although syphilis, tumors or encephalomalacia may affect the same nuclei. Some cases thought to be nuclear have been shown by Bädecker, Thompson and Jacobaeus to be polyneuritic, and others have shown no pathological alteration at autopsy.

2. Encephalitis or Poliencephalitis Hemorrhagica.—Strümpell (1884) described primary cases of infantile paralysis (*q. v.*) in which he referred the lesion to inflammation of the motor cortex; though his theory is too general, it has some anatomical confirmation. In 1885–86 Leichtenstern saw cases which resulted from the infections named under etiology. Foci are found in the cortex, subcortex or central ganglia, containing pus cocci and influenza bacilli. In some cases the autopsy showed nothing and intoxication was then assumed. The disease occurs mostly between the fifteenth and thirtieth years. After an influenza there may be (*a*) *general symptoms,* as headache, vomiting, delirium, mental obscuration, convulsions in the young, high fever, slowed pulse becoming rapid, irregular breathing or sometimes stiff neck or optic neuritis; (*b*) *focal symptoms* appear after a few days, as hemi- or monoplegia, aphasia, conjugate deviation, focal convulsions or hemichorea. The reflexes are affected as in apoplexy. The course lasts one-half to three weeks and is often fatal. This form may resemble hemorrhage, syphilis or softening, but occurs in the young with normal arteries, and the paralysis is preceded by signs of infection, fever, enlarged spleen, somnolence or convulsions. Meningitis is usually differentiated with ease.

3. Subtypes.—These consist of the following: (*a*) *poliencephalitis inferior acuta,* involving the pons or medulla and giving acute bulbar symptoms; there is paralysis of the sixth, seventh to twelfth nerves when it occurs in various combinations or with types (1) and (2). Some of the cases (chorea insaniens, Basedow's disease with mania) may show nothing at autopsy, whence the diagnosis is very uncertain. Confusion with softening, hemorrhage, the late hemorrhage of trauma or tumors is possible. (*b*) Another form is *poliencephalomyelitis,* acute, subacute or chronic, in which the type of Wernicke or of Strümpell-Leichtenstern may co-exist with inflammation and paralysis, frequently symmetrical, of the anterior spinal roots. Hutchinson's facies is observed, *viz.,* fixed eyes, fallen lids, prominent eyebrows, obliterated nasolabial folds and atrophic paralysis of the arms. Differentiation from multiple neuritis is usually possible, and Oppenheim's criteria are well established in its separation from myasthenia gravis, in which there are (i) no atrophy, but (ii) remissions in the paralysis, (iii) great muscular adynamia, (iv) absence of reaction of degeneration and (v) negative anatomical findings.

Prognosis.—The prognosis is unfavorable, as a rule. The peracute cases may die in two days; acute cases last one-half to three weeks and death is most frequent; Oppenheim has shown that they may recover. Much involvement of the mind, decubitus, alcoholic delirium, chlorosis, sinus thrombosis and weak pulse are unfavorable elements. In children recovery is common, but epilepsy, paralysis, mental alteration and possibly multiple sclerosis are sequences.

Treatment.—This is wholly symptomatic. (*a*) The causal factor must be eliminated, (*b*) cerebral congestion avoided and (*c*) the skin,

bowels and bladder, delirium and convulsions, diet and patient's rest must be cared for as in brain hemorrhage, softening or tumor. (*d*) Mercurial inunctions, iodoform inunctions and potassium iodide give the best results. (*e*) Electricity is indicated in the chronic stage.

II. Abscess of the Brain.—Encephalitis suppurativa or apostematosa is not a common disease. Eichhorst found but three among 13,563 medical cases.

General Etiology.—All cases are caused by microörganisms, of which the streptococcus is the most frequent, the staphylococcus coming next and then the pneumococcus and other bacteria. Mixed infections are common; old abscesses are often sterile. Sixty-six to 75 per cent. occur in men. It is most frequent between the twelfth and thirtieth years. Twenty per cent. occur in the second and 33 per cent. in the third decade (Gowers). The cerebrum is involved in about 75 per cent. and the cerebellum in 25 per cent.; the pons and medulla are rarely involved.

General Pathology.—The focus is single in 80 per cent. of cases. The average size is from one to two inches, but may be almost microscopic or occupy nearly the entire hemisphere. The surrounding brain may be inflamed. A zone of œdema is common. The right side of the brain is more often affected than the left. Pressure is less common and less intense than in tumor; very large abscesses flatten the brain and may produce internal hydrocephalus. The abscess consists of pus of variable consistency, yellow or green, acid, sometimes rancid, fœtid or putrid; flocculi of disorganized brain tissue are common. The pus cells are often disintegrated and fatty needles, cholesterin crystals and sometimes corpora amylacea or foreign bodies are found. Putridity is due to communication with the nose or ear or embolism from putrid lung foci. Encapsulation occurs in 50 per cent., chiefly in old, slow cases, and requires from two weeks to two months. The capsule rarely completely or permanently limits the abscess, which usually grows and often ruptures; ultimate quiescence from inspissation and successful demarkation is barely possible.

Special Etiology and Pathology.—1. Infective Foci near the Brain.—Infective foci near the brain are its usual causes, and for this reason brain abscess is seen by internalists, neurologists, surgeons and otologists. (*a*) *Ear disease* explains at least 25 per cent. of brain abscess. Otitis media, the most frequent cause, is chronic in 91 per cent. (Grünert). Insurance companies recognize the risk in these cases. Jansen found one abscess to 500 cases of chronic and one to 2,650 of acute otitis. The ear disease may date back thirty or forty-five years and is especially dangerous when it recurs and when granulations and polypi exist. The acute infections are thus indirectly causative of brain abscess. Infection traverses the bone (92 per cent., Körner) to the dura by way of the thin tegmentum tympani, by the roof of the antrum, sometimes through the petrosquamous suture (in children), by the mastoid cells, lateral sinus,

labyrinth or aqueduct along the sheaths of the seventh and eighth nerves. Körner's figures show that in 42 per cent. there is a fistule between the abscess and the causal focus; in 15 per cent. the dura interposes between the two foci; in 15 per cent. there are dural adhesions and in 17 per cent. the intervening substance is softened brain tissue. Toynbee (1885) remarked that (i) disease of the roof caused temporosphenoidal abscess, but (ii) disease of the mastoid cells chiefly produced cerebellar abscess. Of otitic abscess, temporosphenoidal constitutes 67 per cent. (75 per cent. in children under ten years) and cerebellar abscess 33 per cent. (Körner); the latter is rarely due to any cause other than otitis and almost invariably occurs in the anterior part of its hemispheres. Otitic abscess infrequently involves the occipital, frontal (two personal observations) or parietal lobes. The abscess is single (87 per cent.) and lies in the white substance of the brain just beneath the cortex. The smaller the abscess the nearer it lies to the cortex. Cholesteatomata may cause some cases. (*b*) *Traumatism* produces 25 per cent. of the cases. Punctured wounds are more dangerous than open compound fractures. Fractures of the base may result in abscess by infecting the middle ear. Splintering of the inner table and operations on the brain are possible causes; abscess may occur without skull fracture, contused areas becoming infected through the blood. The injury may be direct or in rare cases on the opposite side by *contre coup*. Gowers states that but 20 per cent. occur under ten years of age. Ninety-three per cent. are solitary abscesses (Oppenheim). Infection travels along the perivascular and perineural lymph sheaths (Biswanger), but may pass by other routes (arterial thrombosis, or by way of the diploë veins, with or without thrombosis, for "retrograde infection" is possible by the valveless veins). The early traumatic abscess is usually cortical; the late variety is usually subcortical. (*c*) *Other contiguous disease* is much rarer, causing but 4 per cent. of brain abscess. Disease of the nose and communicating sinuses (frontal, antral, sphenoidal, ethmoidal) may cause abscess of the frontal lobes and in antrum disease sometimes of the parietal lobes. Disease of the orbit, as phlegmon or penetrating wounds, and still more rarely cervical cellulitis, cranial osteomyelitis, ulcerative tuberculosis, syphilis or tumor of the cranial bones may cause abscess by contiguity.

2. Metastatic Abscesses.—Metastatic abscesses cause 25 per cent. of brain abscess. The abscesses may number from 2 to over 100; they are multiple in 66 per cent. In location they are cortical or subcortical, less frequently in the central ganglia and almost never in the cerebellum alone. The embolus is almost never found. (*a*) *Lung lesions:* "pulmonal cerebral abscesses" are due to empyema, putrid bronchitis and bronchiectasis (45 per cent.), gangrene, abscess and sometimes tuberculous cavities. This form rarely encapsulates. (*b*) *Ulcerative endocarditis,* pyæmia, osteomyelitis, etc., are less common causes; paradoxical embolism may occur through an open foramen ovale, whereby the clot escapes the lungs. Actinomycosis,

suppurating tubercle and oïdium albicans have been described; in thrush Wagner directly traced the growth from the throat to the brain.

3. Idiopathic Brain Abscess.—Gowers found it in 16 per cent.; it may result from a forgotten trauma or ear abscess. Martius and Strümpell believe the meningococcus causes some of these cases.

Symptoms.—Symptoms are absent in 10 per cent. of cases. Symptoms of the *causal affection* are described under diagnosis.

1. Symptoms of Sepsis.—(*a*) *Fever* is frequent. Oppenheim considers it the most important general symptom; Eichhorst describes cases closely resembling typhoid; it is present in acute cases and during the last stages when the abscess spreads or meningitis or sinus thrombosis develops; the original disease may cause it. Macewen's cases showed normal or subnormal temperature. (*b*) Chills are not frequent. (*c*) The pulse is often accelerated, but may later become slowed to 30 or 40, the compression pulse. (*d*) Other septic signs (see Septic Infections).

2. Diffuse Brain Symptoms.—These are due to increased intracranial tension, œdema, inflammation, meningitis or internal hydrocephalus and include (*a*) *headache,* which is most common and usually severe, though less so than in brain tumor; it is usually localized, but even then the stiff, painful neck and headache are general rather than focal symptoms. It is frequently associated with (*b*) *vomiting* and (*c*) *vertigo,* which also occur in simple ear disease. (*d*) *Mental symptoms* are like those of tumor, "slow cerebration, heavy comprehension and a want of sustained attention" (Macewen). (*e*) *General convulsions* are frequent only in the last stages, in cases of extensive abscess or rupture into the ventricle. (*f*) The *pulse* is often slowed, even with co-existent fever, to 40 (even 10) and sometimes with slow or irregular respiration. (*g*) *Optic neuritis* is more common than choked disk; it occurs in 33 to 40 per cent., develops rapidly and may produce amaurosis and atrophy.

3. Focalizing Symptoms.—Focalizing symptoms are often absent and when present are less conspicuous than in tumor because they are obscured by the primary affection or the general symptoms and occur in areas of less physiological dignity. When present they usually signalize advance of the abscess, increase of œdema, softening or increased brain tension.

Frontal or mental symptoms may occur (see Tumor), but foci as large as the fist may remain latent. They may impinge on the *motor cortex.* The symptoms are those of a cortical lesion (dissociated paralysis), which extends more rapidly than tumor and is attended by Jacksonian fits, rigidity and increased tendon and decreased skin reflexes on the contralateral side; in subcortical lesions from traumatic and metastatic (pulmonal) abscess, hemiparesis occurs in 50 per cent., is sometimes progressive, is usually incomplete and at times is apoplectiform in onset (three personal observations), even when not embolic. If hemiparesis is early it rather indicates involvement of the internal capsule, which also causes hemihypæsthesia.

Conjugate deviation is not common. *Temporosphenoidal* lesions are largely otitic, are frequently latent and are less frequently cortical than subcortical, whence the centres are less involved than the deeper association fibers. The left side is more important; lesions here lead (in 42 per cent.) to *sensory aphasia,* partial word deafness, alexia, agraphia, amnestic and optic aphasia. The right side concerns the memory for letters and figures (Oppenheim). *Occipital* lesions or those deeper in Gratiolet's optic radiation, produce hemianopsia (more in traumatic or metastatic than in otitic abscess). *Cerebellar abscess* results almost exclusively from ear disease and is often latent. Ataxia may also be caused by the ear affection or abscess elsewhere, even in the frontal lobes. Occipital headache and stiff neck occur; there may be a tendency to fall forward or backward and hemiparesis or yawning may result from the compression; cranial nerve paralyses are not frequent and result from coincident meningitis. Paralyses of the seventh and eighth nerves are often due to bone disease. Abscess of the pons, medulla (of which Cassier, in 1903, found only 16 cases) and crus is exceptional. (See CEREBRAL LOCALIZATION.)

SYMPTOMS BY STAGES.—(*a*) The *initial stage* is observed mostly in acute traumatic or metastatic abscess. Pus formation usually requires ten to thirty days. The causal symptoms, those of sepsis, and general brain symptoms, appear rapidly but focal symptoms usually appear only after one-half to three months; death may occur from coma, fever and meningitis or the next stage may appear. (*b*) The *latent stage* has no fever, is seen in chronic abscess, probably corresponds with demarkation and encapsulation, lasts for months, years or even decades and is either complete or interrupted by symptoms of the third stage. (*c*) In the *"manifest" stage* symptoms of sepsis and general or focal brain symptoms appear continuously or remittently, with evidence of extension. Most cases are seen in this stage. Gowers estimates that after nervous symptoms once develop, (*d*) *the next, the fatal or terminal stage,* appears in five days (20 per cent.), in ten days (33 per cent.), fourteen days (50 per cent.) and in thirty days (75 per cent.). This stage may immediately follow the latent stage when rupture into the lateral ventricle occurs.

Diagnosis.—Recognition is impossible in latent cases affecting "silent" areas, whence "no brain disease of equal severity so often escapes recognition." When focal symptoms are absent, various psychoses may be thought of. The diagnosis is often made only in the last two stages. Few cases can be detected without consideration of the etiology, which is definitive in 75 per cent., the sepsis, general and focal brain symptoms.

Otitic abscess may be confused with the otitis itself, especially in children; otitis may produce general symptoms, as headache, vomiting, convulsions and even optic neuritis (probably due to serous meningitis), cranial nerve symptoms (nystagmus, diplopia), but all symptoms disappear when the middle ear, attic, mastoid or subperi-

	Brain Abscess (Otitic).	Diffuse Suppurative Meningitis.
Etiology:	Ear disease.	Same; favored by imperforate tympanum and childhood.
Onset:	Acute or subacute, but often some latency.	Acute, stormy, even apoplectic.
Course:	Acute, subacute, chronic (latency stage), from weeks to seldom over one year.	Acute, stormy, more rapid than abscess (66 per cent. last under one week).
Fever:	Normal, subnormal, moderate.	High and continuous.
Pulse:	Slow.	Usually fast and irregular.
Sensorium:	Simple dulness, merging into coma.	Unrest, delirium; dulness later.
Headache, vomiting, vertigo:	Headache almost constant; others less constant.	Headache and vomiting almost invariable.
General convulsions:	Not frequent.	Usual, clonic and tonic.
Retina:	Optic neuritis common. Choked disk not common.	Usually negative because of rapid course.
Meningitic evidences:	Less by far. Stiff neck rare save in cerebellar abscess.	More common psychical and motor unrest, jactitation, hyperæsthesia of special and general sensation, trismus; retraction of abdomen; general rigidity.
Focal symptoms:	Frequent, especially temporosphenoidal, internal capsule and sometimes cerebellar; paralysis of third nerve.	Diplopia, strabismus, unequal pupils, conjugate deviation, paralysis seventh and eighth nerves; these are diffuse, transitory and variable; rarely hemi- or monoplegia; very rarely aphasia, hemianopsia (more frequent in meningitis tuberculosa).
Spinal symptoms:	Seldom. Few cases of absent patellars.	Almost always stiffness and tenderness of neck, back and general muscles, Kernig's sign; paræsthesia and hyperæsthesia, dysuria, involuntary evacuations, absent patellars.
Lumbar puncture:	Rather dangerous.	Positive findings (see pages 170 and 171).

osteal foci are cared for; otitic brain abscess runs an acute rather than a chronic course; the period of latency rarely exceeds one to one and a quarter years and usually averages two months or less. Temperature is often lower than in other forms, the headache may be local and œdema may be seen over the temporal region, with tenderness on percussion and a higher percussion note (Macewen).

Otitis may also cause *suppurative meningitis,* extradural abscess or sinus thrombosis, either with or without brain abscess, from which they must be differentiated. The table on page 971 is borrowed from Oppenheim's classical description.

Serous meningitis (Billroth and Quincke) is often mistaken for the suppurative form and is very important in ear disease. It is attended by slight temperature, internal hydrocephalus, paralysis of the basal nerves, often gives a serous fluid on lumbar puncture and is often cured. *Extradural abscess* was found in 78 per cent. of cases of brain suppuration by Jansen, sinus thrombosis in 18 per cent. and brain abscess in barely 3 per cent. It is more common in acute otitis (43 per cent.) than is abscess (9 per cent.). Local symptoms, as œdema and tenderness, are commoner than diffuse and focal symptoms. *Sinus thrombosis* (*q. v.*) is more often attended by pyæmic symptoms, high fever, chills, sweats, icterus, foci in the lungs, rapid pulse, choked disk, general convulsions and external symptoms of altered brain circulation.

Traumatic abscess develops *early,* is associated with hæmatoma, concussion, contusion or diffuse meningitis, from which differentiation is often impossible or difficult, except that the abscess is slow in evolution. Its presence is often suspected because of delayed healing of the wound. It is usually cortical (whence its focal symptoms) and often provokes fever and other general symptoms. It must also be differentiated from traumatic hemorrhage, which occurs days or weeks after the injury and often in the corp. quadrigemina, pons or medulla; from acute encephalitis, whose course is more rapid; from concussion, in which the symptoms are more diffuse and may involve the brain nerves or arouse fever, though cortical symptoms are absent; from traumatic neuroses, which lack focal and septic symptoms. In *late traumatic abscess* latency may last even thirty years. The abscess is not cortical (affecting the centres), but subcortical (affecting the conduction or association fibers). *Cerebral tumor* (*q. v.*).

Abscess by metastasis. It is well known that lung suppuration affects the brain with especial frequency, whence the chest must be examined; localization is largely in the hemispheres, thus obeying the general topography of simple embolism. The Sylvian district and occipital lobes are most often affected and the multiplicity of localization should be remembered. The course is usually precipitate.

A diagnosis of *idiopathic abscess* without proper etiology is always unsafe and usually impossible.

Prognosis.—*Spontaneous recovery* by demarkation, inspissation, calcification or external rupture is very unusual. Without operation,

death is practically inevitable, (*a*) from rupture into the lateral ventricles (16 per cent., chiefly in metastatic and otitic abscess) and into the membranes (28 per cent., in cerebellar abscess), (*b*) from hydrocephalus internus, (*c*) from sinus thrombosis, (*d*) from pressure on the medulla, or (*e*) from brain œdema. Sudden death may occur, as in tumor, without adequate postmortem explanation.

Treatment.—(*a*) *Prophylactic* therapy concerns the etiological factor. Adequate drainage is always necessary, although granulations in the middle ear are protective and should not be curetted in any routine manner (precisely as uterine curettage in puerperal sepsis is now discountenanced). (*b*) *Palliative* treatment is that of brain tumor and meningitis. Special care regarding vomiting is indicated, lest it rupture the abscess. (*c*) *Surgical* intervention is indicated. Successful cases were those of Morand 1868, Roux 1848, and Schede 1866. Macewen, Barker, Greenfield, Ferrier, Horsley and Schwartze are the pioneers in its practical treatment. Von Bergmann's rule was that operation should never be performed unless the diagnosis was certain, but adherence to this rule would have cost many lives. The contra-indications are (*a*) metastatic abscess, (*b*) a fatal etiological disease, (*c*) inability to stand the anæsthesia or shock, (*d*) ventricular rupture and (*e*) diffuse meningitis, although in the last two cases isolated recoveries are recorded. Sinus thrombosis, circumscribed meningitis and incipient pyæmia do not contra-indicate operation. In Körner's cases (otitic abscess), 51.6 per cent. recovered, Oppenheim reported recovery in 45 per cent. (traumatic cases), Wheeler's series gave 87.5 per cent. and Macewen's series showed 96 per cent.

III. Dementia Paralytica.—It is also known as paralytic or paretic dementia, general or progressive paralysis of the insane and among the laity as softening of the brain. Bayle, 1822, and Calmeil, 1826, first accurately described the affection. It is often classed among the mental rather than the brain diseases, but it is so important in everyday practice and constitutes such a large proportion (10 to 25 per cent.) of all insanities that its description is in place.

Definition.—Dementia paralytica is a chronic inflammation or degeneration of the brain, characterized by progressive vasomotor, motor and psychical disturbances and ending in death after two or three years.

Etiology.—(*a*) Syphilis is the cause in at least 75 per cent. of cases (Mendel and Strümpell). That it is not the sole cause is shown by its rarity in Egypt and Japan where syphilis is endemic. (*b*) Other causes, as *modern life,* with its strenuousness, competition, hurry and worry aid the syphilitic toxins which cause degeneration of the brain after the syphilis is apparently spent (a parasyphilitic affection, Fournier). Short sleep, dissipation, overwork and city life account for its increasing frequency. Krafft-Ebbing summarized the etiology as "syphilization and civilization." These causes may co-

operate with (*c*) *hereditary nervous taint* (20 per cent.) and (*d*) *alcoholism* (20 per cent., though this is denied by many). (*e*) *Certain occupations,* as those of officers, artists and actors, are predisposing factors and, generally speaking, the higher classes are more subject to it. Krafft-Ebbing found no clergymen among his 2,000 patients. (*f*) It occurs between the *thirtieth and fiftieth years,* and (*g*) is 7 to 10 times as frequent *in men* as in women. Other factors are less prominent, as exposure to heat, trauma of the head, plumbism or chronic toxæmia.

Pathogenesis.—The causes enumerated probably produce (*a*) vasomotor paresis in the forebrain, followed by (*b*) lymph stasis in the cortex and pia mater, (*c*) degeneration and atrophy of the brain and cord, and (*d*) interstitial inflammation. Though all cases end in brain atrophy, opinions still differ regarding the role of the inflammation; some regard encephalitis interstitialis (chronic meningoencephalitis) as primary (Mendel) and others hold that it is secondary to encephalitis parenchymatosa (Tüczek and Wernicke), just as in liver cirrhosis it is now thought that the connective tissue formation is secondary in time and importance to cellular alteration.

Pathology.—(*a*) The calvarium is thickened in 33 per cent. The Pacchyonian granulations are often enlarged. (*b*) The dura in 50 per cent. is thick, opaque, vascular and adherent to the skull (*pachymeningitis,* which is often hemorrhagic). In attempting to strip off the pia it is found granular and adherent to the brain cortex (in 80 per cent.), especially over the frontal and motor areas, supplied by the internal carotid artery. Later the adhesions may disappear. (*c*) *Gross brain changes* are constant. There is less opportunity to inspect the brain in the early stages, when it is swollen and hyperæmic than in the later stages when it is *wasted;* it may weigh but a third or quarter of the normal (40 ounces or 1,260 gm.); atrophy affects largely, if not wholly, the areas supplied by the internal carotid artery, especially the frontomotor cortex and the island of Reil. The gray matter is yellow-gray, soft and wasted, but the atrophied white substance is firm. The *convolutions* are greatly *narrowed* and the sulci are widened, the degree of atrophy corresponding to the 6 or 8 ounces of serum which fills the deficit (hydrops ex vacuo). In 80 per cent. granulations are seen on the ependyma of the lateral ventricles, resembling the back of a "cat's tongue," and the ventricles contain excess of fluid (hydrops ex vacuo). Areas of hemorrhage, softening and cyst formation are due to complicating vascular disease. (*d*) The *minute alterations* in the brain are as follows: the ganglion cells are early swollen and cloudy; the later findings are vacuolated, degenerated and atrophied ganglion cells; *wasting of the medullary sheaths* and of the axis cylinders, especially in the association or tangential nerve fibers running parallel to the cortex (Tüczek), which are said to waste before any inflammation can be seen; corpora amylacea represent what is left of them; the same changes occur in the cranial nerves; the bloodvessels show mural proliferation, *exuda-*

tion, colloid or hyaline metamorphosis, thickening and later atrophy or obliteration; the lymph vessels show proliferation, transudation and compression of the cortex, where lymph cysts, from snaring off of lymph vessels, sometimes cause cystic degeneration of the cortex; the neuroglia increases even sevenfold (sclerosis). (*e*) Alteration of the *spinal cord,* described first by Westphal, is present in 90 per cent.; the lateral columns often show descending *degeneration,* but most commonly changes *like those of tabes* are found in the posterior columns and in the posterior roots. Pachy- and leptomeningitis are seen in 40 per cent. As in tabes, the cerebrospinal fluid contains chiefly mononuclear cells. (*f*) Changes in the cranial nerves, especially the first to the seventh, may result from syphilis or alcoholism.

General Symptoms.—As a rule, the symptoms are first vasomotor, second psychical, and third motor, in the order of their usually slow development. It is possible for the psychical symptoms to appear first, with the motor, or more rarely to follow them. The leading symptoms are referable to alteration in the frontomotor cortex. *Three stages* are usually distinguished: 1. In the *prodromal stage,* recognition is often difficult, because the first symptoms are equivocal or point to (*a*) *neurasthenia* because of the "irritable weakness," ready fatigue in mental processes, headache or sense of cephalic pressure, introspection and rheumatic pains. Again, the initial symptoms are (*b*) *congestive,* as headache, migraine or attacks similar to *petit mal.* (*c*) Most important are mental changes. The patient is described as "not himself" but "he does not remark his own alteration" (Schule). Krafft-Ebbing gives a lucid picture of these alterations. The intellect is cloudy; the patient is confused as to time, and comes too early to work or remains after time; he confuses places and may smoke in church; his observation is faulty as well as his judgment; he confuses dreams with actualities and his memory is confused, for he forgets entire epochs, especially recent impressions. Æsthetic and social lapses are common; the subject becomes vulgar, careless, neglects duties, disregards amenities and from lack of will power may steal or dissipate most openly. Emotional outbreaks occur, as violent or brutal conduct or maudlin sentiment over soon forgotten incidents. The patient is often perniciously active. (*d*) Focal symptoms, especially amnestic aphasia, may appear. (*e*) General findings are noted after a time, as small and unequal pupils or other tabes-like findings, double vision, unequal innervation of the face, fibrillary contractions of the tongue, tremor, pains, headache, vertigo, congestive attacks, slow pulse, rough, unmodulated voice, skipping of words in reading (paralexia) and irregular, incorrect writing (paragraphia). This stage covers months or years. In rare instances the symptoms may follow tabes; this is "the ascending form of general paralysis." 2. At the *height* of the disease, (*a*) the psychical symptoms take various types, as maniacal exaltation, melancholia, hypochondriasis or simple progressive dementia. Psychical blindness or deafness occurs. (*b*) Congestive, apoplectiform attacks

are frequent and the general findings become more distinct, as immobile and unequal pupils, hesitant speech with elision of syllables, altered expression, trembling or paresis of the face, incoördination of the hands and gait, disturbances in writing and reading and retention of urine. During the not uncommon remissions some of these symptoms can almost always be found. 3. *The terminal stage* is characterized by complete dementia (the final type of all initial varieties), complete disorder of speech, incoördination so extreme as to necessitate feeding the patient and keeping him in bed, sensory disturbances, vasomotor paralysis, grinding of the teeth, trophic complications and death from pyelonephritis, bed-sores, complications or bulbar symptoms.

Symptoms in Detail.—1. Psychical Symptoms.—The weakness, *alteration in character,* loss of memory and reason and the ethical, æsthetic and moral loss have been described. During the height of the disease there is one of three main mental manifestations: (*a*) *megalomania,* with simple exaltation or mania. This classical expansive type occurs in 66 per cent., is associated anatomically with the characteristic meningo-encephalitis, and is characterized by ideas of grandeur, the patient believing he is a king, God, a millionaire, an athlete, etc. He makes ridiculously large plans, buys extravagantly, lacks will power, indulges himself excessively, changes with great "facility" from one thing to another, is optimistic, friendly, even philanthropic and rapidly runs through his capital. He loses himself, forgets names and confuses the real with the unreal. Periods of acute mania, with fever and grinding of the teeth are expressions of brain congestion; complete dementia is the final outcome, even though remissions occur, during which some mental or somatic change can be detected. (*b*) The *melancholic* or hypochondriacal form is thought to occur with cystic degeneration of the cortex. It may be of the dull or agitated type. The patient dwells on the condition of his bowels; one subject thought they would "burst and flood the world." There is often *micromania,* characterized by "self-belittlement," in which the individual fears everything and thinks himself a "dot, nothing, or denies his existence." These cases may commit suicide. Dementia is the outcome. (*c*) *Simple primary dementia* may be present from the beginning; this type is increasing in frequency, while the first classical form is decreasing. It is thought to be based anatomically on simple cortical atrophy in which inflammation is absent or late and secondary. These subjects are often deceptive in the early stage, being quiet, urbane and optimistic. In the ultimate dementia of all types, mentality is wholly gone, "there is no sense of time, place or identity," and the patient babbles or is speechless.

2. Motor Disorder.—This usually occurs with mental changes, is constant, though variable in its expression, incomplete in degree, extensive as to parts involved and progressive. (*a*) *Speech* is pathognomonically disturbed as a result of mental change, wasting of asso-

ciation fibers and later bulbar atrophy. It is incoördinate, involving letters (*"literal" ataxia*) or syllables. In pronouncing such words as "electricity" the patient characteristically stutters over syllables. It is worse in the morning and after resting. Loss of word memory is the primary trouble; the patient forgets expressions, speaks ungrammatically and "does not notice it." Speech becomes atactic, there is paraphasia, the voice is unmodulated, gives out suddenly, is sometimes hoarse, nasal or slow, but never scanning. Later, speech is disturbed from bulbar alteration, causing dysarthria from disease of the facial and hypoglossal nuclei; it is ultimately unintelligible (absolute aphasia). Closely associated with speech disturbance is (*b*) *disordered reading* (paralexia), which is rather characteristic and occurs early. (*c*) *Writing* is disturbed first because of the mental alteration; the patient has difficulty in collecting his thoughts, although he does not realize the cause (the ataxia by which he drops syllables, repeats words or sentences—*paragraphia*); and it is disturbed secondly because of tremor. The writing is uneven, up and down or zig-zag. These changes are diagnostic. (*d*) *Paralysis of the eye muscles* (in 6 per cent., Marie) is usually partial and transitory. When total, syphilis or tabes is the probable cause. (*e*) The *pupils:* Myosis may occur, often just before maniacal outbursts. Mydriasis is frequently unilateral and results from sympathetic irritation. In 60 per cent. of cases the pupils are now large, now small, and this is considered as especially ominous. They are often irregular or triangular. The Argyll-Robertson pupil, responding to accommodation but not to light, usually appears early, is marked in 47 per cent., partial in 20 per cent. and absent in 33 per cent. Reichert holds that it is always due to degeneration in Bechterew's column (*i. e.,* between that of Goll and of Burdach). (*f*) The *facial nerve:* The paresis of the lips is cortical, as are fibrillary twitchings in speaking or mimetic movements, automatic chewing movements and almost constant grinding of the teeth. The "fatuous" or "masked" expression is psychical. Salivation results from cortical irritation. There may be difficulty in swallowing. (*g*) *The limbs:* (i) The cortical changes cause the small-waved, rapid, constant tremor, the ataxia, loss of muscle sense and the uncertain, ungraceful, tripping paralytic gait. (ii) The gait may be tabetic, with absent knee jerks, immobile pupils and vesical symptoms; or it is more often spastic with increased patellar reflexes, ankle clonus, contractures and changes in the joints. The patellars are usually increased in the early stages. (iii) Neuritic atrophy of the cranial nerves may occur as in tabes. (*h*) *Apoplectiform and epileptiform attacks* may occur. (i) Apoplectiform seizures result from vasomotor paralysis and brain œdema and differ from apoplexy in their incompleteness, evanescence, increased temperature and increased reflexes. They last part of an hour and may produce temporary hemiplegia, but especially aphasia with paresis of the right arm. (ii) Epileptiform attacks occur later, result directly from cortical disease, are usually partial (involving

the face or arm) and are either Jacksonian or are attended by partial loss of consciousness; conjugate deviation of the eyes and increased temperature are also observed. They are not frequent but, once initiated, recur and aggravate the mental alienation.

3. Sensation and Special Senses.—Coincident neurasthenia, tabes and pachymeningitis may emphasize the sensory manifestations, as the lancinating pains of the tabetic form. Sensation is often dulled and, like the analgesia, is cortical in origin. It is suggested that the hypochondriacal complaint that certain organs, as the bowels, are absent, is due to visceral anæsthesia. Attacks of migraine, when they first appear late in life, may indicate an organic origin, and Charcot described a form with hemianopsia and symptoms like glaucoma. Migraine is cortical when associated with paresis of the face, tongue or arm. Alteration of the special senses includes optic neuritis and atrophy (4 per cent.), optic hallucinations and psychical blindness.

4. Other Symptoms.—(*a*) *Vasomotor* changes occur in the skin (lividity, œdema and sweating) and in the brain, lungs, bladder, intestines and stomach (resembling the so-called bilious attacks). (*b*) *Trophic* symptoms are sweating of blood, rough skin, phosphaturia, perforating ulcer of the foot (3 per cent.), bed-sores and friability of the bones, which may be broken without the patient knowing it. The "insane ear" is frequent. Many of these symptoms are spinal and neuritic.

5. General Symptoms.—The pulse is often slow and monocrotic, the temperature low, although the heat centres may be involved in congestive seizures with great febrile elevation, and the body weight is often reduced in the early and late stages, though at the height of the disease it often increases, especially before the congestive seizures. The sexual instinct, at first increased and sometimes perverted, later declines. Insomnia may be severe, protracted or absolute.

Diagnosis.—The diagnosis depends upon (*a*) the *etiology,* as syphilis with mental strain or excesses; (*b*) the grouping of the *psychical symptoms,* as character, æsthetic or ethical changes with (*c*) *motor* symptoms, as the speech, writing or tremor; (*d*) *vasomotor* symptoms, as migraine or apoplectiform insults, and (*e*) the *progressive course.*

Differentiation.—1. Diffuse Brain Diseases.—(*a*) Cerebral *syphilis* may be confused with diffuse meningo-encephalitis.

Progressive Paralysis.	*vs.*	Syphilis.
Headache absent or vague and transitory.		Headache present and severe.
Psychical alteration characteristic; mental symptoms are coördinated.		Somnolence, variability in mental symptoms. Merely coincident.
Epileptiform mono- or hemiplegic seizures rare.		More frequent.
Focal symptoms very rare.		Focal symptoms common, together with diffuse symptoms.

Progressive Paralysis.	*vs.*	Syphilis.
Greater speech disturbance, literal paraphasia, and fibrillary tremor of face and tongue.		Less or no tremor; speech far less frequently disturbed; no literal paraphasia.
Argyll-Robertson pupil.		Absent; oculomotor affections and optic neuritis much commoner.
Progressive, slower. No therapeutic results.		In installments, more rapid jumps, remissions. Therapy helps or possibly cures.
A parasyphilitic disease.		Active syphilis.

(*b*) *Diffuse cerebral sclerosis* cannot be diagnosticated; though it leads to dementia and attacks of delirium, there is not the speech and mental alteration which is present in general paresis. (*c*) *Senile dementia* seldom occurs until after sixty years of age. Its course is longer, the symptoms more gradual and less intense, memory is maintained longer and there is less megalomania than fear of persecution and poisoning. (*d*) *Multiple sclerosis* occurs earlier, is not syphilitic, is complicated by late and mild mental symptoms and an intention tremor, which is wider in amplitude and ceases during repose. The speech is scanning, staccato, and there is no literal ataxia. Nystagmus is common, while it is rare in general paralysis. Motor symptoms are largely referable to lateral column disease.

2. Focal Brain Diseases.—(*a*) Although migraine, headaches, apoplectiform attacks and partial sensory or motor irritation of the Jacksonian type may occur in *brain tumor,* it does not cause difficulty in pronouncing syllables, peculiar mental alteration or immobile pupils; it is characterized by general symptoms, as choked disk or vomiting and focal symptoms, which are foreign to general paralysis. In tumor there is simple dulling of the intellect and dementia only with marked internal hydrocephalus. (*b*) Hemorrhagic pachymeningitis complicates 20 per cent. of cases of dementia paralytica; when it exists as an independent affection, it often leads to intense local headache, vomiting, recurrent hemiplegia or monoplegia, cortical irritation and more rapid course. (*c*) Softening usually produces focal symptoms.

3. Psychoses and Neuroses.—(*a*) *Mania* and *hypochondriasis.* Every case of mania or hypochondriasis between the twenty-fifth and forty-fifth years not due to alcoholism or to acute somatic disease should arouse suspicion of general paralysis (Mendel). (*b*) *Neurasthenia.* In it the onset is more acute; headache accompanies the lassitude and "irritable weakness," the memory is fatigued but never lost, the patient recognizes his incapacity; and oxalates and urates abound in the urine. In dementia paralytica there is phosphaturia, hypochondriasis with absurd conceptions, changes in the pupils, disk and field of vision, alterations in character, speech and intellect which are never seen in neurasthenia.

4. Intoxications.—These may cause many of the forms known as

general pseudoparalysis due to alcohol, lead, etc. (*a*) Chronic *alcoholism* is not progressive, recovery is frequent, delirium tremens and convulsions are common, the course is acute and the alcoholic tremor, headache, hallucinations and neuritis are found. (*b*) Chronic *plumbism* (saturnine encephalopathy) is often progressive, even fatal, and in its last stages indistinguishable from dementia paralytica. It is more acute and 80 per cent. of cases recover. There are headache, cardiac distress, anæmia and gingival lead line. (*c*) Chronic *bromism* is distinguished by its stupor, dementia, bromide acne, fœtid breath, coated tongue, pharyngeal anæsthesia, weak heart and acute and usually favorable course.

Prognosis.—The disease is fatal in 66 per cent. of cases within two years, though remissions may occur. Krafft-Ebbing saw no recoveries in 2,500 cases; recovery indicates an incorrect diagnosis, pseudoparalysis generalis. The following courses are distinguished: (*a*) *acute* or galloping form, which lasts months to a year; (*b*) *classical,* expansive type, which lasts three to four years; (*c*) the depressive type, which lasts two to three years; (*d*) dementia forms, which last four to five years; (*e*) circular form, in which depression alternates with mania; (*f*) female variety which has a longer course than in males; (*g*) spinal form, with a relatively long course; (*h*) juvenile or adolescent; and (*i*) senile form. Death results in 50 per cent. from the disease itself, by apoplectiform seizures, suicide, inanition or bulbar symptoms. In 50 per cent. it results from complications, tuberculosis (18 per cent.), bed-sores, with sepsis (10 per cent.), lung gangrene (6 per cent.), pneumonia (5 per cent.), choking (10 per cent.) and cystitis.

Treatment.—(*a*) *Etiological* treatment. Recovery is barely possible in the early stages and many physicians advise that mercurial inunctions be given. The disease is a parasyphilitic disease, not active syphilis and some consider mercury distinctly injurious. Iodides promote resorption and favor a milder course. (*b*) *Hygienic* treatment, as rest, protection from heat, and quiet surroundings, is indicated. (*c*) The patient should be put in an asylum, for suicide or other violence is always possible. In the simple dementia form alone may the patient be treated at home. (*d*) *Hydrotherapy* is valuable within limits. Cold rubs lessen excitation, although full cold baths are contra-indicated. (*e*) *Medicinal* treatment is unsatisfactory. Drugs produce disproportionate effects and must be exhibited with caution. For delirium and insomnia bromides are most efficacious, but chloral and opiates should be given less frequently or only in combination with hyoscine, digitalis and bromides, or with the ice-cap. Ergot is said to produce remissions, but is valuable chiefly as a vascular tonic for brain congestion. (*f*) The *skin, bladder* and rectum should receive treatment as in apoplexy and spinal diseases.

DEGENERATION OF THE BRAIN.

I. Cystic Degeneration.—Cystic degeneration occurs mostly in the shrunken brain of aged persons and largely in the perivascular spaces of the cortical and basal white substance. No essential symptoms result from the fluid; they are those only of the causal atrophy. Possibly the vessels are prone to dilate and rupture from shrinkage of the brain and consequent lack of support.

II. Chronic Progressive Softening.—Chronic progressive softening, when not due to vascular disease, is a rare affection; it was described by Wernicke. It affects the white matter chiefly, occurs mostly after the sixtieth year, its pathogenesis is obscure and its clinical symptoms are weakness on one side, slight irritative phenomena, as tingling or pain, and involvement of the intellect. The course varies from a month to a few years and remissions are uncommon.

III. Multiple or Insular Sclerosis.—(See SPINAL CORD.)

IV. Diffuse Sclerosis.—This may involve one or both hemispheres of the brain or cerebellum. (*a*) It is often secondary to meningeal hemorrhages at birth, infantile hemiplegia, arrest of development and trauma. In these cases the sclerosis is secondary to brain atrophy. (*b*) Sclerosis without atrophy may develop in congenital syphilis, chronic alcoholism or contracted kidney. The symptoms are indefinite. Hemiplegia, impaired mentality, convulsions, contractures, spontaneous movements, vertigo, syncopal attacks and slow speech have been noted. Miliary sclerosis is very rare and consists of minute foci in the cortex, where the gray and white substance join. Many reported cases are due to artefacts in hardening.

V. Atrophy of Brain.—Genuine atrophy is always acquired. It is often associated with microcephaly and the brain is usually normal. Unilateral atrophy is observed in some cases of chronic insanity. Local atrophy may occur anywhere, is usually associated with sclerosis (*q. v.*) or neuroglia increase, is usually acquired and is very often secondary to tráuma, tumor or hydrocephalus. Some reported cases are clearly agenesis and arrested development. The symptoms are not distinctive. Hemiplegia, hemiathetosis and imbecility may accompany the infantile forms; they occur bilaterally when both hemispheres are diseased. *Senile atrophy* occurs in old age, when the brain becomes smaller and firmer and *hydrops ex vacuo* occurs in direct proportion to the shrinkage. Mental changes are not as common as is usually stated.

VI. Hypertrophy of the Brain.—This is most infrequent. Increase of the neuroglia is sometimes present and so-called local hypertrophy is frequently only gliomatous infiltration. Hypertrophy occurs at birth, in the first year, when it is often associated with rickets, and sometimes in older subjects. The skull is enlarged, the ventricles decreased and the convolutions flattened. Coincident hypertrophy of the lymphatic glands, thyroid and thymus has been observed. The diagnosis is impossible.

VII. Chronic Bulbar Paralysis.—Although mentioned by Robinson (1815) and Dumesnil (1859), this affection was first thoroughly described by Duchenne (1860), as labioglossopharyngeal (laryngeal) paralysis, and his observations were confirmed by Trousseau, who made the first autopsies. Wachsmuth (1864) advanced the name, progressive bulbar paralysis, and Charcot and v. Leyden demonstrated alteration in the bulbar nuclei.

Etiology.—Most cases occur in persons between *fifty and seventy years* of age. Heredity is a factor only when the disease is associated with progressive muscular atrophy. Sixty-four per cent. of cases occur *in males.* Trauma, cold, infection, mental or physical exhaustion and toxæmias are uncertain factors. Rheinhardt holds that it may follow acute inflammation of the medulla.

Pathology.—To the naked eye the medulla usually appears normal, though sometimes shrunken. Microscopically, (*a*) the *motor nuclei* are symmetrically and bilaterally wasted, their cells and processes shrunken. The hypoglossal nucleus suffers most and few normal cells remain; its accessory nucleus is usually normal. The spinal accessory nucleus is next most frequently affected, but the vagus nucleus suffers less. Degenerative atrophy may invade the glossopharyngeal or exceptionally the facial and fifth nuclei. In some cases perivascular exudation occurs within the nuclei and the nerve elements are degenerated (the parenchymatous form), but in others increase of connective tissue or vascular thickening may be seen. (*b*) The respective *nerve trunks* in their course or in their terminal endings show parenchymatous and interstitial change, even within the medulla, and the posterior longitudinal fibers waste in direct ratio to the hypoglossal nuclear atrophy, especially those which form the anterior fundamental fibers of the spinal cord. Sometimes the loop of the seventh nerve wastes. (*c*) The *muscles* supplied by the above nerves waste, as those of the lips, tongue, palate and larynx (the process sometimes extends to the neck and arms). The greatest change is in the tip of the tongue. The changes are those of progressive muscular atrophy, *i. e.,* atrophy unequal in its distribution, granular, fatty or waxy alteration, increased nuclei in the muscular sheaths and in the connective tissue, reddish pigmentation, and sometimes overgrowth of the fatty tissue. If the nuclei of the medulla are the sole seat of the disease, the disease runs a rapid course of a few months to a year (Remak). (*d*) In many cases *pyramidal tract degeneration* may be traced down into the cord or up into the crus.

Symptoms.—Labioglossopharyngeal paralysis, the name proposed by Duchenne, designates the parts paralyzed by the wasting of the motor nuclei. It is almost invariably bilateral and but one case showed unilateral disease. It is slow in onset, afebrile and painless. The first symptom is usually *paresis of the tongue* (hypoglossus) which first shows fatigue and much later actual paralysis. It is difficult to protrude or move up, down or transversely. The linguals, L, N, R and S are formed with difficulty and also later the lingual

palatines, as T and D. The tongue is most often wrinkled and wasted. The *lips* become weak and whistling and formation of the labials (O, U, P, B, M) become impossible. The tongue and lips are more closely associated than any other muscles of the body; anatomically it is probable that the facial fibers for the orbicularis oris originate in or very close to the hypoglossus nucleus. Forced attention may for a while overcome imperfect enunciation, but later the paralysis is complete, the mouth cannot be closed, the lower lip sags and the zygomatic muscles overcome the weak fibers of the upper lip and accentuate the nasolabial furrow. The lips are frequently thin. The expression is peculiar, the forehead is corrugated, the eyebrows somewhat lifted, but the mouth is passive, its expression is "dead" and laughing and emotional displays produce a strange effect, which Trousseau compared to the mask of the Greek actors. The original descriptions graphically picture the patient constantly holding a handkerchief before the lips to collect the free flow of saliva from the open mouth; the salivary secretion is probably also increased from disease of its bulbar centre. The chin muscles are weak. *Swallowing becomes difficult* from the inability of the tongue to propel the food to the pharynx, so that the subject must push the food back with the finger, and again the palate (controlled by the spinal accessory and vagus nerves) is weakened, producing a nasal voice and allowing food to regurgitate into the nose. The *pharynx is also paretic* (supplied by the glossopharyngeal and vagus nerves) and the larynx suffers (spinal accessory and vagus nerves). Semi-solid food can best be swallowed, because fluid more easily regurgitates into the nose and solids more easily reach the larynx. The *larynx* is often involved. The adductors are much more often affected than the abductors, but the palsy is usually incomplete. Talking and coughing are difficult or impossible. *Fibrillary twitchings* are very common, sensation is normal, but the reflexes of the skin and mucosa are usually lost. The electrical reactions are seldom much changed, though there is sometimes a partial or mixed form of reaction of degeneration. The intellect is normal. The patient may be somewhat emotional and forced laughing and weeping are not uncommon. The general nutrition suffers from the dysphagia. In some cases the process extends beyond the nuclei mentioned; the *eyes* may be involved, as in progressive nuclear ophthalmoplegia (which is an identical process in the pons), chiefly in young subjects and in "family forms"; occasionally the cervical, facial, masseteric, temporal and pterygoid muscles are involved; affection of the latter was considered ominous by Duchenne. The optic nerve is very rarely implicated. Bulbar paralysis frequently co-exists with progressive muscular atrophy of spinal origin, affecting the limbs or trunk, and Kussmaul first pointed out that the two processes are identical in nature and differ only in location. The spinal disease may progress upward to the medulla, causing secondary bulbar palsy, or the bulbar palsy may be primary, followed by the secondary and analogous spinal atrophy. In typical bulbar

paralysis there are wasting, paralysis and absence of the reflexes. In some atypical forms there is paralysis but no wasting, and at times the reflexes are increased (clonus in the muscles of mastication); in these cases it is probable that the lesion is in the supranuclear fibers, "the bulbar analogue of amyotrophic lateral sclerosis of the cord," with which it may also be combined.

In the *progressive course* of the malady the pulse becomes rapid (120 to 150) and irregular; tachycardia is often preceded by running together of the heart tones, which Duchenne compared to the "heart beating in water." Respiration is difficult, dyspnœa develops and the expiration is weak.

Diagnosis.—This is based on (*a*) the distribution of the process in the motor nuclei; (*b*) its bilateral symmetry; (*c*) gradual onset, and (*d*) chronic progression. *Neuritis* of the bulbar nerves is very rare; the lips escape, because the nerve fibers to the orbicularis oris, arising in or near the hypoglossus nuclei run to the pons and leave it by the facial nerve fibers; there is more atrophy, more rapid course, more sensory disturbance and the reaction of degeneration is present. It occurs in acute infections, etc. *Brain tumor within the medulla* occurs most often in young subjects and gives the general and local signs of tumor; growths, exostoses, dislocations, caries or aneurysms *without the medulla* but rarely produce bilateral or exactly symmetrical paralyses; the lips escape, dysphagia is the leading symptom and other symptoms foreign to bulbar palsy develop, as convulsions, vomiting or hemiplegia (see BRAIN TUMORS, page 960). The bulbar symptoms of multiple sclerosis and syringomyelia are almost never symmetrical. *Pseudobulbar paralysis* may be confused. Bilateral disease of the cerebral hemispheres, sometimes in the cortex, but more often in or near the internal capsule, especially when due to arterial lesions, may cause dysarthria (Joffroy, 1872). One lesion may be in the cortex and the other in the lenticulostriate area (Halipré); Jolly found this condition in symmetrical sclerosis of both hemispheres. Pseudobulbar palsy causes cerebral symptoms, as mental change, aphasia, hemiplegia, double facial paralysis and hemianopsia; one side is involved after the other and it is attended by no wasting, no loss of reflex action and no reaction of degeneration.

Prognosis.—The disease is almost invariably fatal in one to three years, especially in advanced life or when co-existent with spinal muscular atrophy. Remissions are possible, and in the rarest instances the disease may be arrested in youth or middle life. Death results from inanition, inhalation pneumonia, intercurrent disease or paralysis of the cardiac or respiratory centres.

Treatment.—Treatment is discouraging. Nutrition must be maintained by feeding with the nasal catheter, to which a funnel is attached. Iron and strychnine may be given. Electrotherapy is of little value. Faradization of the neck and tongue may help deglutition; the positive pole should be applied to the spine and the negative pole to the throat. The disease steadily progresses in spite of treatment.

Asthenic Bulbar Palsy; Myasthenia Gravis.—Chronic bulbar palsy *without anatomical lesion* may be discussed in this connection. It was first described by Wilkes (1877), then by Erb (1879), who recognized its light forms, and Goldflam (1891), who with Oppenheim published reports of severe and fatal cases. It is sometimes known as the Erb-Goldflam syndrome. E. Bramwell has collected 90 cases. Its etiology is unknown, but it occurs most often in persons under the thirtieth year, and in 25 per cent. a neuropathic heredity is demonstrable. The cardinal feature is *extreme exhaustion* (*myasthenia*) *after the slightest exertion.* Weakness slowly develops in the eyelids, face, palate, masseters, pharynx, vocal cords and the tongue, although but slightly expressed by ptosis (the first symptom in 33 per cent. of cases), dysphagia, dysarthria and difficult mastication, and disappears after rest, to return anew after a few muscular efforts. In six cases myasthenia was confined to the eyes. From these bulbar symptoms the affection is also called *asthenic bulbar paralysis.* In some cases the arms, legs and trunk are similarly involved, so that walking or arm movements become impossible after short exertion; this, together with the bulbar symptoms, is known as *myasthenia gravis pseudoparalytica.* The symptoms are symmetrical. The *myasthenic reaction of Jolly* consists of normal tetanic response to faradic stimulation, but becoming weaker with each repetition, until at last no reaction is elicited. The reflexes are normal or perhaps exaggerated but soon also become exhausted. Some atonicity of the digestive tract is fairly common. The mind, special senses, sensation, muscles and sphincters are usually normal, although Collins noted a rapid exhaustion of the special senses and Buzzard remarked sensory alterations. Remissions are usual, four years being the maximum clinical duration. *About 40 per cent. of cases die* from exhaustion of the cardiac and respiratory centres but the *autopsy* findings are *negative* (in 66 per cent.) or doubtful. Kalisher found hemorrhages in the medulla, and C. Mayer found vascular and nuclear alterations. Lymphoid cells have been found between the muscle bundles, and Hun and Blumer describe lymphoid and glandular hyperplasia in the thymus gland. The *diagnosis* is based on (*a*) absence of atrophy; (*b*) absence of reaction of degeneration; (*c*) absence of twitchings and sensory phenomena; (*d*) the presence of muscular adynamia occurring with (*e*) remissions in the course of the disease; (*f*) Jolly's myasthenic reaction; (*g*) the slight involvement of the hypoglossus.

Treatment.—Rest and massage are helpful, but iodides and strychnine are less useful. Galvanization of the head and neck is recommended by Goldflam, who has reported recovery from its use.

Apoplectiform Bulbar Paralysis.—Sudden apoplectiform bulbar paralysis does not properly come under degenerative brain disease, but deserves discussion in this place for the sake of topical differentiation. Its usual *causes* are hemorrhage, embolism, thrombosis and

less frequently bulbar encephalitis, trauma or compression, bulbar neuritis, the ascending paralysis of Landry and paralysis after acute infections.

Symptoms.—Its symptoms are as follows: (*a*) The *onset* is apoplectiform with symptoms which are rarely focal, as vertigo, vomiting, dyspnœa, yawning, slowed heart, which often occur without coma. They may progress or regress. (*b*) *Motor symptoms* are frequent, as *double hemiplegia;* monoplegia is possible, or if the lesion occurs at the pyramidal decussation there may be hemiplegia cruciata, *i. e.,* paralysis of one arm and the opposite leg. A unilateral focus may produce bilateral symptoms rarely, however, symmetrical. The pyramidal tracts in the medulla are supplied by the inferior cerebellar artery. Descending pyramidal degeneration follows severe involvement of these tracts. (*c*) *Sensory symptoms* may develop, as hemiataxia or hemianæsthesia (possibly without disturbance of muscle sense). (*d*) The *bulbar nerves* may be affected; paralysis of the tongue, pharynx, masseters, larynx and, because of the vascular supply, frequently of the eyes, face and other pons centres may result. The reflexes of these parts are often suspended, though in the extremities they are increased. The lesion may be at the level of the nuclei or supranuclear. The hypoglossal and spinal accessory nerves are supplied by the anterior spinal and vertebral arteries; the vagus, glossopharyngeal and auditory nerves by the vertebral; the fifth, seventh and eye-nerves (third, fourth and sixth) by the basilar artery. There may be hemiplegia with paralysis of the opposite side of the tongue if the lesion is in the anterior part of the medulla.

Diagnosis.—The diagnosis is usually easy from the acuity of the lesion in the medulla. *Chronic bulbar palsy* is excluded by the onset and the asymmetry and irregularity of the bulbar symptoms, as double hemiplegia, which is unequal on the two sides, or dysphagia without paralysis of the tongue or lip. Exact differentiation between hemorrhage, embolism and thrombosis is frequently impracticable. *Pseudobulbar paralysis of cerebral origin* presents the characters of disease of the hemispheres and is clearest when one attack is followed by a second. Differentiation is impossible in simultaneous lesions.

Prognosis and Treatment.—The *prognosis* is most grave at the onset, but later there is less danger of progression. Improvement and even recovery are possible. *Treatment* concerns chiefly the underlying vascular disease, as in hemorrhage or embolism (*q. v.*).

VIII. Progressive Nuclear Ophthalmoplegia.—This is a degenerative atrophy of the oculomotor nuclei, similar pathologically to chronic bulbar paralysis. It is more conveniently considered under affections of the third nerve (*q. v.*).

HYDROCEPHALUS.

Acute hydrocephalus is an acute accumulation of fluid either between the dura and the brain (acute external hydrocephalus) or in

the ventricles (acute internal hydrocephalus). In some cases the fluid is a *transudate,* being part of a general œdema, resulting from cardiac or renal disease, or due to local causes, as sinus thrombosis. In most cases it is an *exudate.* Acute meningitis is the only certain cause of acute hydrocephalus and is tuberculous in 80 per cent.; it is less often the serous meningitis of Billroth and Quincke. It is difficult to distinguish between transudation and exudation, and diagnosis is uncertain. Quincke thinks some cases are angioneurotic.

I. Chronic Hydrocephalus.—*External form* (fluid between the dura and brain).—The causes are (*a*) compensatory *hydrops ex vacuo,* occurring where brain development is arrested (the congenital form) or when the brain wastes (paretic dementia, softening, hemorrhage, sclerosis, senile atrophy, porencephaly). The hydrops transudate under the dura in the arachnoid meshes or between the pia and brain has no clinical importance. (*b*) *Stasis,* as in sinus thrombosis; and (*c*) *chronic diseases,* as cancer, nephritis or alcoholism are also causes. The symptoms are indeterminate.

II. Chronic Congenital Internal Hydrocephalus.—**Etiology.**—The etiology is uncertain. It has been thought that the cause was fœtal meningitis, inflammation in or obstruction of the choroid plexus, or obstruction in the branches of the vena Galeni, in the lymphatics, lateral apertures of the fourth ventricle near the glossopharyngeal roots, aqueduct of Sylvius or in the foramen of Magendie or Luschka. Hereditary influences and syphilis (10 per cent.) are sometimes suggested; parental alcoholism and trauma are questionable causes.

Symptoms and Pathology.—The large head may be an obstacle to labor. It may measure over 5 feet (167 cm.) in circumference. If it appears after the seventh year the head is not enlarged, and in cretins the head may be undersized. Often the head cannot be held upright. The skull is thin, even membranous and translucent to the candle test. If the disease halts, the bones may thicken and Wormian bones develop. The head projects beyond the base, the sphenoid is luxated forward and downward, the ear meatus points downward, the occiput is forced back, the sella turcica is flattened and widened, the roof of the orbit may be pushed down so as to become palpable, the fontanelles are large, the fissures gape and fluctuation may be felt. The hair is thin, the veins are large and a systolic murmur may be heard, the cause of which is doubtful. The *brain is pale* and may be flattened almost beyond recognition. The membranes are often thickened and the ependyma thick, granular and sclerotic. The average amount of fluid is 1 quart, the maximum 13 quarts. The ventricles, especially the lateral, are enlarged; sometimes the third or fourth ventricle escapes. The enlarged third ventricle easily compresses the optic chiasm. The foramen of Monroe is often very large. The fluid is usually clear, sometimes slightly yellow and rarely blood-stained. It may contain epithelial flocculi; it is neutral or alkaline, its specific gravity is 1,001 to 1,009; and the albumin is 0.2 to 2.0

per mille; more than 2.5 per mille of albumin or a specific gravity higher than 1,009 indicates inflammation.

The *face is small,* triangular, and the eyes are prominent. Much of the sclera is shown and the upper lid is small, though the lower lid may cover much of the iris. *Nervous symptoms* are not unexpected, for the brain may be reduced to a small fraction of an inch in thickness and is a mere flask for the fluid. The demarkation between gray and white matter is lost and in marked cases the convolutions and sulci are obliterated, the basal ganglia flattened, the cerebellum forced into the spinal canal and many structures, such as the fornix or corpus callosum have disappeared. *Mentality,* as a rule, is impaired or abolished, speaking is learned late if at all, will power is lacking and irritability is common. Epileptiform attacks, psychoses, headache and vomiting are fairly common. Eye symptoms are frequent, as diminished vision or blindness from initial optic hyperæmia and subsequent atrophy, strabismus, nystagmus and wide, possibly reactionless, pupils. Other special senses suffer. All the limbs, or the legs alone, often present spasticity, contractures, paresis and increased reflexes, if not secondary degeneration in the pyramidal tracts. The child walks late, if at all. Sensation is practically normal. Puberty may be delayed or the infantile testes may persist. Lumbar puncture shows increased intraspinal pressure (36 mm. of mercury).

Complications.—Complications are largely anomalies of development, as syringomyelia, hydromyelus meningocele, microcephaly, porencephaly, anencephaly and encephalocele.

Course.—The course varies considerably. Some subjects die during or after birth. Many die in the first three years from increase of brain pressure with coma, intercurrent disease or diarrhœa. In some cases temporary remissions are seen, and in still rarer instances permanent arrest of the process occurs, the mind improves, the speech shows some defects or the gait remains paretic and spastic. In very exceptional cases rupture externally is observed after head trauma or spontaneous breaking into the ear or nose occurs, as in Lebert's case, which "leaked" for five years. Gall reported a case which lived fifty years, and Gölis one of seventy-one years duration.

III. Acquired Chronic Hydrocephalus.—Etiology.—The causes are (*a*) *stasis,* either general (cardiac or pulmonary) or local (brain tumor or sinus thrombosis); (*b*) *cachectic* transudation, as in carcinoma or nephritis; and (*c*) *inflammation,* beginning as acute meningitis of the sporadic or epidemic type, or as slowly beginning chronic basal meningitis, especially in young children. The same points of obstruction may exist as in the congenital variety. (*d*) Rickets is possibly a cause or frequent associate. Some forms may be angioneurotic. Some cases show nothing at autopsy.

Symptoms.—Meningitic symptoms usher in some cases in which after weeks new evidences of brain pressure appear, as alteration in the pulse and breathing, unequal pupils or coma. Headache, vomiting, visual disturbance, spastic paresis, epilepsy, altered mentality,

idiocy or involvement of the cranial nerves are seen, as in the chronic congenital form. The shape of the head, however, is not altered.

Diagnosis of the chronic congenital and acquired forms.—Differentiation between the two forms is very difficult, unless there is an accurate history. (*a*) From *rhachitis:* The two affections are often associated, for some unknown reason. The rhachitic head is square rather than globular, it protrudes more in front and laterally than behind, the sutures are less widely separated, the fontanelles are wide but do not bulge, and there are no signs of brain tension. The rhachitic head should be measured, because it seems larger than it actually is. (*b*) From *brain tumor:* The two conditions are sometimes coincident. In each, symptoms of brain pressure are observed. The congenital form is more easily distinguished by the history of the disease from birth and its slower course, but focal symptoms are less common, as hemiplegia, monoplegia and hemianæsthesia, and spinal symptoms are more common than in tumor. (*c*) *Hyperostosis* of the skull (Paget's disease), brain hypertrophy and acromegaly increase the size of the head, but are separable by the same arguments as in rhachitis.

Treatment.—Few therapeutic results can be obtained. *Bandaging* the head is sometimes helpful, but must be carefully done, lest it aggravate the brain compression. *Puncture* of the *lateral ventricles* dates from the time of Hippocrates. Isolated recoveries are recorded, but the evacuation must be slow. Permanent drainage is usually fatal, but occasionally has resulted in cure. Attempts to obliterate the ventricles have recently been made, as in Mikulicz's, Troje's and Leur's efforts to effect anastomosis between the ventricles and the subdural space; McArthur was successful in three cases. Lumbar puncture in acquired forms may be useful; death may result.

DISEASES OF THE CEREBRAL MENINGES.

Inflammation of the dura is termed pachymeningitis, and inflammation of the arachnoid and pia, leptomeningitis or meningitis.

PACHYMENINGITIS.

External pachymeningitis is a surgical disease, being secondary to skull disease (syphilis, trauma), infections and cerebral atrophy. Its symptoms are indefinite, as pain, headache, possibly hemiplegia, or are those of the primary lesion; diagnosis is difficult. Surgical treatment in Macewen's 22 cases gave 100 per cent. recovery.

Internal pachymeningitis is divided into (*a*) the purulent form, which follows bone disease or leptomeningitis; and (*b*) the pseudo-

membranous type, sometimes with fluid between its layers, known as serous pachymeningitis and seen chiefly in general paralysis or in children with perisplenitis. These varieties are not as important as (*c*) the third variety, known as *pachymeningitis hemorrhagica interna,* described by Heschl (1855), and a year later named by Virchow; he regarded the inflammation as primary and the hemorrhage as secondary. Kremiansky (1868) developed the etiology and produced the disease in dogs by administration of alcohol.

Etiology.—(*a*) Most cases occur in *middle and advanced life,* although a few are seen in marantic children. Fifty per cent. occur in persons over fifty years of age, 40 per cent. in those over sixty and 25 per cent. in those over seventy (Gowers). (*b*) Seventy-seven per cent. of cases occur in *males.* (*c*) *Paretic dementia* causes 19 per cent. Pachymeningitis is common in *other dementias* and chronic psychoses, whence its far greater frequency in asylums and poorhouses than in private or hospital practice. It also occurs in arteriosclerosis and encephalomalacia. (*d*) *Chronic alcoholism,* frequently with cardiac and renal disease, is a more frequent cause than the statistics of König show, *viz.,* 6 per cent. (*e*) *Trauma,* as during birth, from forceps delivery or from small pelvis. (*f*) *Hemorrhagic diathesis* (scurvy, hæmophilia, leukæmia, pernicious anæmia), chronic nephritis. (*g*) *Infective diseases,* as typhoid, scarlatina, puerperal fever (9 per cent.), heart disease (18 per cent.), hereditary syphilis (11 per cent.), tuberculosis (23 per cent.), bronchitis and pertussis are also causes.

Pathology and Pathogenesis. — 1. Gross Pathology. — In slight cases the inner surface of the dura is cloudy, yellow or brown from punctate hemorrhages or hæmatoidin deposits. In pronounced cases the dura shows membranous laminæ, which are adherent by proliferation of bloodvessels and by organization. The laminæ may be a quarter of an inch thick. Those layers nearest the brain are the most newly formed. Repeated hemorrhage into the layers of the newly formed tissue or between them is very frequent, and may be an inch thick or reach the size of an egg. The free hemorrhage may spread over the entire surface of the brain, even reaching the retina; older foci are apt to be encapsulated. Cyst formation in the clot, suppuration and calcification are rare issues. The leptomeninges are tinged and often somewhat inflamed. Corresponding to the most frequent seat of pachymeningitis, the convexity of the brain is compressed, especially over the motor areas. The process is bilateral in 56 per cent. of cases and the basal cranial nerves are seldom implicated.

2. Minute Examination.—The fresh fibrin of the earliest hemorrhage and the exudation contain red cells and, nearest the brain, a covering of endothelial cells. Later the exuded fibrin develops capillaries, organizes and again hemorrhage occurs from the newly formed vessels. This organizes in turn, so that successive laminæ develop. The older layers become firm and fibrous.

3. Pathogenesis.—Virchow viewed the affection as primary

pachymeningitis, but Spierling and Hueguenin held that the hemorrhage was primary. Jores has proven that two classes of cases exist: (*a*) In traumatic cases the hemorrhage is primary and a simple clot forms which later organizes into firm connective tissue; this variety is called *regressive.* The same sequence is thought to occur in cases due to the blood diseases, acute infections and arteriosclerosis. (*b*) In chronic pachymeningitis proper the inflammation is primary, the process is *progressive,* the connective tissue formed in layers is looser, more vascular, and its inner, successively formed layers are the seat of repeated hemorrhages.

Symptoms.—In most cases there are no symptoms, because the hemorrhage is slight or the pachymeningitis is overshadowed by the original disease.

1. General Symptoms.—The most frequent is headache, which is *per se* of little differential value. It is diffuse and is most marked in alcoholics. The symptoms increase in value when mental dulness or irritation and coma develop with focal signs (*v. i.*). There may be prodromal delirium. The pulse is sometimes slow and irregular. Choked disk is not rare. Moderate albuminuria sometimes occurs. The temperature may be slightly elevated.

2. Focal Signs.—The most important is *hemiplegia,* which is preceded for some time by coma (Fürstner). The paralysis is contralateral, rarely complete, varies with increase or decrease of the clot, often involves the cortical facial and hypoglossal centres, is sometimes bilateral and associated with aphasia (33 per cent.), hemihypæsthesia (especially involving the muscle sense, causing hemiataxia), hemiamblyopia, contractures in the paretic members and Jacksonian epilepsy. Conjugate deviation of the head and eyes, nystagmus and forced postures may be observed. The patient may grasp at his clothes or at his hair or beard (Fürstner). The pupil contralateral to the lesion is usually larger (Griesinger); the pupils react poorly and later both are wide. The tendon reflexes are increased, but the skin reflexes are decreased or abolished during the coma. The basal nerves are very rarely involved; trismus and rigidity of the neck are uncommon.

Course and Prognosis.—Death may occur in the first seizure or the symptoms may subside for months, years or possibly permanently. Residual signs are common and include headache, ataxia, psychical alteration, paresis and aphasia. Recurrence is frequent, with stupor, coma and hemiplegia until death occurs (*a*) from the fundamental disease, (*b*) from hemorrhage or (*c*) from intercurrent affections. The outlook depends on the etiology.

Diagnosis.—A correct diagnosis is rare. A patient with paretic dementia may die from a pseudo-apoplectic shock or from pachymeningitis or a nephritic subject may succumb to pachymeningitic hemorrhage, uræmia, brain œdema or heart failure. A presumptive diagnosis can be made from (*a*) the age, (*b*) causal disease (paretic dementia, alcoholism), (*c*) preceding dulness or coma, (*d*) hemi-

plegia, or (*e*) irritative phenomena (small pupils and convulsions), (*f*) from variability and (*g*) recurrence of the symptoms.

Differentiation.—It should be distinguished from *localized brain disease,* as (*a*) paretic dementia (*q. v.*) and (*b*) cerebral hemorrhage. Differentiation may be impossible, especially when pachymeningitic hemorrhage is very large. The etiology is different, and in pachymeningitis there is more cortical irritation, the coma and paralysis are more variable and temperature, narrow pupils and optic neuritis are more common. (*c*) In encephalomalacia, the hemiplegia is more often attended by aphasia and hemianæsthesia, and there is less cerebral irritation. (*d*) Brain tumor is easily distinguished. Apoplectiform attacks and progression with remissions occur in both affections. Tumor appears earlier in life and focal signs, intense headache and choked disk are much more common.

Treatment.—Treatment should be (*a*) prophylactic and etiological; and (*b*) that of the seizure. The use of the ice-cap, quiet, elevation of the head, application of leeches back of the ear, and possibly venesection are indicated. Nourishment should be given by rectum. Morphine and chloral relieve the headache, excitement or convulsions. Lumbar puncture has not proven beneficial (Ricken). Operation was first performed by Ceci in traumatic cases and by Annandale, Jaboulai, Monroe and Ballard in other varieties. In the future surgery will probably benefit a larger number of cases.

MENINGEAL HEMORRHAGE.

Meningeal apoplexy may be extradural, subdural, arachnoideal, subarachnoideal and subpial.

Etiology and Pathology.—(*a*) *Trauma* may rupture the anterior, middle or posterior meningeal arteries, with or without fracture of the skull. Rupture occurs usually at the site of trauma, but sometimes by *contre-coup.* The corresponding veins may rupture, also the sinuses or the pial vessels, from the latter of which blood may percolate to the base of the brain, affecting the basal nerves, or reach the spine. The internal carotid may be ruptured and death then occurs precipitately. External hemorrhage is either diffuse or circumscribed and results most frequently from middle meningeal rupture. The blood clot may weigh 3 to 17 ounces or reach the size of the fist, compressing the brain and dislocating the falx. If the dura is injured, blood may be found in its substance. *During birth* (see Infantile Cerebral Paralysis) trauma may be a factor and hemorrhage has even occurred during gestation from maternal trauma, but asphyxia and precipitate labor are more important; large symmetrical hemorrhage of the convexity is common. (*b*) *Arteriosclerosis* and rupture of cerebral aneurysms; (*c*) acute infections, hemorrhagic diathesis, sinus thrombosis and rarely heart and lung disease; (*d*) 50 per cent. of cases occur under forty years of age. Small clots may be absorbed or microgyria, porencephalia, cysts or brain sclerosis may result.

Symptoms.—Clinical latency is not rare in the new-born. Small hemorrhage in the adult may produce no symptoms. Very large ones result in early sudden death. In rupture of the *middle meningeal* artery there are (*a*) symptoms of brain pressure with those of brain concussion or contusion, as loss of consciousness, irritative symptoms, increased breathing, slowed pulse, pallor, vomiting and slight fever (100° to 101°). Either death or improvement may result. (*b*) In other cases improvement from the concussion and contusion occurs; there may be an interval of hours to often of days when *hemorrhage occurs or recurs,* with coma, pressure symptoms, mono- or hemiplegia and convulsions, as in pachymeningitis. Rigidity is less frequent than in meningitis. The pupils are variable. Hemorrhage of the sinuses and pia are said to cause monoplegia frequently; the brain nerves are sometimes reached by infiltration to the base. In the new-born there are asphyxia, slow then rapid pulse and coma, followed later by epilepsy, idiocy and spastic paralyses.

Differentiation.—*From concussion:* an exact diagnosis is possible only after trephining or by the clinical evolution. From *cerebral hemorrhage:* the course is very rapid. Differentiation from slow, ingravescent cerebral hemorrhage is impossible.

Prognosis and Treatment.—Ninety per cent. of cases die from expectant treatment (Wiesmann); 33 per cent. die in twenty-four hours and, when the hemorrhage is due to aneurysmal rupture, 30 per cent. die in ten hours (Lebert). Early surgical treatment is indicated; this was especially described by Krönlein.

ACUTE SUPPURATIVE LEPTOMENINGITIS.

In 1768, Whytt noted the acute hydrocephalic symptoms of meningitis, but failed to recognize the meningitis, which Quinn (1779) described. In 1855 Rilliet and Barthez distinguished the suppurative, secondary, tuberculous and other forms.

Etiology.—Suppurative meningitis is generally a secondary process.

1. Trauma.—Microörganisms from the ear or nose may find ready access to the brain when its resistance is lessened by trauma.

2. Regional Extension.—(*a*) From the *ear:* acute or chronic disease of the middle ear, antrum or petrous bone may result in suppurative or serous inflammation (see Brain Abscess). (*b*) From the *nose:* extension by this route is less common. The disease may be acute or chronic, spontaneous or operative (by sounding with a probe), syphilitic, tuberculous, meningococcic, neoplastic or suppurative, of the nasal mucosa or of the ethmoidal, sphenoidal or antral cells. Infection may travel through the bone, along the veins to the dura or along the olfactory nerve endings. (*c*) From the *eye:* the avenues to the brain are numerous, and yet disease of the eye itself, even panophthalmitis is less important than orbital cellulitis or enucleation, which exposes the sheath of the optic nerve. (*d*) Extension may result from *parotitis,* carbuncles of the face, whose veins com-

municate with the cavernous sinus; scalp affections; erysipelas; bone disease; and sinus thrombosis.

3. Metastatic or Secondary Extension.—(*a*) *Septicopyæmia,* from lung suppuration, empyema, arthritis, endocarditis and kindred affections; (*b*) *pneumonia* (about 0.5 per cent. in 5,540 cases); and (*c*) rarely exanthemata may produce it. In this group there is probably an intermediate link, as otitis media in scarlatina and empyema or lung infarct in typhoid.

Bacteriology.—(*a*) The *pneumococcus* is probably the most frequent variety. This form may be primary or secondary to pneumonia. (*b*) *Pyogenic organisms,* especially the Streptococcus pyogenes and the Staphylococcus pyogenes aureus; (*c*) the *typhoid, colon* and *influenza* bacilli, Friedländer's pneumobacillus and many other organisms have been found.

Pathology.—1. *Macroscopically* a purulent exudate is found between the dura and leptomeninges and in the meshes of the arachnoid. The dura is tense and its inner surface dry. The leptomeninges are reddish, cloudy and are often dotted with punctate hemorrhages. The exudate is clearly purulent, yellow or whitish-green and succulent. The infection spreads rapidly, follows the perivascular lymphatics, as streaks of pus, imbeds the vessels and nerves in its meshes, obliterates the sulci and involves the brain and cord membranes; it is a misapprehension that the epidemic cerebrospinal form involves the spinal meninges and that other suppurative forms do not. The brain is always altered; it is succulent, swollen, springs out as the membranes are cut, is vascular and sometimes locally anæmic from compression by the exudate; its surface is most affected, by such changes as small abscesses, punctate hemorrhages or minute islets of softening or encephalitis. The convexity is most often involved in the metastatic form (convexity meningitis) and the base most frequently in ear disease. There may be primary or secondary sinus thrombosis. The ventricular fluid is often increased and is cloudy, purulent or sometimes serous. The apertures of the ventricles may be occluded (see Hydrocephalus); inflammation may be noted in the velum interpositum, choroid plexus or ependyma. In the spinal membranes the changes are similar and are most marked over the posterior part of the dorsal region. The process is usually diffuse. On incising the membranes the swollen cord bulges out and the same changes are seen as in the brain. The process may extend along the nerve trunks and bloodvessels, even into the orbit.

2. *Microscopically* there are perivascular exudation of red and white cells and fibrin formation. Vascular dilatation and exudation are seen in those cases in which the membranes appear normal to the naked eye. Exudation is most marked over the cortex and near the ventricles, but may also occur deeper in the medullary substance. The ganglionic cells show degeneration. Analogous findings are present in the cord, on its surface or about the central canal. In the optic nerve, exudation, hemorrhage and œdema are seen, possibly

also in other cranial and spinal nerves. Various bacteria are found (*v. s.*), as the pyogenic organisms, the pneumococcus and the meningococcus. Mixed infections are not uncommon. It must be remembered that pathologically (though not bacteriologically), the suppurative and epidemic forms are identical.

Symptoms.—Prodromal phenomena are ambiguous and vary with the causal disease; otitis, pneumonia or pyæmia may long overshadow or totally obscure meningeal manifestations. Complete *latency* is possible, as in a case seen by the author with Dr. Besley; a convalescent pneumonia patient, while dining, felt dizzy and died in a few minutes. The autopsy revealed a massive, purulent cerebrospinal meningitis, which had not produced a single sign or symptom. (*a*) The *onset* may be sudden with chill and fever. (*b*) *Headache* is usually the first and most prominent symptom, and is often associated with vomiting. (*c*) The *sensorium* is disturbed and there is delirium which is succeeded by stupor and finally by coma, in which headache still persists. (*d*) *Convulsions* may occur; they are particularly equivocal in children. (*e*) *Optic neuritis* may develop if the course of the disease is not too precipitate. (*f*) *Rigidity* of the *neck and spine* is soon noted, as well as (*g*) *hyperæsthesia,* hyperalgesia and muscular rigidity. (*h*) *Fever* is usually present, although in very rare instances it may be absent. It is usually higher than in other types, perhaps 104° to 105°, and rather more continuous. Toward the end it may be 107° or may drop to subnormal. (*i*) The pulse is faster and irregular. (*j*) *Irritation* and *paralysis* of the *cerebral nerves* develop (they are considered more fully under the epidemic form) and are manifested by ocular paralysis, nystagmus, strabismus, pupillary inequality and tardy reaction to light, facial paresis, grinding of the teeth or trismus, especially if the inflammation is basal. (*k*) If the process involves the convexity, *cortical irritation* may be manifested by Jacksonian epilepsy and mono- or hemiplegia. (*l*) *Lumbar puncture* reveals purulent or clear fluid and frequently the organisms of suppuration. Increased tension is also observed. (*m*) *Kernig's sign* (see Epidemic Form) is present. (*n*) Other symptoms. Constipation is usually present. The abdomen is often retracted, there may be vomiting, the urine is febrile and frequently contains albumin and peptone. The course is generally rapid; it lasts a few hours to more usually a few days (two to ten) and almost invariably results fatally in coma. The diagnosis, differentiation and treatment are considered under the epidemic type.

SEROUS MENINGITIS.

This is more frequently seen at the bedside than at autopsy. It is often tuberculous. The inflammation is slight, the membranes are glistening, clear or flocculent fluid is seen in the arachnoid meshes and ventricles, which may be the sole seat of the disease, and the pia especially shows exudation. It is often mistaken for œdema of the

brain. The microscope may be necessary to demonstrate inflammation, which frequently is circumscribed.

Symptoms.—In the serous form no definite symptoms can be outlined. The disease certainly exists, although it is relatively infrequent, but with equal certainty it is too frequently diagnosticated. Headache is common; vomiting, rigidity, fever, slow and labile pulse, optic neuritis, hyperæsthesia, spasms and paralyses of the cerebral nerves are much less frequent than in the other forms of meningitis, though they are occasionally observed. The symptoms resemble those of brain tumor. Lumbar puncture in some instances has given absolute relief; the prognosis is most favorable in this type.

CHRONIC LEPTOMENINGITIS.

Etiology.—(*a*) It may result from acute leptomeningitis. (*b*) The leptomeninges thicken after trauma, in atrophic and sclerosing brain diseases, as paretic dementia, hydrocephalus and softening and in alcoholism, gout, nephritis or plumbism. (*c*) Chronic productive inflammation occurs in syphilis, tuberculosis and suppuration.

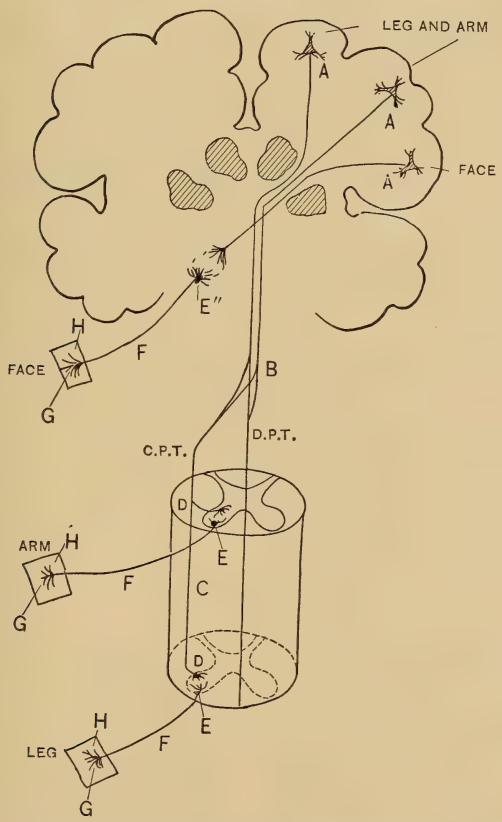
Pathology and Symptoms.—The leptomeninges are diffusely or locally dense, thick, opaque and microscopically show fibrous tissue. In the chronic tuberculous form the membranes are thick, fibrous and caseous; they compress the cortex, especially the psychomotor areas and produce generalized and Jacksonian convulsions, headache, monoplegia, aphasia and mental changes. It may sometimes heal. In the chronic suppurative form the pus loculi are situated in a firm, fibrous tissue. The symptoms, if cortical, are like those of the chronic tuberculous form; if basal, nerve paralysis and optic neuritis are common. In the *chronic simple form* (Gee and Barlow) the process may begin acutely and may persist, especially at the base and in the posterior fossa. Possibly it is a sequel of a localized epidemic type (this theory was rather favored by Koplik in a recent paper) or of hereditary syphilis, trauma or otitis. It is seen especially in children ("simple meningitis of children"); 76 per cent. of cases occur in the first year of life. Rigid neck is the leading symptom, though convulsions and rigidity of the extremities do occur. Paralyses are rare. Fifty per cent. of patients die. The *alcoholic form* is slight, diffuse and cortical; meningitic symptoms are obscured by alcoholic disease of the liver, kidneys, heart or peripheral nerves. Headache is little developed; there is moderate delirium and slight optic neuritis. *Diagnosis* is difficult, and *therapy* is wholly etiological.

DISEASES OF THE SPINAL CORD.

GENERAL ANATOMICAL, PHYSIOLOGICAL AND SYMPTOMATIC CONSIDERATIONS.

The nervous system is made up of numerous similarly constituted units, called *neurones* (Waldeyer). Each neurone comprises (*a*) a *central nerve cell;* (*b*) protoplasmic processes or *dendrites* from the cell which conduct (cellulipital) impulses to the cell; (*c*) an axis cylinder or *axone* arising from the cell and conducting impulses (cellulifugal) from the cell; and (*d*) the terminal ramifications of the axis cylinder, known as end brushes or *arborizations.* The axis

FIG. 62.



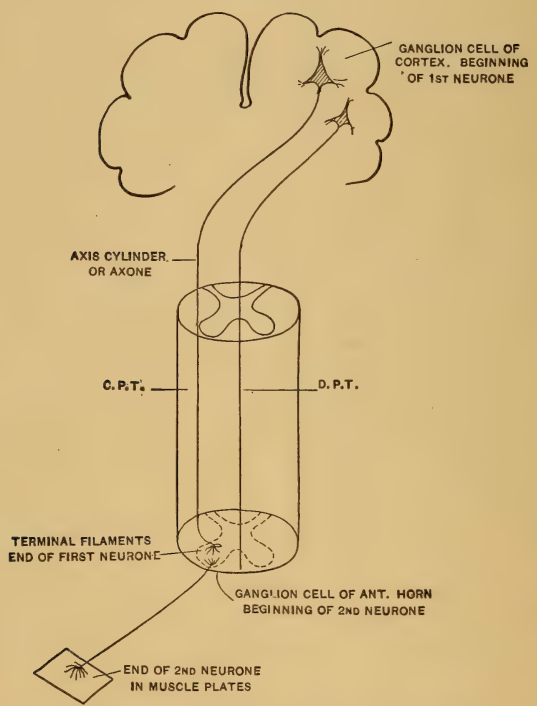
Illustrating the course of the two motor neurones: A, cortical cell of origin of motor tract; B, decussation in medulla; C, course in lateral columns of cord: DD, branches to anterior horns in cord; in second (lower) neurone, E, cells of anterior horns; E″, cells in pons; F, nerve trunks; G, motor end plates; H, muscles.

cylinder often gives off lateral branches, known as collaterals or *paraxones.* The *nutrition of the nerve cell* probably depends upon the integrity of its nucleus, and the nutrition of the cell governs that

of its neurone. Disease of the cell causes degeneration of the entire neurone; division of a process or axis cylinder results in its degeneration below the point of separation from the nutrient cell. Most authorities favor the view that the neurones are wholly independent of each other. At times, and especially in the retina, some degree of anastomosis between the dendrites of different systems exists (Dogiel). The nerve cells are closely grouped in the gray cortex, gray substance of the cord and ganglia of the peripheral nerves. The axis cylinders largely course through their white substance. To comprehend the most important point in spinal diseases, the localization, we must recognize that pathological processes are in general of two varieties: (*a*) those involving certain neurones, or *system diseases,* as tabes, and (*b*) those not involving given tracts or neurones or the *non-system diseases.*

The functions of the cord are (1) conduction of motor impulses to the muscles; (2) conduction of sensory impressions to the brain; (3)

FIG. 63.



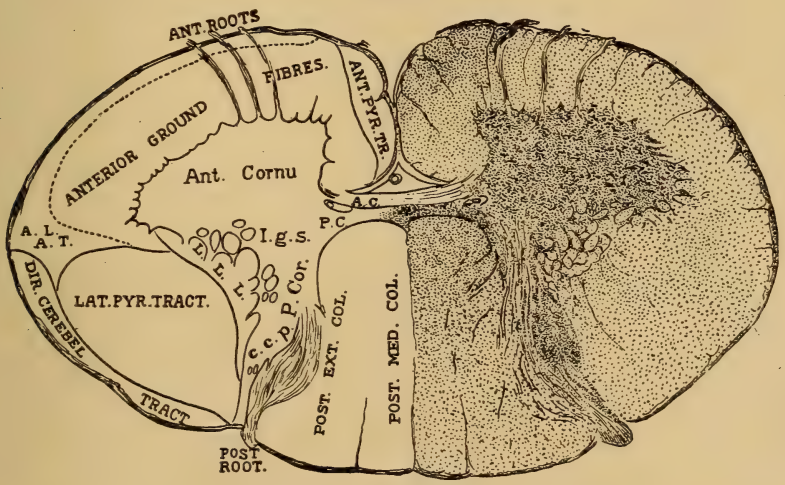
Illustrating the general outlines of the upper and lower neurones and of the direct (DPT), and crossed (CPT), pyramidal tracts.

certain reflexes; (4) centres governing the bladder, rectum, etc., and (5) trophic influences.

1. The *motor tract* is composed of *two sets of neurones or segments.* The *upper segment* has its origin in the cells of the motor cortex (Fig.

62, A), and runs through the corona radiata, internal capsule, crus, pons and in the anterior pyramids of the medulla (whence the name pyramidal tracts), it crosses to the opposite side (Fig. 62, B), running down in the lateral columns of the cord (C) and giving off branches (DD) to the anterior horns, where the tract ends in fine terminal filaments, which surround the motor ganglion cells in the anterior horn. (See Fig. 56.) The *lower segment* begins with the motor cell in the anterior horn (E), the dendrites of which connect with the terminal ramifications of the axis cylinder of the upper segment by contact (Ramón y Cajal) or by concrescence (Held); it extends along its own axis cylinder into the anterior nerve roots and nerve (F), and ends in terminal ramifications (G) (motor end plates) in a muscle, *e. g.,* of the arm or leg, which is on the side opposite to the origin of the cortical motor centre. From the cells of both segments the motor impulse travels downward (cellulifugal conduction). About 75 per cent. of the upper motor neurones cross as above described, are called the *crossed pyramidal tracts* (C. P. T. in Figs. 62 and 63) and lie in the *lateral columns* of the cord. Twenty-five per cent. of the motor fibers (rarely 40 per cent.) do *not cross* in the medulla, but pass down in the *anterior columns* of the same side and are called the anterior or *direct pyramidal tracts* (D. P. T. in Figs. 62 and 63). The anterior direct pyramidal tract usually ends in the mid-dorsal region, but if small may reach only to the cervical enlargement of the cord, or if large may even reach the fourth or fifth sacral nerves. This tract probably crosses to the anterior horn of the opposite side by way of the white portion of the commissure of the

FIG. 64.



Showing the different tracts of the cord. (Gowers.)

cord. There are probably some other motor tracts in the cord. The upper segment is largely (75 per cent.) crossed, but the lower seg-

ment (or neurone) is a direct tract, the cells in the anterior horns supplying muscles of the same side of the body.

The cord, therefore, contains parts of both neurones, the course of which has been ascertained by the *secondary descending degeneration* which follows neurone lesions. Morgagni, Cruveilhier and Rokitansky noted some atrophy in the cord after brain lesions which destroyed the motor paths, but Türck first fully described its occurrence and Bouchard and Barth confirmed his findings. Flechsig, and later Bechterew, added information from embryological studies. After a lesion, as hemorrhage in the internal capsule, the fibers of the upper neurone below the lesion show signs of degeneration. In a few days granule-bearing cells appear and somewhat later the axis cylinders swell, degenerate and stain poorly, especially when studied by the Marchi method. These changes occur because the axones are separated from the nutrient cortical cells. The neuroglia tissue increases and corpora amylacea develop. Degeneration is seen in the opposite lateral pyramidal tract and in the anterior column on the same side as the brain lesion. Sometimes slight degeneration occurs in the lateral pyramidal tract of the same side, which probably has some connection with the anterior uncrossed pyramidal tract of the same side (see Broadbent's theory under CHRONIC SYMPTOMS OF BRAIN HEMORRHAGE). Since the vitality of the neurone fibers decreases as their distance from their trophic cell increases, the degeneration is greatest in the most peripheral part of the axone. The degeneration usually stops at the end ramifications of the axone (see EXCEPTIONS, under BRAIN HEMORRHAGE, PATHOLOGY). (Some fibers lying between the direct pyramidal tract and the direct cerebellar tract degenerate downward; possibly they are part of the lateral pyramidal tract (from Deiter's nucleus in the medulla, Russell.) Fibers lying just anterior to the lateral pyramidal tract also degenerate downward (from the posterior corpora

FIG. 65.

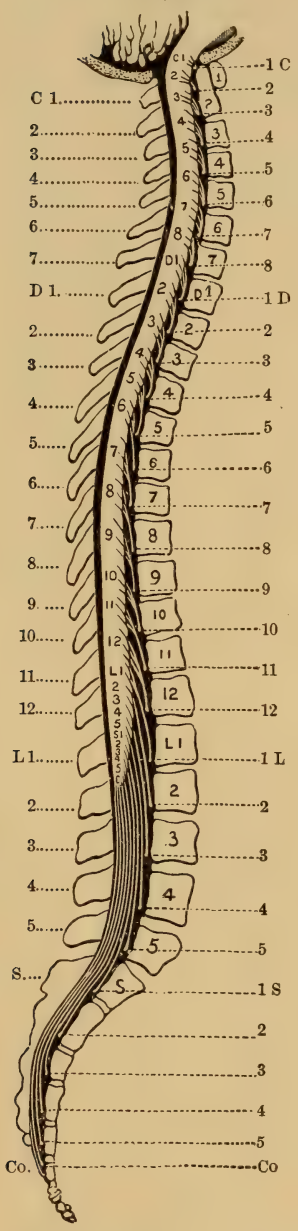
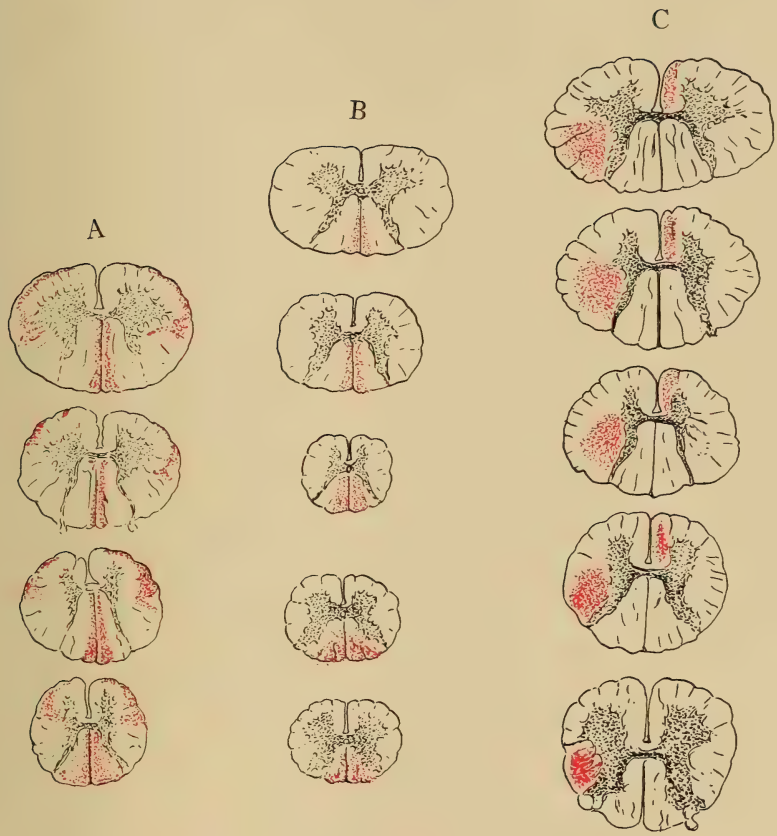


Diagram from Gowers, showing relation of vertebral spines to their bodies and to the nerve-roots.

PLATE XVIII



A. Ascending degeneration in the posteromedian column and anterolateral ascending tracts from lumbar lesion. (After Gowers.)

B. Ascending degeneration after injury to cauda. (After Schultze.)

C. Descending degeneration of pyramidal tracts from right-sided cerebral hemorrhage. (After Mott.)

quadrigemina, Boyce and Sakowitsch). Schultze describes a comma-shaped descending degeneration in the anterior third of Burdach's column. See Plate XVIII.

Degeneration in the Lower Segment.—Disease of the anterior horns, division or disease of the nerve trunk, causes degeneration of the nerve toward the periphery and trophic muscle changes. The degenerated nerve responds neither to the faradic nor to the galvanic current. The muscle does not answer to the faradic current, but to the galvanic, it answers slowly instead of sharply (as is the case with the normal muscle). These differences constitute the *reaction of degeneration* (see NEURITIS).

The two neurones do not correspond in number, because one neurone of the upper segment is connected with several lower neurones, *i. e.,* various cells in the anterior horns at different levels are probably excited by a single pyramidal fiber. For complex movements, as of the hands, there are more pyramidal fibers than for simple movements, as of the intercostal muscles. The anterior spinal root nerves join with fibers from the posterior roots, and passing downward, leave the spinal cord between the vertebræ, thus forming the spinal nerves, whose area of origin in the cord is called a *segment.* The peripheral nerves often arise from several segments, which are sometimes considerably separated; muscles with similar function have similar spinal localization, because *movements rather than muscles are localized in the cord,* as well as in the brain. The appended table gives the localization of the muscles, with special regard to their cells (the beginning of the second neurone), as well as of the skin and reflexes. The cord is shorter than the spinal canal, reaching only to the second lumbar vertebra. The nerve roots descend and therefore their level at the point of exit does not correspond with their level of origin. Fig. 65 shows this relation.

Table giving localization of function in the various spinal segments (from Edinger, Starr, von Leyden, Remak, Thoburn, Ferrier, Yeo, Gowers and Kocher) will be found on page 1003.

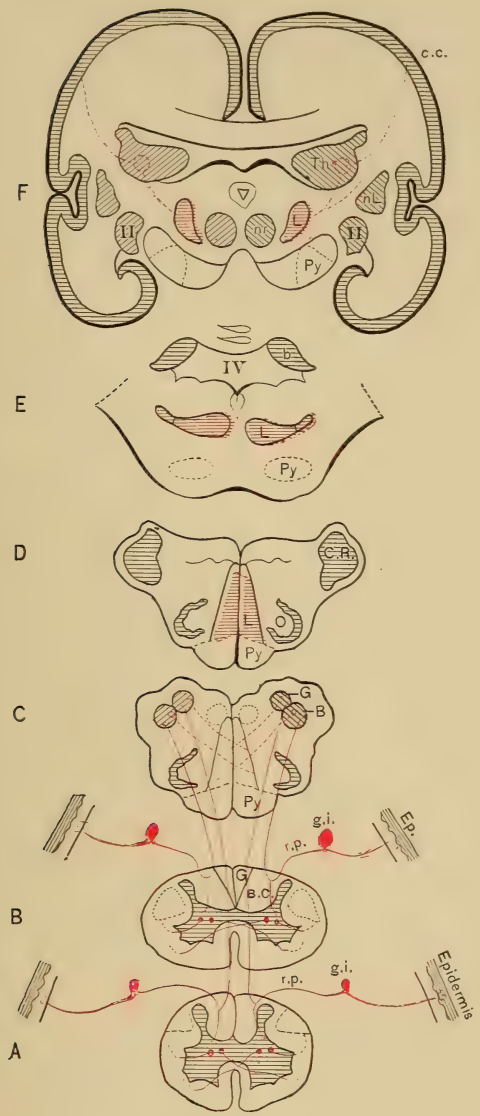
2. *The sensory tract* conducts upward and is more complicated and less clearly understood than the motor tract, because the interpretation of experiments made on animals is frequently confusing. It is composed of three or more neurones. The cells of the *first neurone* are in the intervertebral ganglia, whose cells end in an axis cylinder dividing into a branch to the skin and another to the cord by the posterior root. These ganglia are the homologues of those of the vagus or trigeminus (*q. v.*). Embryologically studied, it would seem less likely that the axis cylinder of the ganglion cell divided than that the branches from the skin (the peripheral sensory nerves) represented the protoplasmic processes or dendrites of the ganglion cells and the branch to the cord represented the axis cylinder. However, all peripheral sensory fibers originate in the ganglia and almost all fibers of the posterior nerve roots come from these ganglia. From the posterior roots the fibers reach the cord, where (*a*) some pass into the posterior

column, (*b*) some into the gray matter and (*c*) others run to the cells of the anterior horns and are clearly part of reflex arcs.

(*a*) The *fibers entering the white substance* (the *posterior columns*) divide into a short descending branch (which probably is concerned in reflex processes, giving off some collaterals and ending in the gray matter) and into a long ascending branch, which is the more important one; they enter the posterior column obliquely and by the entrance of new fibers at higher levels become more centrally located in the column of Goll (posterior median column) in which the lowest fibers (*e. g.,* the sciatic) are most posterior and the highest (*e. g.,* the cervical) are most anterior. The *posterior columns* convey fibers for *muscle sense* (sense of posture and movement association) and possibly also for simple tactile sensation. "Sensations we do not feel" travel in these columns (Gowers). The posterior median (Goll's) column ends in the nucleus gracilis of Goll (or postero-median nucleus) in the medulla. It is uncrossed, *i. e.,* direct. The posterior external (Burdach's) column ends in the nucleus cuneatus of Burdach (or posterior external nucleus) in the medulla. It is likewise uncrossed or direct. The first neurone ends in these nuclei. The *second neurone crosses* above the motor decussation in the medulla (interior arcuate fibers) to the opposite side and unites with fibers that have already crossed in the cord, thus forming the fillet (see below, *b*). This neurone is crossed. The posterior nuclei communicate with the opposite cerebral cortex, with the cerebellum of the same and opposite side and with the external arcuate fibers.

(*b*) The *fibers entering the gray matter of the posterior horns* also divide into ascending and descending branches. Some lie at the zona terminalis, the most posterior part (Lissauer's zone) of the posterior horn. All these fibers end with terminal ramifications about ganglion cells in the gray matter; they form the beginning of a second neurone which crosses in the commissure of the cord and courses upward in the antero-lateral column and anterior ground fibers of the opposite side (just anterior to the lateral pyramidal tracts) and they unite in the medulla with the fibers of the second neurone of (*a*) (*v. s.*). (See Plate XIX.) The fibers entering the posterior horn convey *temperature and pain conduction* and travel between the posterior median column and the central canal. *Tactile sensation* is probably conveyed in the antero-lateral column. Gowers holds that the antero-lateral columns convey most of the upward sensory impulses, since their division causes anæsthesia. Both (*a*) and (*b*) then run in the median lemniscus of the fillet and in part of the longitudinal bundles of the formatio reticularis of the pons, tegmentum of the crus and posterior limb of the internal capsule, beyond which their course is somewhat uncertain. Some fibers run directly to the motor cortex or back of it to the parietal lobes, while others end in the optic thalamus, whence perhaps they are connected by a *third neurone* coursing to the cortex.

PLATE XIX



Scheme of Sensory Conduction. (Strümpell.)

A. Entrance of posterior sensory roots into lumbar cord: *g.i.*, intervertebral ganglion; *r.p.*, posterior root; part of the fibers end in the posterior horns, from whose cells other fibers arise and enter the lateral columns, partly on the same and partly on the opposite side; other fibers from the posterior roots, course upward in the posterior columns and form in—

B. (the cervical cord), Goll's columns (G); B.C., Burdach's columns.

C. Medulla oblongata, G and B are the nuclei wherein Goll's and Burdach's columns end. From these, new fibers arise which decussate and form the fillet (lemniscus, L) in—

D. L, lemniscus; Py, pyramidal tract; O, the olive; *c.r.*, corpus restiforme.

E. Pons; IV, fourth ventricle.

F. L, lemniscus of fillet; *n.r.*, nucleus ruber; Py, pyramidal tract in crus cerebri; N.L., nucleus lenticularis; T.H., optic thalamus (beginning of new neurone to cortex?).

Segment.	Muscles.	Skin: Sensation.	Reflexes.
2–3 Cervical.	Sterno-cl-mastoid, trapezius, scaleni, neck muscles. Diaphragm (3, 4, 5, 6 cerv.).	Neck and occiput.	Sudden inspiration by sharp pressure below the ribs.
4 Cervical.	Lev. ang. scapulæ, rhomboideus, supra- and infraspinatus, deltoid, supinator longus, biceps.	Shoulder (anterior part, Dana).	Widening of pupil from irritation of neck, 4–7 cervical.
5 Cervical.	Supinator brevis, serrat. mag., clavicular portion of pectoralis maj., teres minor.	Radial side of arm (volar and dorsal surfaces) to the insertion of deltoid. Post. surface of shoulder?	Scapular reflex (fifth cerv to first dorsal). Tendon reflexes of this group of muscles.
6 Cervical.	Pronators, coraco-brachialis, brachialis ant., triceps, long extensors of hand and fingers. Lower neck muscles and middle part trapezius, 6, 7, 8, C and 1 D.	Dorsal and volar surfaces of radial side of hand to midline of middle finger, and up to base of hand, narrow strip on volar and dorsal surfaces up to axilla, connecting with above area.	Tendon reflexes of muscles named.
7 Cervical.	Costal portion of pectoralis maj., latissimus dorsi, teres maj. Long flexors of the hand and fingers.	Ulnar part of hand (dorsal and volar) from middle of the fourth to middle of third finger and connecting with it, a moderate strip on volar and dorsal surfaces of arm.	Volar reflex of hand, periosteal reflexes of radius and ulna. Tendon reflexes of muscles named.
8 Cervical.	Extens poll. long. et brev. Small muscles of hand.	Dorsal and volar surfaces of hand to middle of fourth finger, narrow strip on dorsal and volar surfaces of arm up to axilla.	Corresponding tendon reflexes.
1 Dorsal.		Narrow strip on ulnar surface arm and forearm down to base of hand (The upper part, perhaps, belongs to the second dorsal segment.)	Tendon reflexes.
2–12 Dorsal.	Muscles of back (also lowest part of trapezius) and abdomen. Intercostals D, 1–10.	Chest, back, abdomen and upper gluteal region. (Umbilicus, tenth dorsal; ensiform, sixth to seventh dorsal).	Abdominal reflex. Epigastric reflex (according to Dinkler, is at the ninth dorsal, the middle and lower abdominal at the tenth and twelfth, respectively).
1 Lumbar.	Abdominal muscles, quadratus lumborum. Ileopsoas.	Pubic area, anterior aspect of scrotum.	Cremasteric reflex (1–3 lumbar).
2 Lumbar.	Sartorius (or third lumbar), flexors of hip. Quadriceps femoris (extensors of knee), cremaster.	Anterior and inner side of hip, inner side leg to malleolus, inner side of foot, external surface of hip, lumbar regions.	Patellar reflex (2–4 lumbar). Erection (lumbar cord). Uterus (lumbar cord).
3 Lumbar.	Internal rotators of thigh.		
4 Lumbar.	Adductors of hip. Sartorius (?). Abductors of hip. Tibialis anticus. Calf muscles.	Posterior surface of hip, thigh, external surface of leg and foot	Gluteal reflex (4–5 lumbar).
	Flexors of knee (?), gluteals (extensors of hip).	Back of foot.	
5 Lumbar.	External rotators of hip. Flexors of foot (?). Extensors of toes. Peronei.		
1–2 Sacral.	Flexors of foot and toes. Small muscles of foot.	Skin of sacrum, anus, perineum, genitalia.	Plantar reflex; ejaculation (3–4 sacral).
			Achilles tendon reflex.
3–5 Sacral.	Muscles of perineum.		Bladder and rectum reflex.

The brackets to the left concern only the muscles, and indicate the origin of the muscles from several nuclei.

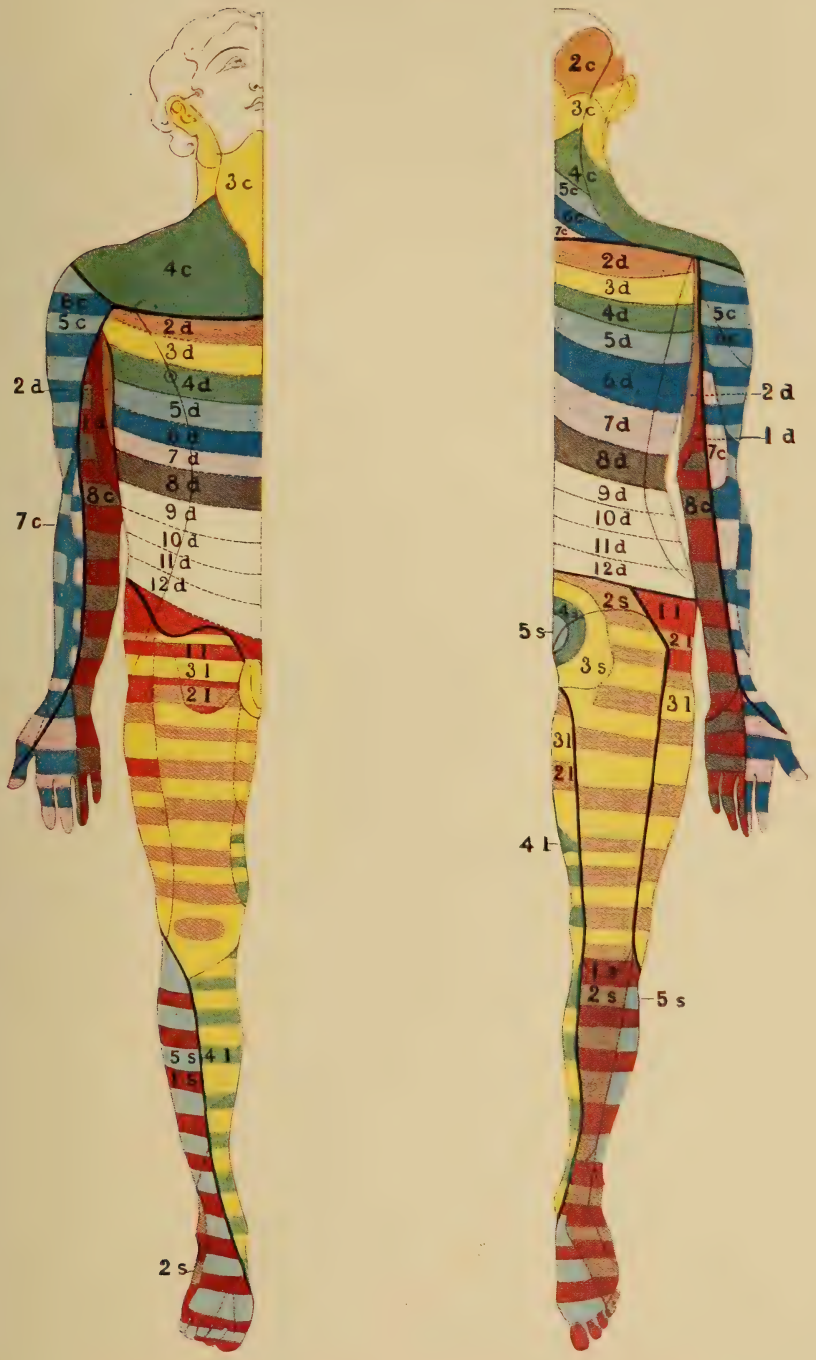
The *sensory tracts degenerate upward* (ascending degeneration). Plate XVIII, Fig. A, shows degeneration in the posterior and antero-lateral columns. Most ascending degenerations stop in the medulla. The *direct cerebellar tract,* which begins at the first lumbar nerve, also degenerates upward. It increases somewhat in size as it rises because of reception of additional fibers, but most of its fibers come from the lower dorsal and first lumbar nerves; some of them come from the gray substance of Clark's columns to which collaterals are sent from the posterior fibers; as to its function Flechsig believes that it conveys muscular impressions from the lower part of the trunk and the legs; its destination is the middle lobe of the cerebellum by way of the restiform body; some fibers then cross to the opposite lobe.

The *spinal sensory nerves* descend lower than do the corresponding motor nerves and skin sensation for a given area is often supplied by two, three or more nerves, whence anæsthesia results only when all of them are diseased (Sherrington). See Plate XX.

Lesions of the *conus medullaris* down to the fourth sacral nerve produce paralysis of the bladder and rectum and simultaneously saddle-shaped anæsthesia on the anus, gluteal region, perineum, genitalia, under surface of the thigh (pudendal and coccygeal plexuses), but the scrotum escapes because the spermatic plexus and external spermatic nerve connect with the lumbar plexus. Motor paralysis is absent because the limbs receive their supply from the lumbar and upper sacral segments. The cremaster reflex is normal. If sexual desire and erection are preserved with loss of vesical and rectal function, conus localization is rather indicated. Pain is not a prominent feature. Sensory dissociation, rapidly occurring trophic changes and rapid advance of the disease argue for conus disease. Lesions of the *cauda equina* (where the roots lie closely together) are usually symmetrical. The onset is insidious and the progression slow; pain is prominent and usually the first symptom; then the leg reflexes are lost, followed considerably later by paralysis, atrophy and anæsthesia (especially in the perineum, ext. genitalia and a narrow zone about the anus). For caudal lesions, speak remission and improvement in the vesical and rectal functions and the slow appearance of muscular wasting, reaction of degeneration and trophic alteration. In its upper part, the above symptoms with paralysis of both legs are noted; paraplegia dolorosa is frequent (see Plate XVIII, Fig. B). A lesion below the third lumbar vertebra is usually limited to the pudendal and sciatic distributions.

3. In the gray matter there are *reflex centres* which are enumerated and localized in the table given above. The reflex arc comprises the afferent sensory fiber with its termination in the gray matter and the motor nucleus with its efferent motor fiber. Collaterals probably connect the centres at different levels. Hutchinson first suggested that the fibers of the *oculopupillary reflex* ran by way of the sympathetic nerve (rami communicantes) to the cord. Irritation of its centre produces dilatation of the pupil (mydriasis), especially in

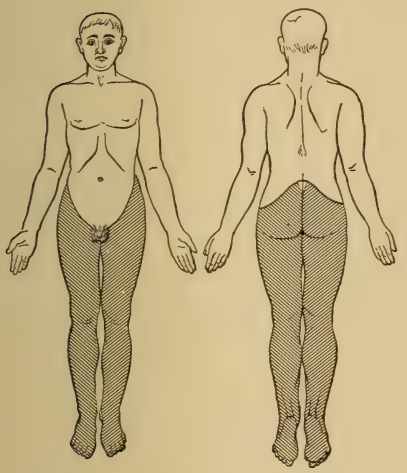
PLATE XX



These figures have been devised by Wichmann in order to show the distribution of the sensory areas corresponding to the segments of the spinal cord. The colors used correspond to those of the normal spectrum, red, orange, yellow, green, blue, indigo, and violet, with brown; respectively—the first, second, third, fourth, etc., segments in each portion of the spinal cord—cervical, dorsal, lumbar, and sacral. The last four segments in the dorsal region are left white. The advantage of the plate is that it shows very clearly the overlapping of the segments that has been demonstrated by Sherrington. The dark heavy black lines on the arms indicate the division between the two sides of innervation—that from the upper portion of the cervical enlargement and that from the lower portion. The heavy black lines of the legs indicate the divisions between the sacral and lumbar areas of innervation. The figures and letters indicate the segments in which the supply has been drawn and have been introduced for the sake of making the diagram more available for ready reference. C—cervical, D—dorsal, L—lumbar, and S—sacral. The horizontal bands of color without dividing lines between them indicate that both segments innervate the areas involved. (Musser.)

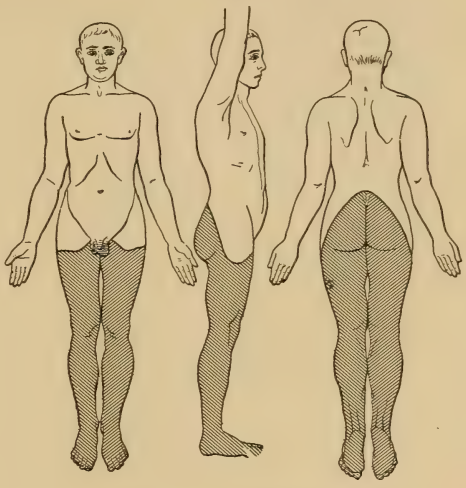
spinal caries. Paralysis from disease of the centre produces myosis, narrowing of the eye fissure, sometimes retraction of the eyeball and less often flattening of the cheek; Madame Klumpe-Déjérine found that this paralysis was produced by section of the eighth cervical and

FIG. 66.



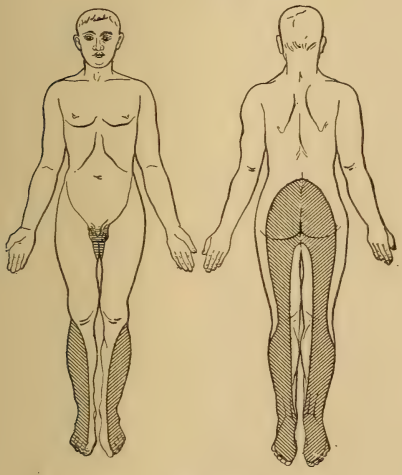
Lesion at the level of the second lumbar segment (Strümpell and Müller, from whom also following figures are borrowed).

FIG. 67.



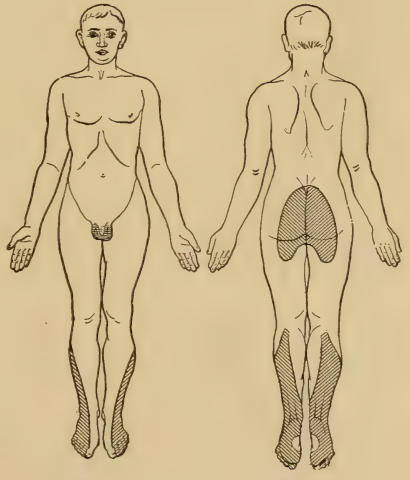
Lesion at third lumbar segment.

FIG. 68.



Lesion at fifth lumbar segment.

FIG. 69.

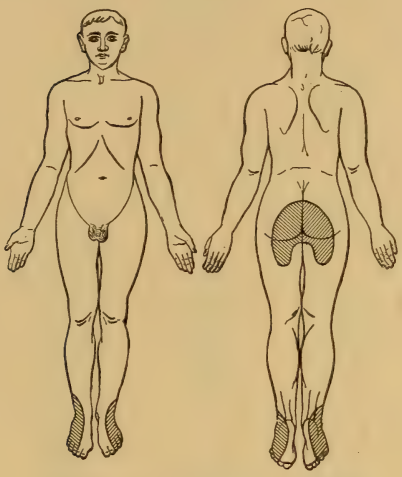


Lesion at first sacral segment.

first dorsal segments; *i. e.,* the localization is the same as for paralysis of the forearm and anæsthesia of the ulnar surfaces of the hand and arm. It occurs more often in disease of the nerve roots than in disease of the cord itself. The *tendon reflexes* will be considered under

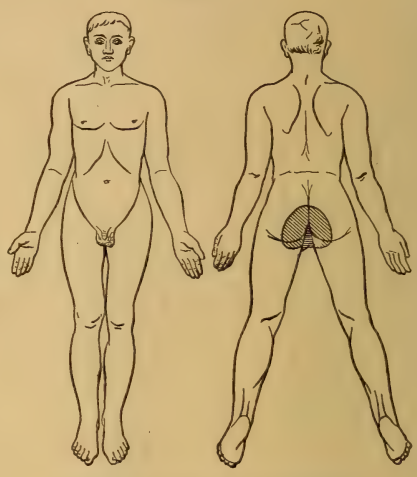
the individual cord diseases. They are inhibited from the brain and probably also from the cord. Evacuation of the *bladder and rectum* is partly a reflex and partly a voluntary process. There are muscular

FIG. 70.



Lesion at second sacral segment.

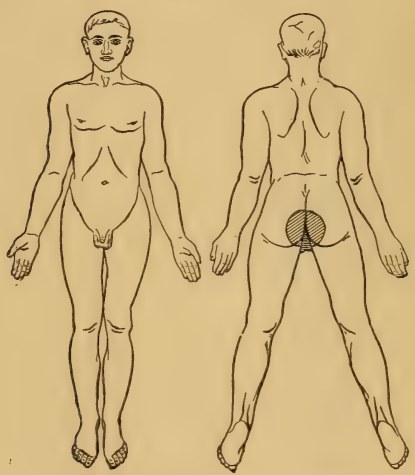
FIG. 71.



Lesion at third sacral segment.

fibers to evacuate the organs and sphincters to retain their contents. The former are stimulated by distention and the latter relax; they are controlled normally by the will. If inhibition fails, the bladder

FIG. 72.



Lesion at fourth sacral segment.

or rectum is evacuated, sometimes with the knowledge of the patient, as in disease of the pyramidal tracts, or at other times unconsciously, as in coma or when the sensory nerves of the parts are diseased.

When the lumbar centre itself is affected there is continuous evacuation. As to the rectum, Gowers discovered that disease of the centre discloses itself by relaxation of the sphincter on digital examination; disease above the centre, by normal tonicity of the sphincter.

From the above considerations, the leading points are given regarding the *level of the lesion* and the *course of the chief systems. Total transverse disease* of the cord produces paralysis of the muscles, at and below that level, and sensory interruption. From these symptoms the upper level of the lesion is determined, but it might be difficult to decide whether the cord were affected through a narrow horizontal level, or whether all the cord, below it, were diseased, unless the reflexes below were noted. Hyperæsthesia usually discloses the level of the lesion. *Lesions of the pyramidal tract* (first neurone) produce motor paralysis at the level of the lesion, increased reflexes and spasticity (hypertonic muscular rigidity) but with no muscular atrophy, reaction of degeneration or sensory disturbance. In lesions of the *posterior columns* the chief alteration is sensory disturbance, especially in the muscle sense, and therefore incoördination or ataxia. The reflex arc may be interrupted. Lesions of the *anterior horns or anterior roots* produce a flaccid paralysis followed by atrophy and the reaction of degeneration if the process is acute. (If it is chronic the atrophy is more marked than the paralysis and the reaction of degeneration is absent or atypical. The trophic cells are not specialized in the horn, but all cells are probably trophic.) The motor nerve (Nasse, 1839) and the muscle degenerate; the reflexes are abolished; the muscle tension (myotatic irritability) is lost, there is vasomotor paralysis (since the fine fibers of the anterior roots are vasomotor) and contractures result (from sensory irritation, Goldscheider). If the lesion is in the anterior horn there may be fibrillary muscular contractions. Irritation of the anterior horns may increase the reflexes and induce contractures. In lesions *of the posterior horns* there are disturbance of the temperature and pain sense and some alteration of the tactile sense; the reflexes may be disturbed. In lesions of the *posterior roots* the reflexes are disturbed and there is anæsthesia and ataxia. Irritation of these roots causes pain, hyperæsthesia in the form of a girdle sensation in the trunk or lancinating pains in the extremities and reflex contractures. The importance of the spinal ganglion as the trophic centre must be remembered (Waller, 1852). *Vasomotor fibers* leave the cord through the anterior roots by the rami communicantes and course down in the lateral columns or in the intermediolateral tract from the centre in the medulla. Most of the vasoconstrictor fibers leave the cord between the third dorsal and second lumbar segments, while the vasodilator fibers are much more diffusely scattered through the cord.

DISEASES OF THE VERTEBRÆ.

I. Fracture of the Vertebræ.—It constitutes but ⅓ of 1 per cent. of all fractures (Gurlt).

Etiology.—Fracture in the dorsal and lumbar regions occurs mostly in the vertebral bodies (66 per cent.), while in the cervical region the processes are often fractured (50 per cent.). Falls, sudden flexion or blows are the most common causes, though in rare cases it is caused by simple muscular exertion. Previous disease, caries and tabes may favor fracture. Corresponding to the obvious angle in the spine, the upper portion usually slips downward and forward, crushing, compressing or even severing the cord, while the abundant epidural plexus of veins ruptures and forms an hæmatoma back of the cord. Gurlt found fracture most often in the fifth and sixth cervical, the last dorsal and first lumbar vertebræ. The cord is the seat of hemorrhagic softening or later of inflammation (myelitis). Marked or permanent injury results in ascending and descending degeneration.

Symptoms.—They may be (*a*) *local, i. e., direct.* Dislocation with deformity (in 68 per cent.) is the most frequent sign; *pain* is frequent; crepitation (in 38 per cent.) is uncommon save when the processes are fractured; its elicitation should not be attempted. (*b*) *General symptoms* include shock and concussion. (*c*) *Spinal symptoms* are more or less complete *paraplegia* (in 71 per cent.), *i. e.,* paralysis of both arms, legs or of parts below the lesion. The reflexes are decreased as a result of the shock; in complete division of the cord the patellars are totally abolished, even when their lumbar centre is not implicated, as in cervical fracture (Bastian, Bruns, Bowlby); Jackson held that the cause was withdrawal of the cerebellar influence; others have suggested that shock or irritation was causative; the abruptness of the lesion is certainly a factor since the patellars are present in slow compression (Gerhardt). The paralysis may be unilateral (see Brown-Séquard's Paralysis under Trauma of the Cord). *Sensation* is *less involved,* but radiating pains into the extremities may occur when the lesion is at the cervical or lumbar enlargement; they are sometimes associated with muscular spasm. Continuous *erection* (priapism) is almost constant, especially in cervical fracture, and ejaculation occurs at the time of the lesion. Paralysis of the bladder and rectum is usual. The bladder can often be emptied by suprapubic pressure. Meteorism may result. Decubitus (bedsores) is common.

Fracture of the first and second cervical vertebræ usually results in sudden death; most cases result from caries (Bergmann). Some subjects die in a few days, while in a few instances recovery is possible. The author has seen two cases with deformity in the spine and pharynx without any cord compression. There are *pain in the neck,* pain on movement, fixation of the head, often palpable deformity in the pharynx, dysphagia, dyspnœa, vomiting and paralysis of the tongue, palate and of all the limbs, usually with preservation of the reflexes. The temperature may reach 111°. The pulse is often slowed (to 40 or 20) by paralysis of the accelerator fibers of the heart which run from the medulla by way of the lateral columns to the fourth to ninth dorsal anterior roots and cardiac plexus. Their

irritation may produce paroxysmal tachycardia. In fracture of the *third and fourth cervical* vertebræ involvement of the phrenic nerve results in respiratory paralysis and early death. In fracture of the *fourth cervical to second dorsal* vertebræ, paraplegia, which increases after the injury from hemorrhage, is the chief symptom. In the arms it is often somewhat asymmetrical. Atrophy may follow injury to the cervical enlargement. Pains, hyperalgesia and tonic or clonic spasms in the arms are frequent. Anæsthesia is usually less marked than the paralysis. The intercostal muscles being paralyzed, the patient breathes with the diaphragm and the accessory respiratory muscles of the neck. Vasomotor manifestations may occur in the pupils, face, trunk or extremities. In fracture of the *third to twelfth dorsal* vertebræ, paraplegia, anæsthesia and often paralysis of the bowel and bladder result. The temperature is often low in lesions of the lower cord. *Lumbar fracture,* except in the first lumbar vertebra, is infrequent. Paralysis is not a frequent result, unless there is marked dislocation when paraplegia, especially in the peronei, and atrophy, pains in the legs along the sciatic and crural nerves and anæsthesia occur (*v. s.,* LESIONS OF THE CONUS AND CAUDA).

Diagnosis.—The diagnosis is based on the trauma, cord symptoms and deformity which, though sometimes absent, serve for differentiation from spinal concussion. In doubtful cases the examination for tender points, fixation of the spine and radiating pains must be made most carefully lest the cord injury be thereby increased.

Course.—Healing is difficult, uncertain and incomplete, because callus formation is rarer than cartilaginous and fibrous union, and dislocation may occur after trauma. Early death frequently results from fractures in the higher vertebræ while in those lower down a later death is likely to occur from cystitis, decubitus or sepsis. The *prognosis* is most favorable in fractures between the third dorsal and second lumbar vertebræ (Gurlt). According to Burrell,—from whose collation of 244 cases (1905) most of the above statistics are taken,—the average mortality is 65 per cent.; the mortality is 86 per cent. in cervical, 76 per cent. in upper dorsal, 56 per cent. in lower dorsal and 50 per cent. in lumbar fractures.

Treatment.—Surgical interference is not indicated in fresh cases, unless a comminuted process may press on the cord. If paralysis persists from pressure of a process on the cord, especially in the lumbar region, operation is indicated.

II. Dislocation of the Vertebræ.—Dislocation is traumatic, or spontaneous from previous vertebral disease. Traumatic luxation is sudden, injures the cord to a greater extent and results from falls or blows on the head or neck. Fracture very frequently accompanies dislocation. Luxation from previous vertebral disease is gradual, sometimes sudden and injures the cord less, since it is often incomplete (subluxation). It occurs most often in the cervical region, especially in the first and second or fifth and sixth vertebræ. It is

infrequent elsewhere in the spine. The vertebræ sometimes are torn apart without actual displacement.

Symptoms.—The head is often bent forward toward the sternum or shoulder. Movement is suspended or limited. Tenderness and pain are elicited and the local deformity may be felt or seen. The cord is injured by direct compression, contusion, hemorrhage and later by myelitis, with much the same symptoms as in fracture. Sometimes no spinal symptoms are observed. Sudden death is very common in the higher dislocations from pressure on the medulla.

Treatment.—Reduction is indicated even when the cord is not injured and may relieve all symptoms.

III. Caries of the Vertebræ.—Etiology and Pathology.—Pott's disease (Pott, 1779), spondylarthrocace, is the most common vertebral disease involving the cord. Caries is almost always tuberculous. It is most common after the fourth year and in adolescence, but may be seen at any age. Tuberculosis in other organs is frequent in Pott's disease; it usually begins "spontaneously," in one or several vertebral bodies or more rarely in the articulations. It may follow trauma. The spongy portion of the vertebral body is first involved. Granulation tissue is the earliest finding; later caseation occurs with extension to contiguous parts, the connective tissue, ligaments, processes and cartilage. The collapse of the altered body usually produces an angle and prominence in the spine called *Pott's boss,* and the diseased body slips backward, sometimes compressing the cord. By extension the nerve roots may be involved; they are bathed in pus, sometimes show connective tissue induration and, in cases of long standing, atrophy. The outer surface of the dura is very frequently inflamed (pachymeningitis externa); collections of thick, caseous pus gather in the cellular tissue between the dura and the posterior surface of the body of the vertebra; the cord is more injured by caseous or purulent pachymeningitis than by deformity of the spine. It was once thought that the cord was not only compressed but also inflamed, whence the name, compression myelitis.

Compression paralysis (so-called compression myelitis) may result from (*a*) vertebral disease, tuberculosis, cancer or exostoses; (*b*) disease of the spinal cord and its membranes, as tumors, pachymeningitis hypertrophica, syringomyelia or hydromyelia; or (*c*) compression by aneurysm or echinococcus. Pathologists differ as to the actual changes in the compressed cord. Myelitis may develop in some cases, but "compression myelitis" is probably nearly always softening (myelomalacia). In some cases with marked paralysis, the postmortem examination shows no changes in the cord, whence it is certain that pressure may interrupt nerve impulses without structural cord disease. In other cases all gradations of alteration are found, from a few granule-bearing cells or swollen axis cylinders to marked disintegration, corpora amylacea, neuroglia overgrowth and induration.

Symptoms.—Symptoms usually develop in the following order:

1. SPINAL COLUMN.—Angular deformity (*kyphosis, gibbus,* Pott's boss) is common but not invariable; it is most marked in upper dorsal disease and is at times accentuated by periosteal induration. Frequently there is only lateral deviation (scoliosis); ordinary lateral curvature of the spine never affects the cord or its nerve roots. *Tenderness* over or beside the affected vertebræ and *pain* are usually localized and develop early. The application of heat, which is not felt over the normal vertebræ, may cause pain over the diseased focus. Pain and tenderness are very common in the neuroses, but the hyperalgesia is largely cutaneous, is usually diffuse and varies more than in caries. *Rigidity of the spine* is most conspicuous in disease of the cervical or lumbar region, where movement is normally most free; it is usually an effort to spare pain. *Gravitation abscesses* are frequent and follow along the planes of least resistance in the tissues. Dysphagia from retropharyngeal abscess or low compression of the œsophagus may cause errors in diagnosis.

2. NERVE ROOTS.—*Eccentric pain* is usually the earliest symptom; it radiates into the trunk or extremities and indicates involvement by pachymeningitis of the posterior nerve roots. It is much less acute than in cancer and is especially elicited by movement. Sensory are more common than motor root symptoms and irritative symptoms, as hyperæsthesia, are more common than anæsthesia. Root symptoms are most pronounced in lumbar and sacral disease and least pronounced in dorsal disease.

3. SPINAL CORD.—Compression (*a*) interrupts function at its level, disturbs reflexes, impairs rectal and vesical evacuation and (*b*) interrupts upward and downward cordal conduction, producing spastic paresis or paralysis, anæsthesia affecting all varieties of sensation and increase of skin and tendon reflexes, whereas root disease does not interrupt conduction. Cord symptoms develop slowly; Oppenheim placed the average time at one year; weakness of the legs is followed by paresis, this in time by paraplegia, which may develop suddenly. Anæsthesia and other sensory manifestations are less common than are motor symptoms, because the sensory tracts are more resistant or more protected, since the greatest compression is exerted on the outer lateral surface of the cord. Ataxia is obscured by the paralysis. Marked anæsthesia, paralysis and atrophy are referred to the cord. The reflexes are increased (unless their arc is destroyed at the level of the lesion) since cerebral inhibition is interrupted.

Special Localization.—1. DORSAL CARIES.—This is the most frequent type. Gradual deformity is usual, with formation of a sharp or obtuse angle. Acute deformity, such as might result from a fall or lifting a heavy burden, is much less common. Paraplegia develops in varying degrees of intensity with increased muscle tonus (spasticity), increased leg reflexes, or even clonus, or paralysis of the bladder and rectum; it sometimes develops with contractures but rarely with marked sensory changes, other than intercostal root pains

in the chest wall, which sometimes produce precordial oppression or a girdle sensation. Herpes zoster may develop. The level of the lesion may be marked by a zone of hyperæsthesia or hyperalgesia. Trophic changes in the skin are common, as glossiness, smoothness or dryness. There is no essential atrophy save from disuse, nor is there any reaction of degeneration, unless the anterior horns are diseased. In cases of partial paralysis the tendon of the tibialis anticus becomes prominent (tibialis phenomenon). The tendon reflexes may be increased even when the paralysis is apparently flaccid. They are almost always increased below the lesion. Exaggeration of the patellars is an early symptom, often antedating the paralysis. The skin reflexes below the lesion are often increased, but may be decreased in cases of severe compression, though they are seldom absent. The Babinsky reflex is often found (see BRAIN HEMORRHAGE).

2. CERVICAL AND UPPER DORSAL CARIES.—This form ranks next in frequency; radiating pains reach the shoulder, arm and hand on one or both sides. Anæsthesia is more common, along the ulnar nerve, over the sternum and scapula, because the posterior roots are more often affected. Paralysis may result from compression of the cord or from disease of the anterior roots, and involves the small muscles of the hand, forearm and triceps. The fore-finger and thumb often cannot be apposed (the ape hand) and from atrophy of the interossei, the *main en griffe* results (*v. i.*). The forearms are flexed and pronated, while the arms are adducted (Thoburn). The tendon reflexes usually persist. In other cases marked atrophy occurs instead of paralysis and the reaction of degeneration sometimes develops. Spastic para-paresis or -plegia in the legs from compression of the pyramidal tracts usually accompanies the diplegia (paraplegia) brachialis, which, however, sometimes occurs alone, when the anterior horns or roots are diseased. Myosis is common from injury to the oculospinal centre (Rollet, 1864) and there may be narrowing of the eye fissure and less often retraction of the eyeball, which is more frequently unilateral than bilateral, or differs in degree on the two sides. Flattening and reddening of the cheek are less common. These are less symptoms of disease of the cord than of the anterior roots. The reflexes of the legs are increased; total compression of the cord may destroy the tendon reflexes by secondary changes in the lumbar cord; when the paralysis is flaccid the urine is retained at first and later there is incontinence. There is usually constipation.

3. DISEASE OF THE FIRST TWO CERVICAL VERTEBRÆ.—(Rust's Disease.)—In disease of the atlanto-occipital articulation, nodding is impossible, while the involvement of the odontoid-atlas articulation prevents turning of the head. In body-movements the patient supports the head with the hands (Rust). The head may be held as it is in torticollis, and induration under the occiput is common. Pains radiate to the eye, ear and occiput. The typical paralysis in disease of the two upper cervical vertebræ is brachial paraplegia, which usually occurs without atrophy. Spasms in the face, anæsthesia in

the neck and upper chest, aphonia and other symptoms indicating involvement of the medulla oblongata may be present. Charcot observed a slow pulse with attacks of syncope. The spinal accessory supply to the head and palate and the hypoglossus supply may be affected. The course is usually more rapid (one to two years) than in carcinoma; recovery is possible (v. Bergmann). Degenerated axis cylinders do not regenerate. Gravitation abscess may reach the neck or pharynx. Sudden death is not unusual, because dislocation readily occurs, since the disease is in the articulations rather than in the vertebral bodies.

4. Lower Dorsal and Upper Lumbar Caries.—This type produces atrophic flaccid paraplegia, destruction of the lumbar reflexes, urinary incontinence, anæsthesia of the legs, pain in the abdomen or possibly in the groins or anterior thighs. When it occurs in the lower *lumbar region* there are flaccid atrophic paralysis, absence of the patellars and spastic paralysis below the knees, with clonus of the Achilles tendon (see Conus and Cauda Localization).

Diagnosis.—Cases may be classed under three groups (*a*) those with spine symptoms, (*b*) those with spine and spinal cord symptoms and (*c*) those with cord symptoms only. Cases are almost unmistakable in which the paralysis follows signs of bone disease in early life. Repeated examinations usually determine the nature of doubtful cases in which bone-symptoms develop after compression of the cord or nerve roots and in which there are early increase of the skin or tendon reflexes, root-symptoms, which are usually bilateral, as cervical or intercostal neuralgia, lumbago, anæsthesia and muscular atrophy or paralysis. The Röntgen rays may aid in diagnosis. Gravitation abscesses, fever and tuberculosis elsewhere are significant. In persons of advanced years, cancer, tumor within the spinal canal and aneurysm must be considered (see Cancer of the Spine).

Prognosis.—The prognosis is unfavorable, since about 50 per cent. of cases die. Relative recovery is possible, especially in children, but even then the process may break out anew. A rapidly fatal course is rare. Absolute paralysis may disappear after months or even years. The prognosis is best in disease of the lower dorsal region and is least favorable in disease of the atlas and axis. Gowers remarks that recovery or improvement is more frequent than in any other disease with symptoms of equal severity. There is hope of recovery in most cases and yet there are few in which it is sure. Cystitis is common and leads to pyelonephritis; bed-sores are frequent and death results from sepsis, mixed infection, amyloid disease or lung tuberculosis.

Treatment.—(*a*) *Medical* treatment includes administration of iron, arsenic and cod-liver oil; fresh air and a full diet are equally important. The skin and bladder should receive special attention. Electricity is contra-indicated. (*b*) *Orthopedic* and *x*-ray *treatment* produce the most successful results. Rest in bed on the back should be enforced for months. Extension, fixation by braces of the neck and

back, and for lighter cases or those involving the lower spine, the plaster cast, are described in works on orthopedic surgery. (*c*) *Surgical intervention* is indicated only when other treatment has been given thorough trial. The results are not encouraging and disease of the affected bodies is difficult of access. The dura should never be incised. According to Chipault, the *indications* are (i) disease restricted to the arches, (ii) cold abscess, (iii) flaccid paralysis, since Babinsky noted that this occurs in moderately severe dorsal disease, and (iv) healing of the bone focus and replacing of granulation tissue by connective tissue, as in the cases first operated on (Macewen. 1888). The *contra-indications* are (i) multiple vertebral disease, (ii) lung tuberculosis and (iii) disease of the cord itself. Victor Horsley advises orthopedic treatment for two months in children and then, in case of failure, laminectomy. In adolescents and in those of advanced years, he considers that laminectomy is indicated practically at once.

IV. Carcinoma of the Spine.—Carcinoma is almost always secondary to carcinoma of the mamma and less often of the digestive tract, uterus, prostate or thyroid; it generally involves the bodies, usually of several vertebræ.

Symptoms.—Angular deformity is less common than in caries. A tumor may be felt. Bruns considers a sinking in of the vertebræ or softening of the spinal column as rather characteristic. Rigidity is common but is less conspicuous than in caries. Tenderness is common. The *spinal symptoms* closely resemble those of compression by caries, and are present in 60 per cent. of cases (Schlesinger). Compression of the posterior roots is most common and produces neuralgic pain, at first paroxysmal and later constant, lancinating and incoercible; it is located along the spinal column, while in caries pain is felt over the spine or in the side of the chest (Gull). Closely associated are hyperalgesia and anæsthesia, tenderness on pressure, painful reflex muscular spasm and herpes zoster. Compression of the cord is less common and occurs late; if paralysis develops it is usually rapid and painful. The veins of the lower extremities thrombose frequently and fatal pulmonary embolism may result (Schlesinger).

Diagnosis.—Diagnosis depends upon (*a*) recognition of a primary cancer (which is sometimes latent or overshadowed by the secondary deposit); (*b*) local signs (tumor, spinal irregularity, arcuate kyphosis); (*c*) cachexia; (*d*) advanced age; (*e*) pain, usually bilateral and increased by movement; paraplegia dolorosa (Cruveilhier) is much more common and incomparably more intense in cancer than in caries, from infiltration of the sensory roots; anæsthesia with pain (anæsthesia dolorosa) is also common; and (*f*) rapid onset and course. *Sarcoma* may be primary or secondary. Cachexia is less common; it is more often seen in youth than in elderly persons and is more frequently multiple. *Vertebral syphilis* is very uncommon, as gummata in the bone or periosteum, but responds to antisyphilitic

treatment. Syphilis simulates caries rather than carcinoma. Actinomycosis rarely causes compression of the cord; it is recognized by its presence in other organs and by the sulphur bodies in the sinuses and aspirated fluid. *Arthritis deformans* (*q. v.*) is recognized by exostoses, tenderness, ankylosis, in rare cases by crepitus, by alterations in other joints and by root-symptoms caused by narrowing of the foramina (radiating pains, vasomotor symptoms and atrophic paralysis). *Aneurysmal erosion* of the spine, cervical pachymeningitis, hydatids, exostoses, tumors within the spinal canal, glands compressing the intercostal nerves and tabetic pains in the trunk must be considered. The *prognosis* is absolutely unfavorable and the *treatment* is directed chiefly against the pain.

DISEASES OF THE SPINAL MENINGES.

Few diseases of the vertebræ and cord do not to some degree affect the meninges but, with few exceptions, the meningeal symptoms have little independent clinical interest.

I. Hyperæmia.—At autopsy the blood gravitates to the dependent parts and congests or discolors the meninges. Stasis, tetanus, eclampsia, chorea, carbon monoxide and strychnine congest the membranes.

II. Hemorrhage.—(*a*) Between the vertebræ and dura, hemorrhage is frequent from rupture of the subdural plexus of veins. It surrounds the nerve roots and is most severe over the posterior cord; it results mostly from trauma and to a lesser extent from tetanus, strychnine poisoning, vertebral disease, stasis or aneurysmal rupture. (*b*) In the arachnoid sac hemorrhage (hæmatorrhachis) is equally frequent and results from the above named causes and from asphyxia, extraction by the foot during labor, hemorrhagic diseases or leptomeningitis. The entire spinal canal may be filled with blood from brain hemorrhage, especially at the base. (*c*) In the pia, hemorrhage rarely attains pathological or clinical interest.

Symptoms.—Small hemorrhages cause no symptoms and meningeal apoplexy may be overshadowed by brain or traumatic cord symptoms. Pain, localized in the back or sometimes generalized, rigidity, radiating pains from compression of the posterior roots, hyperæsthesia or hyperalgesia, painful reflex muscular spasms, motor weakness (and paraplegia), anæsthesia, sphincter disturbance and abolition of the tendon reflexes are the common symptoms. They develop suddenly, affect the lower more than the upper cord, increase for a day or so, produce inflammatory reaction for two or three days and usually subside in from four to eight weeks. Physical signs vary with the location; for instance, brachial paraplegia is present in cervical, and involvement of the legs in lumbar or dorsal hemorrhage.

Diagnosis and Treatment.—In *diagnosis* the most important symptom is irritation, while in hemorrhage of the cord (hæmatomyelia) paralysis prevails and pain is less frequent. In non-traumatic cases, exact diagnosis is impossible. The *treatment* is symptomatic. Absolute rest in the lateral decubitus or on the face should be enjoined.

III. Pachymeningitis.—(*a*) This may be *external* (peripachymeningitis), as described under caries. It may follow penetrating bedsores, deep cellulitis, psoas or retropharyngeal abscess. Its symptoms are those of compression by vertebral caries (*q. v.*). Root symptoms and fever are frequent. The prognosis is unfavorable and the treatment is etiological, expectant or surgical. (*b*) *Internal pachymeningitis hemorrhagica.* Its causes are those of the cerebral form, as paretic dementia, alcoholism, trauma and sometimes tuberculosis. (*c*) Charcot (1871) and Joffroy described a form involving chiefly the cervical region (*pachymeningitis cervicalis hypertrophica*), in which the inner surface of the dura is thickened by annular concentric fibrous deposits. The dura adheres to the vertebræ and leptomeninges and compresses the nerve roots, which indurate. Several cervical segments are involved. The process is most marked on the dorsal aspect of the cord, which is flattened anteroposteriorly and shows peripheral induration by extension of the process to it through the pial vessels (meningomyelitis); this causes the frequent ascending and descending degeneration. Cold, syphilis, alcoholism and trauma are probable causes.

Symptoms.—Charcot described *three stages;* (*a*) the *neuralgic stage,* which corresponds to involvement of the posterior nerve roots, in which pains radiate from the nape of the neck into the ulnar and median nerve supply. They are continuous, with paroxysmal exacerbations, with localization at times in the arm joints, with rigidity as in caries, paræsthesia in the arms and some motor weakness. Hyperæsthesia is common. (*b*) The *second stage* is *atrophic paralysis* of the arms in which the process involves the anterior nerve roots. The pains are replaced by atrophy and paralysis in the ulnar and median nerve areas, while the radial area is usually unaffected; this produces flexor paralysis in the forearms and hands, and the claw hand (*main en griffe*) results from contracture; the wrist is overextended, the first phalanges are extended, the second and third are flexed; ulnar and median anæsthesia and the reaction of degeneration are found. The shoulder and elbow muscles may be involved. Muscular atrophy is not constant and other deviations from Charcot's complex are at times noted, as absence of contracture or extension to the lower cord. (*d*) In the *third stage* the legs show a *spastic paralysis* without atrophy and the bladder and rectal functions and sometimes the sensory conduction are disturbed from secondary changes in the cord.

Diagnosis.—The process is usually localized with ease as a disease in the cervical region. Marked pain may also occur in tumors, cervical spondylitis, syphilitic meningomyelitis and at times in syringomyelia. Von Leyden and others maintain that diagnosis is usually impossible and that the process is only a cervical localization of a meningomyelitis. Amyotrophic lateral sclerosis (*q. v.*) and progressive spinal muscular atrophy have no such severe pains and syringomyelia (*q. v.*) is characterized by dissociated anæsthesia, as

well as by muscular atrophy, painless felons and trophic changes in the joints. Tumors (*q. v.*) are difficult to differentiate. Neuritis causes symptoms limited to the limbs; the nerve trunks are tender.

Prognosis.—This is usually unfavorable and chronic progression, cystitis, etc., usually develop. Instances of recovery are recorded.

Treatment.—Warm baths, counterirritation, sedatives, narcotics, mercury, iodides and care of the skin and bladder are indicated.

IV. Spinal Leptomeningitis.—This closely corresponds to the description given under cerebral meningitis, as to the spinal, pathological and clinical findings. It may occur (*a*) in the acute form, from trauma, extension from pleuritis, bed-sores or other adjacent infections, from general infections, and from epidemic, suppurative or tuberculous meningitis; or it may occur (*b*) in the chronic form, from tabes, trauma, myelitis, syphilis (*q. v.*) or alcoholism. The symptoms are pain in the back, rigidity, hyperæsthesia and radiating pains; the course is afebrile.

V. Syphilitic Affections.—(See SYPHILIS.)

TUMORS OF THE SPINAL CORD AND ITS MEMBRANES.

These are rare; about 200 cases are reported. Brain tumors are six times as frequent (Schlesinger).

I. Tumors of the Membranes.—These may arise from the dura or leptomeninges. In a collection of 20 *extradural tumors* Horsley found five sarcomata, four lipomata, four tubercles, three echinococcus cysts and one each of myxoma, fibrochondroma, carcinoma and fibrosarcoma. Cysticercus often causes no symptoms. These growths cause more erosion of the vertebræ than compression of the cord. *Intradural tumors:* Horsley collected 38, of which there were twelve myxomata, seven sarcomata (endotheliomata), seven fibromata, four psammomata, four tubercles, two parasitic and two syphilitic tumors. Lipomata, echinococci, cysticerci, neuromata and angiomata are rarer. Though usually single, multiple occurrence may be noted in sarcomata, parasitic tumors and neuromata. This group compresses the cord or its nerve roots. The cord is altered as in caries; there are œdema, flattening, discoloration, peripheral hardening and central softening and secondary ascending and descending degenerations. The membranes thicken and the nerve roots flatten and atrophy.

Symptoms.—Extradural growths produce more vertebral than cord symptoms. The first symptoms are usually those of compression of the *nerve roots, e. g.,* radiating pains, first on one side, then on both, which run into the arms, trunk or legs, according to their localization, and advance from one root to another; hyperæsthesia; paræsthesia; anæsthesia, which is less common since it implies involvement of at least three roots (Sherrington's law); atrophic paralysis with vasomotor symptoms; rigidity; and spontaneous muscular contractions. Local tenderness is rare. The symptoms recall those of carcinoma of the spine aside from its vertebral signs. Later, evidences of *com-*

pression of the cord appear, as paresis, perhaps of one side first (Brown-Séquard's type, *v. i.*) or paraparesis with atrophy if the anterior horn is involved at that level, with increased reflexes (unless the arc is broken), with spasticity and contractures; and with sensory changes. In this type, as in carcinoma of the vertebræ, the paraplegia dolorosa of Cruveilhier is common, as is anæsthesia dolorosa (the "eccentric projection" or reference of pain to the anæsthetic areas). Finally, there is total paraplegia, anæsthesia and bladder and rectal paralysis.

II. Tumors in the Cord Substance.—These are six times less common than tumors in the membranes; they include *glioma* (which is most frequent in the cervical and upper dorsal regions), sometimes gumma, sarcoma (in the pia), tubercle (in the gray substance or posterior horn) and cysticercus. Most of them are circumscribed, though glioma tends to vertical diffusion. Unlike meningeal tumors, they are rarely metastatic.

Symptoms.—In intramedullary growths, root symptoms are absent, especially in gliomata and, since there is gradual compression of the cord, the clinical picture may resemble chronic myelitis and syringomyelia. The cord symptoms are disturbed conduction; spastic paraplegia; marked increase of reflexes (unless the arc is broken or absolute cord compression develops); bilateral symptoms, or if unilateral, they soon reach the other side; extensive muscular atrophy, if the cervical or lumbar enlargement is diseased, often then with the reaction of degeneration; and sensory changes involving all varieties of sensation or certain varieties only, as pain or temperature (dissociated or partial anæsthesia). Some writers, as Bruns, maintain that the lesion and symptoms are at first unilateral. Then the Brown-Séquard complex is found (*q. v.*). As the tumor grows, total paraplegia and anæsthesia may, in rare cases, develop below the level of the lesion.

Localization.—Accurate determination of the localization is most important but rarely possible until the cord itself is compressed. The most common error is in locating the tumor too low. Atrophic paralysis, in the cervical or lumbar region, is the most certain aid, and sensory changes are less reliable. The actual lesion is one or two (three or four) segments higher than the anæsthesia (Sherrington's law) because anæsthesia results only from involvement of at least three roots. The hyperæsthesia usually represents the level of the lesion and local spinal tenderness, though rare, is very important. Finally, the spinal segment involved must be considered in terms of the corresponding vertebræ (*v. s.*, page 1000). (*a*) When the tumor is in the *upper cervical* cord there are pains in the cervical plexus and perhaps anæsthesia; at first there may be a spinal hemiplegia differing from cerebral hemiplegia in that the face and hypoglossus escape and the anæsthesia is contralateral to the paralysis; then there is spastic paralysis of all four extremities, which rarely lasts long, since affection of the phrenic nerve causes early death. (*b*) In tumor of the *cervical enlargement* there are atrophy, flaccid paralysis and pain

in one arm, spastic paresis in the leg of the same side, anæsthesia of the entire opposite half of the body and opposite limbs; and then paralysis of both arms and legs with anæsthesia. (*c*) Localization in the *dorsal cord* often produces unilateral signs, as paresis of one limb with its half of the trunk, and anæsthesia of the corresponding parts of the other side; spastic paraplegia of legs and abdomen, retention of urine and later incontinence; and then girdle pains. (*d*) In tumors of the *lumbar enlargement* there are unilateral pains in the lumbar plexus and atrophic paralysis in the ileopsoas, quadriceps and adductors; there is total anæsthesia when the cord is involved in the region of the lumbar plexus; later partial anæsthesia (affecting the sense of temperature and pain) develops in the region of the sacral plexus; still later there is complete paralysis and anæsthesia in the region of the lumbar plexus, with atrophy, absence of the patellar reflexes, with the reaction of degeneration, while the legs and feet are spastic, and with clonus of the Achilles tendon. (*e*) If the localization is in the *sacral cord* there is atrophic paralysis of the leg and foot, of the posterior muscles of the thigh, of the gluteal and perineal muscles, anæsthesia, total paralysis of the bladder and rectum, impotence, loss of the Achilles tendon reflex (while the patellars may persist), early decubitus and cystitis. (*f*) If the tumor is in the *cauda,* the symptoms are usually bilateral from the beginning; paraplegia is rarely as symmetrical as it is in cord lesions; there is the reaction of degeneration; pain is violent, since many sensory fibers are in close contact; Minor described bilateral and even unilateral sciatica as an early symptom. The sacral plexus is chiefly or first involved, while growths of the lumbar enlargement affect both the sacral and lumbar plexuses.

Diagnosis.—The diagnosis of meningeal tumor, always difficult, is based on (*a*) local pain and rigidity, which are especially marked in the extradural type; (*b*) hyperalgesia; (*c*) radiating pains, *i. e.,* root symptoms which later, by gradual transition, give way to (*d*) cord symptoms, which are often unilateral at first and then bilateral, as interrupted sensory and motor conduction and increase of the reflexes. *Carcinoma* of the vertebræ produces more pain on movement than other tumors. In *caries* there is less pain, local vertebral changes eventually appear, and the course is usually more rapid. In *myelitis* the arms are less often paralyzed; radiating pains are much rarer than in tumor and a girdle sensation is more common. The local pain sometimes suggests *aneurysm,* lung disease or peripheral neuralgias, the tender points of which are always absent. Root symptoms, early interruption of cordal conduction and absence of trophic disturbance in the bones and of dissociated anæsthesia argue for tumor and against *syringomyelia.* A diagnosis of *tuberculosis* or syphilis is fortified by its presence in other organs. Age; in youth, tubercle; at puberty, sarcoma and glioma; in the third and fourth decades, glioma, tubercle or sarcoma; and in advanced years gummata are most frequent. *Diffuse sarcomatosis* is usually cerebrospinal, rarely

invades the cord substance, as do gummata and tubercles, affects the posterior cordal surface most frequently (the lateral surface little and the anterior very rarely) and generally in the lumbar and lower dorsal regions; it occurs largely in the young and its course is rapid. Abscess of the cord is most rare. *Tabes* in one remarkable case was simulated by multiple tumors of the posterior roots. *Tumors of the cord* are more difficult of differentiation, for root symptoms are often lacking.

Course and Prognosis.—Slow development, progression either steady or "by starts," sometimes remissions and eventual compression of the roots or cord are usual, with death from cystitis or decubitus. Sudden paralysis is less common. The average duration is one to three years, but a longer course is possible (even fifteen years).

Treatment.—1. Therapeutics.—Narcotics must be employed to relieve pain, but should be used sparingly in the early stages, because if given freely, their effect may fail later if the course is protracted. Syphilis is always a possible factor; absence of evidence or history of the disease counts for little, and the patient should receive the benefit of antisyphilitic treatment.

2. Surgical Treatment.—Gowers in 1887 localized a case of fibro-myxoma which Horsley removed successfully; this was the first case on record. Tumors of the cord are less favorable than those of the meninges. Intramedullary tumors may only crowd the nerve paths, but their removal is often attended by laceration or acute myelitis, which may result in damage equal to that of the tumor. Accurate localization is most important. Urban's osteoplastic resection is valuable; it consists of folding back the cut muscles and bone that they may be replaced and the strength of the spinal column may not be impaired. Death may result from shock, escape of cerebrospinal fluid and less frequently, with modern methods, from infection. Starr records 16 complete recoveries. Harte in 1905 collected 92 operations; the operative mortality was 47 per cent.; of the balance, 59 per cent. were cured, 34 per cent. improved and 7 per cent. not improved.

CIRCULATORY DISTURBANCES; HEMORRHAGE; TRAUMA OF THE CORD.

I. Hyperæmia of the Spinal Cord.—Postmortem hypostatic congestion is common, especially in the dorsal aspect of the cord. The white matter is usually pale. The cord may be passively congested, as in cardiac insufficiency. Vascular variability was formerly considered causal of spinal irritation which, however, is hysterical and neurasthenic. No definite clinical picture can be constructed.

II. Anæmia of the Spinal Cord.—Paralyses referred to anæmia of the cord are mostly due to neurosis, neuritis, hemorrhages in the cord or nerve sheaths, and, in pernicious anæmia, to degeneration. Paralysis following profuse hemorrhage from the stomach or uterus may be

properly referred to cord anæmia. Stenson (1667) demonstrated experimentally that compression of the aorta produced paraplegia due to anæmia of the cord, the motor cells of which appear to be especially susceptible to ischæmia. Compression of the aorta after ligature of both subclavian arteries has identical effects.

III. Embolism and Thrombosis (Myelomalacia) of the Spinal Cord.—Embolism of the spinal arteries is extremely rare, but has been observed in endocarditis (v. Leyden, Weiss). No separate clinical symptoms can be recognized, although experimentally Money observed choreiform movements. Thrombosis is very rare. Venous thrombosis is secondary to other lesions of the cord. Arterial thrombosis is rather more common. It has been found in syphilis, multiple sclerosis, senility and perhaps bears some relation to acute processes in the cord, as disseminated myelitis. It is often impossible to decide whether an area of softening is ischæmic (myelomalacia) or inflammatory (myelitis).

IV. Hemorrhage in the Cord (Hæmatomyelia).—Etiology and Pathology.—Most cases of this rare lesion occur in men between 20 and 40 years of age. Trauma causes 90 per cent. of cases of hæmatomyelia (Oppenheim). (*a*) Capillary hemorrhages rarely cause symptoms; they occur in foci of softening, inflammation, stasis, intoxications, infections or blood diseases. (*b*) The larger hemorrhagic focus may result from trauma, muscular effort, caisson-disease, etc.; it compresses and destroys the cord substance; it rarely exceeds the size of a hazelnut (round or oval form) but may extend through the entire length of the cord (tubal form of Levier), following exactly the less resistant gray matter, while the firmer white columns (pyramidal tracts) usually resist its lateral diffusion; if it invades the white matter, hemorrhage usually occurs in the posterior columns; it occurs mostly in the cervical cord; all large non-traumatic hemorrhages occur in the gray matter. Effused blood undergoes the same fate as in other parts; it turns brown, then yellow-red, and ultimately deposits dark pigment and hæmatoidin crystals. The nervous tissue is disorganized by large hemorrhages, which form in it a cavity. Eventually a pigmented apoplectic scar or cyst results.

Symptoms.—The symptoms vary with the different forms. (*a*) In the accessory form there is bleeding into a tumor, cavity, softening or inflammation (Charcot, Hayem, Köster); punctate hemorrhage may accompany other affections, as hemorrhagic purpura, stasis or convulsive disorders, and few or no symptoms develop. (*b*) The traumatic form is less important in spinal fracture than concussion (see Trauma); it may develop during dystocia or fœtal extraction. (*c*) The *spontaneous* form is rarer than it is in the brain, because the cord is firmer, subject to lower blood pressure, and most rarely the seat of miliary aneurysm; it results from sudden exertion in man (and more often in the horse), from repeated coitus or rarely from arteriosclerosis.

The onset is sudden, without prodromes or loss of consciousness.

Previous symptoms may suggest a hemorrhage into an inflammatory focus. Local symptoms must vary with the structures involved and their level; they embrace paraplegia, monoplegia, the Brown-Séquard syndrome, paralysis with or without atrophy or spasticity, anæsthesia either total or partial, as might be anticipated from its location in the gray substance and posterior horns; pain at the level of the lesion, perhaps from involvement of the posterior horn; less often eccentric pain, girdle sensation and muscular rigidity. The reflexes and sphincters vary with the level. In general the reflexes are increased in small and abolished in large hemorrhages. The symptoms advance rapidly and may suffer an accession from secondary reaction or myelitis. Early death is rare. Late death from cystitis or decubitus is more common. Recovery is rare compared with recovery in cerebral apoplexy; it occurs most often in the punctate type; it is rarely absolute and some disturbance in motility or sensation usually remains.

Diagnosis.—The diagnosis is usually only probable; it is based on the sudden onset of paresis and anæsthesia and their rapid advance. For differentiation from *hæmatorrhachis,* see page 1015; in the latter, meningeal irritation is more common, as marked and radiating pains, rigid spine and muscles, hyperæsthesia of the skin and muscles; paralysis is absent or incomplete; and recovery is more complete. From *acute myelitis,* differentiation is often uncertain and frequently of only academic interest, since each affection may complicate the other and the prognosis and treatment of each are similar. In myelitis and poliomyelitis elevation of temperature is distinctive.

V. Caisson or Divers' Paralysis.—According to Pol and Watelle (1854), this occurs in workers who are subject to pressure of at least 2, and usually 3 or 4, atmospheres. Symptoms appear when the individual comes suddenly into the open air. They include transient pain in the ears, sometimes deafness and vertigo, headache and pains in the limbs, joints and epigastrium; in 35 per cent. there is paraparesis or paraplegia, sometimes with anæsthesia, and usually with urinary retention; much less often there is hemiplegia or involvement of both arms; epistaxis, mental symptoms; loss of consciousness and symptoms of brain-pressure, as vomiting or slowed pulse. Men of heavy build, alcoholics and "new hands" seem to be predisposed; hard exercise after coming into the air produces unhappy symptoms, which are not severe or are absent if the interval of work does not exceed one hour.

Pathology and Pathogenesis.—The pathology and pathogenesis are undetermined. Hoppe-Seyler (1885) and Bert found that lessened atmospheric pressure liberated oxygen, nitrogen or carbon dioxide in the blood and tissues, which ruptured the vessels and tissues and promoted punctate hemorrhage. In the first autopsy v. Leyden found clots in the cord substance, particularly in the posterior and lateral columns, surrounded by round cells (reactive disseminated myelitis); most changes were in the less compact dorsal cord; secondary degeneration is found and changes in the gray substance from hemorrhage;

Catsaris actually saw gas bubbles in the blood. Under moderate pressure the gas is given off to the lungs, while in extreme instances it is given off to the tissues. The symptoms apparently result less from the gas bubbles (air embolism) accumulating in the blood under high pressure than from its sudden reduction and the reactive apoplectic myelitis. Andrew H. Smith holds that the lesion is cordal congestion and stasis.

Prognosis.—A few subjects die at once; others die from cystitis, as in myelitis; about 50 per cent. recover completely and 50 per cent. partially, with spastic paresis. The mortality ranges from 2.7 to 15.8 per cent.

Treatment.—The return to normal atmospheric pressure should be made gradually; recompression is successfully employed. Smith recommends ergot.

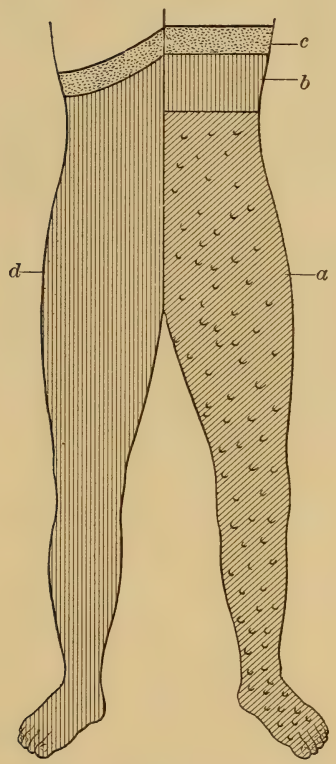
VI. Trauma of the Cord.—Compression by caries, tumor and carcinoma have been considered; other surgical lesions can only be touched on here. *Crushing* of the cord may be associated with hemorrhage, myelitis and secondary degenerations. *Total division* of the cord may result from fracture, gunshot or stab wounds; it is extremely rare; early death is usual; its symptoms are absolute paraplegia, anæsthesia, bladder paralysis and abolition of the patellar reflexes. *Gunshot* wounds usually result in fracture, crushing and sometimes actual division; as a rule the prognosis is unfavorable. *Stab* wounds may produce bilateral symptoms or Brown-Séquard paralysis; 20 per cent. recover; 60 per cent. improve and 20 per cent. of cases die (Wagner and Stolper). *Restitution of function* in lesser injuries results not from regeneration but from compensation by other paths of conduction. Laminectomy is indicated only in crushing injury of the lower half of the spine, because cervical cases almost never recover (Hahn).

VII. Brown-Séquard's Paralysis.—This was described by Séquard and Türck simultaneously in 1850. According to Brown-Séquard's original proposition, *on the side of section* (cervical cord) there occur (*a*) paralysis of voluntary motion, muscle sensibility and vasomotor tonus; (*b*) hyperæsthesia of trunk and limbs, to touch, pain, heat and cold; (*c*) vasomotor paralysis of face and eyes (higher temperature, narrow pupils and moderate contracture of certain facial muscles). *On the contralateral* side there is anæsthesia of all varieties of sensation, except muscle sensibility.

Symptoms.—Exact hemisection of the cervical cord produces a *spinal hemiplegia* on the side of the section, but in injuries and experiments the trauma is rarely wholly complete or strictly unilateral. (In some cases decussation of the uncrossed pyramidal tracts may occur in the cord lower down than the usual crossing point in the medulla (Flechsig) and spinal hemiplegia with contralateral monoplegia may result from unilateral lesion. Déjérine and Thomas speak of a third "homolateral pyramidal" tract, which may permit restitution of motor function.) The most characteristic clinical type is the mid-

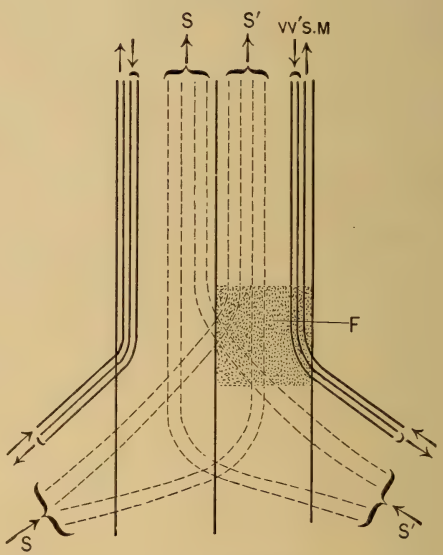
dorsal lesion, but in cervical section or in pathological foci, paresis of the arm with paralysis of the leg is more usual, because the cervical motor tracts are less compact than they are in the lower cord; *i. e.*, paralysis is more frequently limited to a group of muscles than to an entire upper extremity. "Conversely, paralysis of the leg may be incomplete, while that of the arm is complete, owing to the escape of fibers for the leg which cross lower down in the cord" (Gowers). The paralysis often decreases with surprising rapidity, and if the anterior cells are intact it gradually becomes simple weakness. The

FIG. 73.



Brown-Séquard's paralysis from a left-sided focus. A, vaso-motor and motor paralysis; B and D, cutaneous anæsthesia; C, hyperæsthetic zone. (Erb.)

FIG. 74.



(Combined from Brown-Séquard.)—F, Focus in left side of cord. V, Pyramidal fibers having crossed in medulla. V′, Vasomotor fibers, not crossing in cord. *Sm*, Fibers for muscle sense, not crossing in cord. SS S′ S′, other sensory fibers crossing in the cord. The focus explains the classical signs and also how few of the crossed sensory fibers to the left side are involved (merely a zone of anæsthesia), while all sensory fibers to the right side are severed (hemianæsthesia).

motor are restored more readily than the sensory functions. There is an inactivity atrophy of the muscles without abolition of faradic irritability and without reaction of degeneration; the paralysis may be spastic or flaccid. Respiration is rarely affected and imperfect movement of the chest has never occurred. Involvement of the thoracic and abdominal muscles usually indicates a bilateral lesion. Swelling and œdema in the paralyzed members have been observed,

and also swelling with pain in all the joints on the paralyzed side. The *deep reflexes* are exaggerated on the side of lesion; they are abolished in a sudden total transverse lesion of the lower cervical or upper dorsal cord. The skin reflexes are abolished or decreased on the side of paralysis and on the opposite anæsthetic side they may be normal, absent or increased.

Hyperæsthesia (Fodera, 1823) occurs on the paralyzed side and also as a zone above the anæsthetic area and near the level of the lesion. If the lesion be cervical or lumbar the anæsthetic or hyperæsthetic areas above are not zonular but irregular. Hyperæsthesia has been referred to the wound itself or to paralysis of inhibition. Hyperæsthesia or hyperalgesia concerns tactile, pain and heat and cold sensations and their absence infers incomplete section (Kocher). It usually disappears rapidly. The fibers, the section of which results in hemianæsthesia, lie in the lateral tracts.

The *zonular anæsthesia* on the side of the injury and of motor paralysis is explained since the lesion involves not only the sensory fibers (which are ready to decussate to the other side and produce the crossed hemianæsthesia), but also sensory fibers from the contralateral side, which have just decussated to the side of section. Abolition of *muscle sensation* on the side of the lesion is in accord with the uncrossed course of the muscle sense fibers in Burdach's column, entering Goll's column higher up. Muscle sense is preserved on the side opposite to the lesion. Vasomotor paralysis occurs on the side of section, as the vasomotor fibers course in the anterolateral columns. "*Sensation is affected on the opposite side,* but not quite up to the level of the lesion, because the decussation of the sensory tract is not immediate but occurs somewhat above the entrance of the nerve" (Gowers). Complete crossed hemianæsthesia occurs in 60 per cent. and partial anæsthesia (analgesia and thermoanæsthesia) in 40 per cent. of cases.

Prognosis.—In clinical and experimental cases almost complete restitution to normal is possible; it is not due to reëstablishment of conduction in the severed tracts, in which ascending and descending degeneration occurs, but rather to assumption of function by the uninjured side of the cord (Kocher). The prognosis and *treatment* of Brown-Séquard's paralysis vary with the etiology of this symptomatic syndrome, which may be syphilis, tumor, sclerosis, myelitis, tuberculosis, trauma, hæmatomyelia, fracture or luxation.

VIII. Concussion of the Cord.—Gussenbauer referred the severe symptoms, as paralysis, to hemorrhage, which is produced in turn by movement of the cerebrospinal fluid. Long ago Ollivier explained the symptoms as being due to molecular alteration in the nervous tissue; this was confirmed by cases of experimental concussion which resulted in degeneration of the cells and axis cylinders (Schmaus and Bikeles). Myelitis and chronic degeneration may follow concussion. Erichsen's "railway spine" is one of the traumatic neuroses. Obersteiner considers that long continued moderate

jarring, as in the case of tramway drivers, may injure the cord. *Symptoms* develop at once or some time later, as paraparesis, paraplegia, retention of urine, and anæsthesia less often than paræsthesia. Some cases recover after months to a year or two. In others the symptoms are permanent; in still others death, usually early, occurs. In *diagnosis,* fracture, hæmatorrhachis, hæmatomyelia and traumatic neurosis should be considered. Simulation, in order to obtain damages for accidents, may be difficult to exclude, but some railway surgeons and medical experts in corporation employ err in discounting the importance of concussion and traumatic neurosis (*q. v.*). Paraplegia and retention of urine are indicative of structural alteration of the cord. *Treatment* consists of rest, local application of cold and avoidance of neurotic tendencies.

INFLAMMATION OF THE CORD.

Myelitis is inflammation of the cord. It was described by Oliver and Abercrombie a century ago; the term has been made to cover many affections not properly inflammatory, as compression paralysis. Oppenheim and Marie disputed the opinion of Gowers, v. Leyden and Erb that myelitis is a frequent affection.

I. Acute Myelitis.—Etiology.—There are two prominent causes, infections and intoxications. The affection occurs largely in males between ten and forty years of age.

1. Acute Infections.—Experimental myelitis has been produced by the B. pyocyaneus, B. diphtheriæ, B. coli, B. typhosus, strepto- and staphylococci, etc. French writers have contributed most to this subject. It has followed epidemic meningitis, variola, measles, rheumatism and other infections, caused (*a*) by the germs themselves or secondary infection (focal myelitis resulting) or (*b*) by their toxins (disseminated myelitis resulting).

2. Intoxications.—These include poisoning by alcohol, ergot and lead, and perhaps auto-intoxications, as in cancer or severe anæmia.

All other causes are doubtful; trauma or compression by caries or cancer is often considered of etiological importance but probably causes softening or hemorrhage, to which infection may of course be added; exposure to cold and dampness most likely reduces the physiological resistance, thus indirectly favoring localization of bacteria; syphilis (*q. v.*) may affect the meninges and cord (meningomyelitis), but the so-called syphilitic myelitis is usually ischæmic softening from vessel changes. The etiological importance of sexual excesses and emotional shocks is at present sharply questioned.

Pathology.—On macroscopic examination we find redness and swelling; on cross section, blurring of the cord tissues and decreased consistency, even to fluidity; "red softening" (hemorrhagic myelitis) and in older cases "yellow softening" from fatty change, or "white softening" and cavity formation occur. Myelitis can usually be seen to best advantage after hardening in Müller's solution; the nor-

mal tissue then appears green and the diseased, yellow. In the ultimate stages, atrophy, cyst formation, induration and very rarely abscesses are found. *Microscopically* there are, *in fresh cases,* degenerated swollen axis cylinders, the disintegrating medullary sheaths from which myelin drops may form; granules and granule corpuscles (leukocytes or vessel endothelial cells); extravasated red and white cells; turgid vessels the nuclei of which proliferate; choking of the perivascular lymph spaces with cells; degeneration, blurring and vacuole formation of the nerve cells, the processes of which shrink and the nuclei vanish; granular amorphous deposits of coagulated albumin in and about the cells; in some cases the parenchymatous far exceeds the interstitial alteration and often parenchymatous myelitis were better called parenchymatous degeneration. The gray matter (poliomyelitis) is involved much more often than the white (leukomyelitis). In *older cases* there are corpora amylacea, empty spaces corresponding to destroyed nerve fibers; large "spider" cells with many processes (Deiter's cells); and there is substitution of destroyed tissue by interstitial proliferation. In rare instances bacteria are found, as streptococci or colon or typhoid bacilli, but probably the toxins are more important than the organisms. The microscope differentiates between the ischæmic inflammation and postmortem softening. The *degree and the extent* of inflammation vary greatly; *transverse myelitis* may destroy an entire cross section; or *disseminated* foci may involve at random various structures at different levels. *Meningomyelitis* (perimyelitis, annular or cortical myelitis) chiefly affects the periphery of the cord, whence concentric invasion along the septa may split the cord structures; this is frequent in the syphilitic form. *Poliomyelitis* (inflammation of the gray substance) is a type of myelitis, but, involving only the anterior horns, is usually classified by itself. Degeneration and inflammation of the motor and sensory *roots* may follow myelitis in the cervical or lumbar enlargement; secondary degenerations are common; the inflammation may ascend or descend the cord, but usually affects only one or two segments.

Symptoms.—The symptoms vary with the extent, form and localization. The localized form, limited to a small segment, is called *transverse,* while the diffused form is known as *disseminated myelitis.*

Acute transverse myelitis is the prototype. The cord symptoms are usually first in time and always first in importance and constitutional symptoms, as fever, chills, malaise and in children convulsions sometimes may precede but usually attend the cord symptoms. The symptoms resulting from *involvement of the cord* are (*a*) *paralysis,* which is usually early, rapid but not apoplectic in onset and is prominent, paraplegic, progressive, flaccid and rather complete. The flexors are usually weaker than the extensors. The toes alone may be moved; twitching of the paretic muscles is usually associated with pain; clonic muscular spasms are sometimes present. The paralysis is often preceded for a short time by sensory phenomena, as numbness, or by moderate darting pains in the limbs, joints or back. The par-

alyzed limbs show a transient rise of temperature, followed by somewhat subnormal temperature. The limbs are often dry and may not sweat even after injection of pilocarpin. (*b*) *Sensation* is frequently involved. The initial tingling pain has been considered. Anæsthesia may be complete to the level of the lesion, but more often incomplete (hypæsthesia) and rarely partial, according to the cordal tissues involved. It is higher anteriorly than posteriorly; a zone of hyperæsthesia or "girdle pain" marks the upper level; it is caused by early irritation or late cicatrization. Severe pain suggests vertebral or root disease rather than myelitis; gastric crises like those of tabes, dysæsthesia (Charcot), *i. e.,* diffuse vibrating sensations over the entire limb from local stimulation, as by a needle, and ataxia (usually obscured by the paraplegia) are much less common. (*c*) At the onset all *reflexes* may be lost, but they are usually increased below the focus, because of lessening of the cerebral inhibition traveling in the lateral pyramidal tracts, the skin reflexes being increased early and the muscle reflexes later; involvement of their centres causes permanent loss of the reflexes; for instance, they are lost in the arms and increased in the legs in cervical myelitis; lost in the trunk and increased in the legs in dorsal myelitis; lost in the legs in lumbar myelitis. (*g*) The *sphincters* are affected almost constantly and early, perhaps even before the onset of paraplegia; initial difficult urination (ischuria) or urinary retention, possibly with sphincter spasm and later with incontinence, is usual; when the bladder is full it may dribble from relaxation of the sphincter, known as ischuria paradoxa or retention with incontinence; the patient may or may not be conscious of the retention, depending on whether the afferent sensory fibers are involved or not; in lumbar myelitis incontinence prevails from the beginning. The urine is at first alkaline and later ammoniacal on the advent of cystitis. The sphincter ani acts involuntarily, *i. e.,* is spasmodically contracted; the paresis of the bowel and abdomen are factors in constipation; in lumbar myelitis the sphincter is paralyzed. (*e*) *Trophic changes.* Muscular atrophy and the reaction of degeneration are caused by disease in the anterior horns and the muscles also waste from inactivity. The skin is usually dry, sometimes glossy or vesiculated. The joints are sometimes swollen and the limbs œdematous. Bed-sores are frequent over the sacrum, trochanters, heels, between or even over the knees, from the bed covers; they result from neglect or in lumbar myelitis from trophic disturbance (disease in the gray matter or posterior horns). The same factors are said to cause cystitis, ulceration in the bladder or urethra and the tendency to cellulitis.

Course and Prognosis.—The onset may take place in a few hours (the apoplectic form), or in several days to a week (the subacute form). *Four stages* are described, which, however, blend: (*a*) the more or less acute *initial stage;* (*b*) the *advancing degeneration;* (*c*) the *restitution;* and (*d*) the *stationary* (residual or chronic) stage. Complete paraplegia and anæsthesia may persist, but more

often sensation returns after some weeks, while movement returns far more slowly and incompletely, usually with some residual spastic paraplegia. Contractures may develop, especially in the adductors

CORD SYMPTOMS GROUPED ACCORDING TO LOCALIZATION.
(Amplified from Morton Prince.)

In order of frequency.	*Dorsal myelitis* most frequent.	Then *Lumbar myelitis.*	Lastly, *Cervical myelitis.*
Paralysis.	Trunk, back, intercostal and abdominal muscles (favoring mucus stagnation in lungs). Arms normal. Paraplegia, first flaccid then spastic (lateral columns involved).	Trunk and arms normal. Paraplegia flaccid throughout (ant. horns involved).	Neck muscles, diaphragm, arms, trunk and legs paralyzed. Typically, atrophic flaccid paralysis of arms (ant. horns involved), with flaccid, later spastic paralysis of legs (lat. columns involved); however, paraplegia brachialis with paralysis of legs is very rare. In rare lesions high in the cervical cord, the neck muscles atrophy and both arms and legs show simple, then spastic paralysis, without wasting.
Atrophy.	In trunk; difficult to elicit. In legs; only from inaction.	None in arms or trunk. Atrophy in legs (ant horns involved).	Atrophy of neck muscles if high lesion (rare). Usually of arms only (ant. horns involved at level of lesion). In legs only from inaction.
Reaction of degeneration.	Present in trunk muscles; absent in legs (or only quantitative reduction).	Present in legs or in mild cases quantitative reduction.	Present in arms (rarely in neck).
Sensation.	Girdle pain and hyperæsthesia, between ensiform and navel. Anæsthesia corresponding with motor paralysis.	Pain in loins or legs; hyperæsthesia in loins. Anæsthesia of legs.	Pain and hyperæsthesia in arm nerves (with highest localization); anæsthesia in arms (lower localization); and the trunk and legs.
Reflexes.	Superficial reflexes, initial loss, rapid return and increase. Deep reflexes, initial loss, slow return and increase.	Lost. Lost.	Superficial reflexes; initial loss, rapid return and increase. Deep reflexes; initial loss, slow return and increase.
Sphincters.	Bladder, initial retention, later intermittent overflow or incontinence. Cystitis common.	Incontinence from beginning (sphincter paralysis).	Same as in dorsal myelitis.
	Bowels, usually spasm of sphincter ani; constipation.	Incontinence (sphincter paralysis) disguised, perhaps, by constipation.	Same as in dorsal myelitis.
Trophic changes, etc.	Decubitus from neglect, etc.	Decubitus, cystitis, etc., from neglect and trophic alteration.	As in dorsal lesions. (Occasionally very high temperature.)
Pupils.	Klumpke's paralysis of pupil (if in upper dorsal).	Absent.	Paralysis of pupil (lowest cervical region). Optic neuritis in isolated cases.
Priapism.	Often present and painful; sometimes ejaculation at onset.	None. Impotence.	Priapism often present.
Mode of death.	Cystitis, decubitus.	Decubitus, cystitis.	Medulla symptoms, vomiting, slow pulse; usually by involvement of phrenic nerve with early death.

and the knee flexors, in which case the prognosis is particularly unfavorable, for profound disease is indicated. Contractures, muscular contractions and increased reflexes are caused by secondary degenera-

tion. The immediate outlook in myelitis is always uncertain and ultimate recovery is usually incomplete; it depends (*a*) on the etiology, the prognosis being best in acute infections; (*b*) on the intensity of inflammation; (*c*) on its transverse or vertical dimensions, and (*d*) on the rapidity or tardiness of signs of improvement. Complete recovery in mild circumscribed forms is barely possible, since indirect symptoms, due to collateral œdema may disappear. Death results from extension to the medulla (causing slow pulse and vomiting), phrenic nerve phenomena, and sepsis following cystitis, pyelitis or pyelonephritis. A form of recurrent myelitis is said to exist (*myelite à rechutes*).

Diagnosis.—The early onset of structural cord symptoms, as retention of urine, followed by paraplegia and anæsthesia, is especially significant after acute infections. The upper level of the myelitis corresponds to that of the paralysis or anæsthesia. The lower level may be determined by the condition of the reflex centres. Application of a weak faradic current shows to what extent the gray matter is involved. *Anterior poliomyelitis* (*q. v.*) does not involve sensation and but rarely extends back of the anterior horns. Compression by *caries* or cancer usually causes local vertebral changes and root symptoms precede cord symptoms. The so-called *syphilitic myelitis* is ischæmic myelomalacia; it begins acutely without pain and is rarely relieved by antisyphilitic remedies. *Hemorrhage* in the cord produces most sudden symptoms and acute spinal pain without prodromes. For differentiation from *Landry's paralysis,* see page 1038, and from *multiple neuritis,* see page 1080. *Meningeal hemorrhage* and meningitis produce unmistakable root symptoms. *Abscess of the cord* is rare, for few more than a dozen cases are reported; its symptoms are those of myelitis plus meningeal symptoms. *Hysterical paraplegia* is characterized by other stigmata of hysteria, motiveless variability of the symptoms, little myotatic irritability, little extensor spasm (and then both legs move together when one spasmodically extended leg is lifted); rarely by incontinence of urine or fæces, no trophic changes, no cystitis, no reaction of degeneration and no decubitus.

Gower's scheme for diagnosis from the *method of onset:*

Disease.	Onset.	Disease.
Pressure and Growths.	Sudden (few minutes).	Vascular Lesions.
	Acute (few hours or days).	
	Subacute (1 to 6 weeks).	Degeneration.
	Subchronic (6 weeks to 6 months).	
	Chronic (more than 6 months).	Inflammation.

In the *chronic stage* the diagnosis of what has gone before must be uncertain, unless the history is most definite; the diagnosis is usually provisional and atypical cervical pachymeningitis, tumors and most often multiple sclerosis of the spinal type should be considered.

Treatment.—This is symptomatic. In the *acute stage* the indications are (*a*) absolute rest in bed for at least two weeks, keeping the

patient on one side or on the face, possibly with local abstraction of blood. Blisters should be avoided lest they develop into decubitus. (*b*) Gowers recommends spt. etheris nitrosi with digitalis to equalize the circulation and promote diuresis. (*c*) When hemorrhagic myelitis is suspected ergotin (grs. iij to v) may be given three times daily. (*d*) Mercury and iodides are usually without effect in syphilitic myelitis (softening), possibly injurious, although some rationally recommend their use unless unfavorable effects appear. (*e*) Care of the skin. Though bed-sores may be trophic, experience shows that care greatly reduces their frequency and extension. Pressure and heat should be avoided by change of posture, rubber cushions, soft thick pads of cotton or the water bed. As in typhoid, the use of two beds is very beneficial and the sheets should be stretched smooth. Alcohol rubs are antiseptic and harden the skin. Moisture and filth are most dangerous; the bowels should be irrigated if they move involuntarily; incontinence of urine is more dangerous, especially in women; hard urinals, if left between the legs, may cause equal damage; the urine should be gathered in bags of oiled silk filled with cotton; in women mild antiseptic gauze and impalpable boric acid should be disposed about the genitalia. (*f*) Care of bladder is imperative. Cystitis develops in most cases in which the catheter is used, or in which there is incontinence with retention; residual urine is easily infected, probably by infection ascending the dribbling, patent urethra; the urethra should be irrigated before the absolutely clean catheter is introduced; lavage with Thiersch's solution in cystitis with retention or incontinence and grs. v to x, each of urotropin and salol t. i. d. are indicated. (*g*) The paralyzed muscles should be gently massaged from the very onset.

In the *subacute or chronic stage,* tonics, massage after four weeks, mild faradism of the bladder and rectum are indicated. Contractures are modified or prevented by warm baths and massage. Hot baths are directly dangerous.

II. Acute Multiple Disseminated Myelitis.—This affection is relatively infrequent. It is subacute in onset; foci develop irregularly, together or successively in different parts of the cord or perhaps of the brain (*disseminated encephalomyelitis*), with acute ataxia, scanning speech, dysarthria, tremor, nystagmus, optic neuritis, spasticity or weakness of the limbs. Sensation and the sphincters are usually not affected. The clinical picture is necessarily irregular, because the inflammation may occur anywhere; von Leyden distinguishes (*a*) the atactic and (*b*) the paraplegic forms. A myelitis which spreads after the first two or three days is of the disseminated type and may be fatal in a few days to a few weeks; once thought very fatal, numbers of recoveries have been reported lately. Treatment is as in the acute transverse variety.

III. Chronic Myelitis.—This may be the stationary stage of acute myelitis or in very rare cases myelitis may be chronic from the beginning, *i. e.,* a gradual onset and slow progression. Many deny the

existence of this form and classify it under the paraplegic state, multiple sclerosis or chronic system-disease. If myelitis, chronic from the onset, is to be recognized, its symptoms differ from the acute form only in the mode of onset.

IV. Acute Poliomyelitis of Children.—Acute poliomyelitis, also known as atrophic paralysis, was first described clinically by v. Heine (1840) and pathologically by Cornil (1863), although Prévost and Vulpian (1865) first accurately defined the changes in the ganglionic cells of the anterior horn. The famous contribution of Charcot and Joffroy (1870) fixed the trophic relation of the special cells involved to the atrophy of the nerves and muscles supplied and stated that the disease was due to primary inflammation of the cells themselves. Archambault and Damaschino found the primary inflammation in the vessels (interstitial myelitis).

Definition.—An acute, probably independent, infection, characterized pathologically by acute myelitis of the anterior horns and clinically by the incidence in children of an acute flaccid paralysis with wasting in the muscles, loss of reflexes, the reaction of degeneration and later contractures.

Etiology.—In some statistics it constitutes as high as 8 per cent. of the diseases of childhood.

1. *Age.*—The disease largely occurs between the second and third years of life; 60 per cent. of cases occur in the first three years and it is ten times as frequent in the first decade as in all the others. Its occurrence in youth is explained by Lange by functional activity of the cord, as learning to walk, etc. It has no causal connection with dentition, although often synchronous with it.

2. It may follow such *infections* as measles or scarlatina, and Marie thinks that it may result from many others (*infectiones innomminées*). Angina precedes most cases and is apparently the atrium. In some localities it has occurred as an independent epidemic, as Caverly's series of 140 in Vermont or Medin's series of 44 in Sweden. Alice Woods describes an epidemic of 60 to 70 cases in San Francisco in May and June, 1901. In 1907 there were nearly 3,000 cases in New York. Starr collected 54 epidemics.

3. Eighty per cent. of cases occur in the *summer months* (Sinkler). Sex is of no importance, save after the tenth year, when the disease is almost entirely confined to males (Gowers).

Pathology.—The affection is called poliomyelitis (Kussmaul), because it is an inflammation of the anterior gray matter, supplied by the central arteries, branches of the anterior median artery of the anterior longitudinal fissure. A single vessel does not supply all the anterior gray matter, but branches up and down, supplying a cylindrical vertical area which measures two inches or more; this is the usual dimension of a poliomyelitic focus. The inflammation may extend somewhat back of the anterior horns into the white matter of the antero-lateral columns. Fresh cases are rarely seen, since

death is unusual; to the naked eye the focus is red, soft, juts above the cut section and is mostly confined to the anterior horn, *i. e.*, it involves the beginning of the second or lower motor neurone. Microscopically, the lesion is inflammation in and about the vessels above named, in the anterior roots, and in the vessels even before they enter the cord. The vessels are engorged; their perivascular spaces are full of round cells and granule-cells; there is exudation of red and white cells with actual hemorrhage in the severe cases, as shown by blood pigment; the ganglion cells in the anterior horn are swollen, glistening, granular, pale, distended, decreased in number and surrounded by leukocytes; their dendrites may shrink, their nuclei disappear, or the whole cell may disintegrate, while here and there some cells escape; no definite cells are involved, since the process depends solely on the vessel affected. The involvement is sometimes bilateral and in severe cases is distributed through several segments, especially in the lumbar cord. As may easily be anticipated, disease in the trophic cells of the lower motor neurone must be followed by degeneration of their axis cylinders, nerve roots, peripheral nerves and muscles (which become granular, fatty and later indurated). In *old cases* macroscopic inspection shows shrinking of the anterior horns, perhaps also of the contiguous white matter, with lack of differentiation between the gray and white matter. Microscopically, the (formerly inflamed) vessels are thickened and pigmented; groups of ganglion cells have disappeared and their nerve fibers are degenerated or wholly wasted, which led Charcot to hold that the process was essentially in the ganglionic cells, but the connective tissue also is rich in cells and stains deeply with carmine, because it is thicker than normal; in other words, a cicatrix remains from the primary inflammation. In *severe cases,* identical changes have been found in the medulla, pons and centrum ovale. Acute poliomyelitis differs from acute myelitis and disseminated myelitis only in its localization, for which there is no universally accepted reason; the gray substance is more vascular and Lamy's experiments, in which he injected powder into the abdominal aorta, always showed embolic softening of the gray matter first.

Symptoms.—There are *three clinical periods:* 1. The acute stage (including prodromes). 2. The stage of diffuse paralysis, when degeneration appears and regeneration occurs. 3. The stationary stage, with some improvement and determination of the paralysis to given groups of muscles.

1. The Acute Stage.—The typical onset is acute, appearing in a child previously well, with fever (75 per cent.), often with vomiting, headache, somnolence and sometimes with convulsions or coma, which are followed by a sudden complete and flaccid paralysis in one limb or more than one limb; it corresponds to the acute inflammation in the anterior horn. The fever averages 101°, may reach 105° or more and lasts about 24 hours. Convulsions are not as common as in the cerebral type; they occur mostly in younger subjects and are attrib-

uted perhaps to teething, which was once thought the cause of the paralysis. The simple motor paralysis usually reaches its maximum in twenty-four to forty-eight hours; it is not progressive; its distribution will be considered under the next stage. In many cases *prodromata* exist, as *tonsillitis* or *angina,* swollen joints, malaise, anorexia, pain in the back and limbs or diarrhœa. *Unusual variations* in the onset are possible; the fever may last one or two weeks before the paralysis develops, it may last a few hours only or may be absent. The paralysis may develop during the night. Abortive cases are most exceptional and are probably often due to neuritis; the paralysis may follow some acute infection, as measles or scarlatina. Sometimes paralysis is not noticed for a long time, until, for example, the child fails to walk. In some cases pain is the first symptom in older children and is referred to the back or extremities. Convulsions involve principally the extremities and sometimes occur in the form of clonic muscular contractions; Strümpell observed them for more than a month before the paralysis developed.

2. The Stage of Degeneration.—This results from localization in the anterior horn, which produces (*a*) paralysis, (*b*) disturbed nutrition, (*c*) vasomotor changes and (*d*) loss of reflexes. (*a*) The *paralysis* is motor only; it is sudden, complete, flaccid and non-progressive; though at first diffuse, it usually regresses, although incompletely, leaving some permanent paralysis. *General localization,* according to Starr's combined statistics of about 600 cases, gives: (i) Paralysis, most frequently of one leg, then (ii) of both legs, and (iii) and (iv) of all four limbs or of one arm (with equal frequency), (v) of arm and leg of same side (spinal hemiplegia), (vi) of arm with opposite leg (crossed or alternating paralysis), (vii) of three limbs and least often (viii) of both arms. The trunk muscles may be involved, but the cranial nerves are exempt, save in severe and diffuse foci. The bladder is usually unaffected, except in the early stage, from general toxæmia, or in severe cases. As to its *special localization,* the *leg* is paralyzed in 75 per cent. of cases (E. Remak); in one form the lasting paralysis affects the quadriceps femoris with the adductors (upper lumbar lesion), but the sartorius is not involved; in another form the muscles below the knee supplied by the peroneal nerve are involved, as the tibialis anticus, extensor digitorum communis, ext. longus or brevis, ext. hallucis longus, peronei (lower lumbar and upper sacral lesion) of which some muscles may escape, as the tibialis ant., since total paralysis is rare. If both legs are involved, the paralysis is rarely symmetrical. Poliomyelitis usually involves several segments, because of the arterial distribution. The arm is paralyzed in 25 per cent., chiefly in the form of the combined shoulder paralysis of Remak, involving the deltoid (sometimes alone), biceps, brachialis, supinator longus with the supra- and infraspinatus and teretes (lesion in upper cervical enlargement, fifth to sixth segment); in some cases the forearm type may be seen, with involvement of the flexors or possibly of the extensors, while the

supinator longus often escapes (lower cervical lesion, seventh and eighth segments). The trunk or neck muscles usually show improvement in this stage. (*b*) As to *disturbed nutrition,* the muscles and nerves corresponding to the level of the poliomyelitis rapidly degenerate, as shown by early reaction of degeneration and muscular atrophy. The reaction of degeneration consists of failure of the nerve and muscles to respond to the faradic current and usually appears in one or two weeks (Duchenne) or rarely earlier; it also includes a lack of response of the nerve to the galvanic current and finally too prompt a response of the muscles to the galvanic current and more quickly to the positive than to the negative pole; this latter reaction lasts for two, three or six months and then gradually disappears in a year or two. Muscular atrophy may be apparent in two weeks and is more clearly seen in the arm than in the leg or trunk, whose adipose deposit may obscure it. Fibrillary contractions are rather frequent. The wasted flaccid member hangs and swings in a lifeless fashion. (*c*) *Vasomotor paralysis* causes the low temperature (1 to even 10° below normal) and the livid cyanotic or sometimes clammy skin. (*d*) The skin and tendon *reflexes* are abolished in the limb at first; the skin reflexes usually return later. The *sphincters* are almost always intact and sensation is very rarely affected. True trophic changes are almost unknown.

3. Stationary, Chronic, Residual Stage.—Some improvement may occur, but this stage is generally marked (*a*) by accentuation of *muscular atrophy,* connective and sometimes adipose tissue replacing the contractile substance; it is most marked in the legs in old cases, and involves also the nerves, arteries and tendons. (*b*) The *bones* become thin, flexible, friable, arrested in development and therefore shorter, since the bone cortex wastes and its medulla becomes more spongy; scoliosis may result. (*c*) There may be pronounced vasomotor lividity. (*d*) In the *joints,* nodules may appear as in cerebral hemiplegia; the joint structures are relaxed and the "flail joint" may develop with luxation or subluxation. (*e*) *Contractures* are due principally to the mechanical influence of the unopposed antagonist muscles on the posture of the joint; if all the muscles are paralyzed they sometimes result from static causes, as from standing or attempting to walk, *i. e.,* from neglect; trauma and malposition of the luxated joints or tendons may also cause contracture or actual deformity; the arm, the shoulder, wrist and fingers are usually flexed and the elbow usually escapes involvement. In the leg, deformity (pes equinovarus) is more common than in the arm.

Diagnosis.—The initial vomiting, fever and convulsions are usually misinterpreted until the diffuse purely motor paralysis appears and consequently the physician is unjustly blamed for what he cannot anticipate. The age, acute paralysis, rapid atrophy, reaction of degeneration and loss of reflexes are positive diagnostics, together with the absence of sensory and sphincter disturbance.

Differentiation.—(*a*) The various forms of *muscular atrophy* in

children are gradual in onset, progressive, symmetrical and often familial. The atrophy precedes and exceeds paralysis and there is no arrest in development. The neural progressive muscular atrophy of Hoffmann, due to disease of the nerves or nerve roots, may produce paralysis, atrophy and the reaction of degeneration, but the affection is progressive, familial and involves sensation. (*b*) Birth palsies are distinguishable by their history and more favorable course. (*c*) *Multiple neuritis* (*q. v.*) has several distinctive features. Poliomyelitis is due to a local inflammation, is unilateral and spinal in type—or if bilateral, it is asymmetrical. Multiple neuritis is hæmatogenous and toxic, and therefore affects the spinal, and often the cranial nerves symmetrically and bilaterally and usually involves the arms and legs; ataxia, sensory disturbance and local tenderness over the nerve trunks are common, its onset is more subacute and convulsions are rare; the reaction of degeneration is less intense and the issue is much more favorable. (*d*) The *cerebral palsies* of children (see page 950) affect the upper motor neurone and therefore never produce marked degenerative atrophy or loss of faradic irritability. Its hemiplegic form involves the face and its paraplegic form is spastic. Disturbance of the intellect, convulsions and hemichorea are common. In rare cases cerebral and spinal paralysis may co-exist. (*e*) *Myelitis* also begins acutely, but usually in older subjects. Poliomyelitis may be confused only when it is bilateral, located in the cervical or lumbar enlargement, when sensory changes occur (2 per cent.), the sphincters are involved, producing a flaccid atrophic paralysis of an arm and a spastic paresis of the leg, the latter of which is only transitory. Bedsores, so common in myelitis, are "almost unknown" in poliomyelitis. (*f*) Painful affections causing immobility of a joint, hip-joint disease, Parrot's syphilitic pseudoparalysis and infantile scurvy bear a superficial resemblance to poliomyelitis. Westphal noted that in the newborn the electrical reaction normally resembles the reaction of degeneration. (*g*) Hysterical contractures, sometimes with atrophy, disappear under anæsthesia and show no reaction of degeneration.

Prognosis.—(1) *Regarding life;* there is little danger after the fever subsides; life is very rarely threatened from extension to the medulla oblongata. In epidemics the mortality may reach 10 per cent. (2) *Regarding relapses;* second attacks almost never occur. (3) *Regarding recovery;* improvement is the rule. The diffuse initial paralysis usually becomes localized, but complete recovery is infrequent. (Starr asserts that 25 per cent. recover completely.) A localized initial paralysis may show but little improvement, and an extensive initial paralysis may diminish greatly. A definite prognosis of the ultimate extent of the paralysis cannot be made for two weeks and then only by the electrical examination. Muscles which show no reaction of degeneration after fourteen days will probably recover; those showing slight loss of faradic irritability will recover in great part; after six weeks only those muscles are paralyzed which will remain paralyzed; if there is no recovery in three months there will be none or it will be slight.

Treatment.—In the *acute stage* rest, a light diet, laxatives, diaphoretics and diuretics are indicated. After administration of urotropin, Cushing found formaldehyde in the spinal fluid; Starr recommends urotropin, gr. v every 4 hours. After the fever the patient should be kept in bed for weeks and should be given lukewarm baths. Electricity is contra-indicated. As the paralysis recedes a fuller diet and careful passive movements are beneficial. In the *chronic stationary stage,* with its inevitable and permanent paralysis, fresh air and liberal nourishment are important. Strychnine is valuable because of its general tonic action and its almost specific physiological action on the motor elements of the cord. It should be given for three or four weeks after the paralysis becomes stationary. Full doses may induce muscular spasm, pain and irritating paræsthesia. Electricity cannot restore totally destroyed tissue, but it hastens recovery in partial lesions; if applied to the spine it is useless, but the constant stream to the muscles and the faradic current to the skin are helpful. Contractures may be modified as well as deformities due to neglect, to which gymnastic movements, massage and orthopedic apparatus are essential. The child should be kept in bed for a long period. In some cases tenotomy, arthrodesis and tendon transplantation, by attaching the tendons of sound muscles to those of paralyzed muscles (Vulpius, Nicolodoni) are indicated. Nerve transplantation is proving very successful.

V. Acute Poliomyelitis in Adults.—This is rare in comparison with the infantile form. It was established by Vogt in 1858, Moritz Meyer, in 1861, and anatomically by Gombault, in 1873. The *etiology* is practically the same as that of the infantile variety; most cases occur between the twentieth and thirtieth years. The *symptoms* and course are essentially the same, with the following variations: fever is almost always present, which with the prodromes lasts longer than in children, even a week. There is the same flaccid paralysis with atrophy and the reaction of degeneration, but it regresses less and results less frequently in contractures. The types of Remak are more clearly developed in the adult. Pain is more prominent. Friedländer observed mixed cases with early, transient anæsthesia. The *diagnosis* is more uncertain in adults, chiefly because multiple neuritis and to a lesser degree myelitis and hæmatomyelia are more difficult to exclude, but in all three affections sensory alterations are usual.

VI. Subacute and Chronic Poliomyelitis.—Many reported cases are probably multiple neuritis, although Oppenheim published perfectly clear cases. *Etiology.*—Metallic poisons and syphilis are important factors. *Pathologically,* two main types are distinguished; in the larger group there is no inflammation but rather a primary atrophy of the ganglionic cells, which heightens its resemblance to spinal progressive muscular atrophy, than which, however, it runs a more rapid course; in a smaller group of cases there is early perivascular exudation in the anterior horn and later sclerosis, wherein it resembles acute poliomyelitis, although it is slower in its course. The anterior

roots are involved and in most cases there is degeneration in both pyramidal tracts, possibly also in the posterior columns or roots, in Clarke's columns or rarely in the medulla. The *course* is gradual; one extremity becomes slowly affected (in one to three years), sometimes with extension to others after months or years. There are paresis with atrophy, fibrillary tremors, partial or complete reaction of degeneration, decrease or absence of the reflexes and normal sensation. Life is rarely immediately threatened, but the ultimate outcome is unfavorable; if recovery occurs, neuritis, not poliomyelitis, is the actual lesion. *Differentiation* is as follows: (*a*) Multiple neuritis (see ACUTE POLIOMYELITIS); (*b*) from *progressive spinal muscular atrophy,* even an anatomical differentiation is not easily made. As a rule, the following clinical distinctions hold; in progressive muscular atrophy, atrophy precedes and exceeds the muscular weakness, fiber after fiber of muscle very slowly wastes, the atrophy is especially marked in the distal parts as in the hands and fingers, and the reaction of degeneration is usually less complete. In chronic poliomyelitis, atrophy always rapidly follows paralysis; it is an outright atrophy (*atrophie en masse*), it occurs more often in the proximal parts, as in the shoulder, and the reaction of degeneration is marked. (*c*) In amyotrophic lateral sclerosis the paralysis is spastic with increased reflexes, contractures and often with bulbar symptoms. *Treatment* is the same as that of the chronic stage of acute poliomyelitis.

VII. Landry's Paralysis.—Landry's paralysis (acute ascending paralysis) was described by Landry and independently by Kussmaul in 1859. Landry's description included five cases of his own and five others; the famous Cuvier died of this disease in 1832. Acute ascending paralysis is a symptom complex rather than a disease in itself and may be conveniently classified under inflammatory affections of the cord. The *etiology* is vague. The affection occurs mostly between twenty and forty years of age and three times more frequently in males than in females. It has followed acute infections, as diphtheria, typhoid or influenza and chronic infections as syphilis and tuberculosis.

Pathology.—In the early and in many recent cases no anatomical findings existed, and on this Westphal (1876) founded his three criteria: (*a*) that the disease is a progressive, ascending and finally fatal affection with (*b*) normal electrical reaction and (*c*) negative findings at autopsy. Though negative findings are frequent, recent cases have given definite though varying postmortem results, as structureless exudation in the cord, disseminated foci of inflammation in the cord, medulla and pons, changes in the pyramidal tracts, anterior horns and in the spinal and sometimes in the cerebral peripheral nerves. By some writers two forms are distinguished: (*a*) the *bulbar or medullary form,* in which sensation and electrical reactions are normal, and (*b*) the *peripheral neuritic form,* with sensory and

electrical alterations. The parenchymatous swelling of the liver, spleen, kidneys and lymphatics indicates a general infection or toxæmia. Bacteriologically, an anthrax-like bacillus (Baumgarten), the pneumococcus, typhoid bacillus, bacilli in all the peripheral nerves and streptococci (Œttinger and Marinesco) have been found.

Symptoms.—The onset is acute without or with prodromes, as head- and back-ache, muscular spasms and paræsthesia. Paralysis commences in one foot, then in the other, and in the legs, thighs, abdomen, back, arms, even in the face or the eyes, possibly with optic neuritis; the paralysis is progressive and ascending; when the medulla is invaded, dysarthria, dysphagia and paralysis of the tongue occur and finally death results by respiratory paralysis or in atypical cases by circulatory paralysis. The proximal parts of the limbs are more involved than the distal and some muscles may be unaffected. Fever is usually absent; the sensorium, sensation, sphincters and electrical reaction are usually normal. There are no tremors, no twitchings, no convulsions, no contractures, no trophic disturbance. The reflexes are usually decreased or absent. In rare cases there may be partial reaction of degeneration (which usually has not time to develop), atrophy and moderate disturbance of sensation or the sphincters. The paralysis may be of the descending type, in which event the bulbar involvement is fatal before the paralysis in the legs is well developed.

Course and Prognosis.—The disease lasts two days to two weeks, and usually terminates with a respiratory paralysis. Bulbar symptoms are ominous, but not necessarily fatal. The central form of paralysis is fatal and the peripheral form probably includes most of the recoveries. In rare cases, death may occur in a few hours or only after months. In the still rarer cases of recovery, some chronic paralysis may remain. If recovery ensue, the paralysis regresses in an order inverse to that of development. A *diagnosis* is usually easily made, because of the ascending motor paralysis with loss of reflexes and without marked mental, sensory, sphincter or electrical alteration.

Treatment.—The treatment is that of myelitis or multiple neuritis. Strychnia and other stimulation are indicated if the bulbar symptoms are manifested. C. L. Greene reports a case which was kept alive by artificial respiration forty-one days after respiratory failure set in; he advises calomel, colonic flushings and oxygen inhalations.

MULTIPLE SCLEROSIS.

Insular or disseminated sclerosis, *sclérose en plaques disseminées,* was described pathologically by Cruveilhier and Carswell (1838); Frerichs, in 1849, made the first diagnosis, and Charcot (and Vulpian), in 1863–64, gave a description of the typical case.

Etiology.—In fully 50 per cent. of the cases no cause is found and little is known of its etiology. It occurs equally in either sex and 90 per cent. occur under the fortieth year; cases resembling multiple

sclerosis (pseudosclerosis) occurring after the fortieth year are considered arteriosclerotic. Three main factors are thought to exist: (*a*) *Acute infections,* as typhoid, pneumonia, measles, smallpox, etc.; Marie adopted this theory. (*b*) The idea that chronic intoxications with alcohol, lead, etc., are causal. (*c*) Strümpell and Müller favor the developmental theory.

Pathology.—Macroscopically, the *sclerotic foci* are usually seen with ease as irregular grayish-red areas as large as a pea or walnut; they are multiple (numbering even over 100), are distributed at random and occur largely *in the white substance,* although Oppenheim and Taylor found equal involvement of the gray matter. In most cases their distribution is *cerebrospinal,* in rarer cases in the cord alone, and most rarely in the brain only; the brain cortex is less frequently involved than the centrum ovale, central ganglia, corpus callosum and the anterior under surface of the pons and the crus. The medulla is quite often and the cerebellum rarely involved. The white matter in the cord is more frequently affected than in the brain. *On section,* the focus is a trifle sunken and transparent, is fairly well demarked, firm save in the freshest foci, and on hardening in Müller's solution, appears light green, while the normal tissue is a darker green color. Microscopically, the medullary sheaths in the white substance are thinner or absent and though the *axis cylinders* are somewhat wasted and at times varicose, their *remarkable persistence,* first noticed by Charcot, causes *the incomplete character of the symptoms* and the usual *absence of ascending and descending degenerations.* The ganglion cells suffer some atrophy and pigmentation, though in general they resist the sclerosis fairly well. The foci stain deeply with carmine, are not sharply demarked from the healthy tissue and show corpora amylacea, granule-bearing cells, increase of the fine meshed neuroglia fibrils, nuclear proliferation and sometimes Deiter's cells. In contrast with the system-degenerations, the primary change is interstitial with secondary changes in the nervous tissue. Some pathologists, as Ribbert, see significant changes in the bloodvessels of early cases, as arterial thrombosis, followed by exudation and neurogliar proliferation; this theory is vigorously opposed by Strümpell. The cerebral nerves suffer often, especially the optic nerve, in which proliferation of connective tissue is followed by loss of the medullary sheaths, while the axis cylinders are unaffected. The nuclei, roots and nerve trunks of the third, eighth, seventh and other cranial nerves may suffer similar alteration. The spinal nerve roots are not infrequently involved.

Symptoms.—The clinical picture is so variable that a definite description cannot be made to cover all cases. Charcot's description included the intention tremor, the nystagmus, scanning speech, motor weakness and rigidity and apoplectic insults, although he recognized atypical forms (*formes frustes*).

1. Motor Symptoms.—These are the most important. (*a*) *Muscular weakness* is very common. Paresis is more frequent than par-

alysis, since some axis cylinders remain intact. The movements are slow and fatigue rapidly follows their repetition; this statement applies to isolated rather than to synergistic movements. The paresis is most apparent in the legs, there is usually some in the arms and frequently some in the musculature of the eyes, head and speech. It sometimes does not develop until late in the disease, may at no time be conspicuous, and is commonly associated with increased reflexes and spasticity. The gait is spastic, scuffling and the feet seem to stick to the ground; to this is sometimes added a cerebellar, atactic or paretic element. Muscular atrophy is rare but may result from an adventitious focus in the anterior horn. The electrical reactions are practically normal. (*b*) *Muscular rigidity is the most frequent finding,* is referred to disease of the lateral columns and is most developed in the legs. It increases with successive movements; for example, each step becomes more stiff in walking. It sometimes amounts only to a sense of stiffness; in other cases the spasticity is so extreme and the muscles so hard that the limb cannot be moved even with great effort. Contractures develop, which usually affect the adductors and extensors first and the flexors later and draw the heels up to the buttocks. Contractures often affect the muscles of the neck. (*c*) *Tremor* is one of the most striking phases of the disease, being present in 75 per cent. of cases. It is caused, as is the paresis, by the loss of the medullary sheaths (Charcot) which allows diffusion of nerve impulses, by foci in the pons or thalamus (Erb) or by lack of coordination between antagonistic muscles (Strümpell, v. Leyden); the axis cylinders remain intact. There is no tremor during rest, but it appears when voluntary efforts are attempted, whence the name intention tremor. The hand carrying a glass of water to the mouth shakes so much that all the water may be spilled by the rhythmic tremor, which possesses a wide range, and occurs five or six times to the second. It is apparent in the head when the patient sits, because the muscles of the neck are in constant action to support the head; it disappears in the recumbent posture. Similar tremor occurs in the face, larynx, trunk or legs on changing posture, as rising; in the fingers it produces handwriting which becomes more erratic with each word written; and in the thoracic muscles it causes a species of cogwheel respiration. (*d*) *Nystagmus* occurs in 50 per cent. of cases and is elicited by having the patient follow with his eyes the index finger when moved to one side of his field of vision, his head remaining stationary. His eyes show short, jerking lateral movements. It is most common on lateral or vertical movements. Spontaneous nystagmus is rarer, as nystagmus on attempting to focus the eyes. It must be remembered that it may occur without organic disease, as in general weakness or convalescence. There is no satisfactory explanation; it is considered a tremor or an incoördination. (*e*) *The speech* is *scanning* (54 per cent.) *syllabic* or *staccato;* it is slow, monotonous, unmodulated and sometimes explosive, especially for the letters c, p, g and d. The speech muscles become fatigued easily and

the end of a sentence is often elided; the sounds are often nasal, interrupted by yawning inspiration or by expiration at the beginning of each syllable. Leube observed the vocal cords oscillate during their movement. The cause of the peculiar speech is not clear; it may be due to tremor or ataxia from foci in the pons or medulla. (*f*) *Paresis of the eye muscles* (in 17 per cent. Uhthoff) affects the third or sixth nerves, convergence or associated movements. About 50 per cent. are nuclear. Transient diplopia, unequal pupils, rhythmic spontaneous widening and narrowing of the pupils (hippus) and strabismus are less common. (*g*) *Apoplectiform seizures* (in 20 per cent.) develop without prodromes and are marked by partial, rarely by complete coma, increased pulse, high temperature, aphasia and hemiplegia, which is usually fugitive and flaccid, rarely enduring or spastic. They may recur. In most cases negative pathological findings exist, but Leube and Leyden refer them to acute encephalomyelitis. Though usually hemiplegic, they may exceptionally produce paraplegia, hemianæsthesia or ataxia, or monoplegia. Less frequently, unilateral convulsions occur.

2. Sensory Symptoms.—These are less frequent (30 per cent.) because the sensory fibers are more resistant to disease. Freud found sensory symptoms in 80 per cent. of his cases. *Subjectively,* dull pains, formication and paroxysmal headache are frequent and lightning pains, crises, girdle sensation, trigeminal neuralgia, palpitation and dyspnœa are very uncommon. *Objectively,* any species of sensation may be involved, especially in distal parts of the extremities, where it is usually transient. *Ataxia* is usually referred to disease of the posterior columns or sensory tracts of the pons or medulla; it is marked in 50 per cent. of cases and may produce a typical tumbling cerebellar gait.

3. Special Senses.—The optic nerve is affected in 45 per cent., usually as a pallid partial atrophy involving its temporal side, and less often as neuritis or complete atrophy with total blindness. The change is more conspicuous pathologically than clinically. Optic neuritis is transient and runs a rapid course; in 60 per cent. of cases it produces central scotomata, although at times it narrows the field of vision. It may be the first sign of the disease. Hearing and taste may be affected.

4. Psychical Symptoms.—Slight mental impairment is common; the patient is indifferent and singularly complacent; the memory is weak; psychoses are rare. Compulsory or forced laughing and weeping are partly without motive and partly emotional; they probably should be referred like the analogous compulsory yawning and swallowing to lesions in the medulla, pons or crus.

5. Bulbar Symptoms.—These may, in rare cases, resemble the symptoms of bulbar palsy, as dysphagia, anarthria and tongue atrophy.

6. Reflexes.—The tendon reflexes are increased, most markedly in the legs. The patellars are exaggerated, there is ankle clonus and tickling of the sole produces dorsal flexion instead of the normal

plantar flexion of the great toe (Babinsky's sign), showing disease of the pyramidal tracts. The skin reflexes are usually said to be normal, but Strümpell found the abdominal reflexes absent in 70 per cent. of his cases. The pupillary reflexes are usually normal.

7. Sphincters.—Lasting or severe symptoms in the bladder or rectum are uncommon, though slight disturbance occurs in most cases. The bladder is often hypertonic and irritation is frequent.

8. Trophic Symptoms.—These are rare; they consist of skin eruptions, œdema, sweating, chronic arthritis and erythromelalgia.

Course and Prognosis.—The onset is usually gradual; the spinal symptoms are most prominent and in most cases the weakness in the legs appears first and for a long time is hard to interpret; it occurs without pronounced sensory symptoms, bladder disorder or atrophy. Sometimes the onset is acute, with loss of consciousness, transient hemiplegia, vertigo, headache, eye symptoms, vomiting and optic neuritis. In the average case the leg paralysis is followed by scanning speech, nystagmus and tremor, which cover a chronic course averaging three to six (or even more than twenty years). A rapid course, as twenty-three months, may occur in the bulbar form. The symptoms remit and recur. Progression to a fatal ending is inevitable. Charcot distinguished *three stages* (*a*) the period from the development of the first symptoms to the *tremor and paresis,* (*b*) the period in which the patient is *bed-ridden,* and (*c*) the *terminal stage* with dementia, decubitus, sphincter paralysis, cystitis and septicopyæmia.

Diagnosis.—The typical case is unmistakable, because of the cardinal symptoms, (1) intention tremor; (2) spastic paresis; (3) nystagmus; (4) scanning speech; (5) ataxia; (6) increased reflexes; (7) optic atrophy; (8) the slow, uneven course with remissions and recurrences; (9) the variability of the symptoms; (10) apoplectic insults; and (11) moderate involvement of the mentality, sensation, bladder and rectum.

Differentiation.—Transient hemiplegia may suggest hemorrhage, syphilitic thrombosis, embolism or tumor (*q. v.*). If these can be excluded, multiple sclerosis or paretic dementia must be differentiated (*q. v.*). *Paralysis agitans* is usually but rather unnecessarily differentiated; the absence of the cardinal signs of sclerosis multiplex and the presence of a constant fine tremor of the hands and fingers (not of the large joints, and much less of the head) which is not increased by movement, its persistence during rest, the mask-like facies, festinating speech, peculiar attitude, gait, propulsion, retropulsion, muscular rigidity without increased reflexes and the advanced age are clear and characteristic points.

Multiple Sclerosis.——*vs.*——Syphilis.	
Occurs in the medullary substance.	Occurs in the periphery of the cord (meningomyelitis and root symptoms).
A focal disease.	Diffuse.

Multiple Sclerosis.	*vs.*	Syphilis.
Nystagmus, scanning speech, tremor.		More frequent mental obscuration, insults, hemiplegia, etc.; speech may be disturbed or slow in pons lesions but is not scanning.
Transitory amaurosis, hemianopsia, complete optic atrophy, choked disk or neuritis are very rare.		Amaurosis, etc., are frequent.
Partial optic atrophy occurs.		There is more disturbance of vision.
Mydriasis 1 per cent., myosis 4 per cent. Argyll-Robertson pupil never present.		Mydriasis is very common. Argyll-Robertson pupil frequent.
		Cortical symptoms may be present; rectum and bladder more frequently involved; results may be obtained in early stages from antisyphilitic remedies.

Hysteria more than any other disease simulates early multiple sclerosis; the two affections often occur together and in both the symptoms are changeable. In hysteria sensory symptoms are much more prominent, there are often limitation of the visual fields, monocular diplopia, contractures and convulsions, but there is no optic atrophy, nystagmus is extremely rare, as is the scanning speech; and intention tremor may occur, but is not so wide. The movements in hysteria are often accompanied by contraction of the antagonist muscles, which is a most valuable diagnostic aid. In extremely rare cases, described first by Westphal as *pseudosclerosis,* similar symptoms prevail though the autopsy shows nothing; the tremor affects the arms chiefly; there is no ataxia or nystagmus; the facies is somewhat rigid and outbursts of anger are frequent; syphilis is a possible cause. *Birth palsies* may be accompanied by jerky movements, incoordination of the legs and even scanning speech, but the history of very early onset is conclusive.

Strümpell groups the *atypical forms* as (*a*) the latent group; (*b*) those resembling chronic myelitis, or (*c*) pure spinal spastic paralysis, maybe resembling amyotrophic lateral sclerosis if the anterior horns are involved or chronic bulbar palsy if the medulla is affected; optic atrophy in such cases indicates multiple sclerosis. (*d*) Cases resembling tabes, with ataxia and pains (probably combined system disease, *v. i.*); (*e*) the hemiplegic form; and (*f*) the psychical form.

Treatment.—Treatment is futile. Drugs, as silver and arsenic, are inert and hydro- and electro-therapeutic measures are useless. The patient should live in a warm climate during the winter months.

SYRINGOMYELIA.

The term refers to cavities in the cord, and was first employed by Ollivier (1824). The affection was first described by Morgagni.

Syringomyelia must be distinguished from hydromyelus (hydrorrhachis interna), which is a dilatation of the central canal due to developmental anomalies, possibly to stasis of cerebrospinal fluid resulting from pressure; it is usually an accidental finding at necropsy or if symptoms are present they are those of syringomyelia.

Etiology.—Sixty-three per cent. of cases occur between the tenth and thirtieth years and 70 per cent. in males. Other factors, as trauma, are wholly uncertain.

Pathology and Pathogenesis.—1. Gross Pathology.—A cavity may be seen from without. The cord is often lax, sometimes fluctuating or flat. The cavity is usually localized but may sometimes extend from the medulla to the filum terminale; it is most often found in the lower cervical or upper dorsal region, less frequently in the dorsolumbar segments or in the lower brain; in size it ranges from a narrow slit to a cavity as wide as the cord itself; it is not absolutely symmetrical and may be unilateral; it may communicate with the central canal, may be multilocular from septa or strands of tissue and diverticula may be found. Its wall is smooth, sometimes pigmented from old hemorrhages. Sometimes the cord is normal, but it usually suffers thickening and sclerosis develops from the ependyma, posterior commissure or posterior horns in the form of hard or soft, tumor-like, somewhat asymmetrical neurogliar proliferations, especially in the cervical region; these later break down and form secondary cavities. The posterior horns and columns are often affected, the anterior horns and columns less frequently and the lateral columns very rarely. Hemorrhage into the gliomatous tissue or the cavity may result from rupture of the new-formed bloodvessels.

2. Minute Pathology.—The essential finding is overgrowth of the neuroglia, which in part breaks down and forms cavities. This hyperplasia is most probably due to some congenital anomaly, which also explains its frequent association with dilatation of the central canal (hydromyelus). The neurogliar overgrowth is rich in fibers, whereas the ordinary glioma (gliomatosis) especially abounds in cells; syringomyelia and gliomatosis may co-exist. The proliferation is seemingly embryonic in character, contains some large sharply nucleated cells, here and there a few epithelial cells, is firm as a whole, but somewhat softer towards the posterior horns, is often hyaline nearest the cavity from compression and abounds in bloodvessels. The cavity is usually lined with cylindrical epithelium like that of the central canal, sometimes only with neuroglia; its contents resembles cerebrospinal fluid, but is occasionally blood tinged or less often gelatinous, mucoid or milky. *Degenerations* occur in parts actually involved and at times in distant parts, as in the medulla. The nerve fibers are not much involved, although secondary degeneration may occur in the posterior and less frequently in the lateral columns.

The exact *pathogenesis* is obscure. Congenital hydromyelus (with primary neurogliar hyperplasia and secondary cavity formation), inflammation and hemorrhage are not causal factors.

Symptoms.—There are *three cardinal symptoms.*

1. Anæsthesia.—*Partial or dissociated anæsthesia* occurs in the typical case, in which the sensation of touch, pressure and locality is preserved while heat or cold and pain are not recognized as such but as simple pressure. *Thermo-anæsthesia* usually concerns perception of both heat and cold, sometimes of only one of them, or one in one place and the other in another; *analgesia* is frequently preceded by hyperalgesia; it concerns the superficial or deeper structures and usually corresponds with the thermo-anæsthesia. Thermo-anæsthesia and analgesia begin in the hands and fingers and are later found on the upper trunk; sometimes the entire trunk, legs, mucous membranes and face may be affected, depending on the location of the cavity. The patient may suffer burns or injuries in these parts without pain. Dissociated anæsthesia is most probably caused by disturbance of conduction of temperature and pain in the gray substance of the cord, which is frequently invaded by neurogliar proliferation and the cavity formation; tactile sensation traverses the white columns. In some cases the partial anæsthesia is unilateral. Anæsthesia may involve all varieties of sensation. Subjective sensory disturbances include paræsthesia and burning, tabes-like pain in the neck, arms or trunk and sometimes in the joints, spine or legs.

2. Muscular Atrophy.—Muscular atrophy is due to involvement of the anterior horn and affects the arms mostly, because the disease is largely in the lower cervical and upper dorsal cord. It is slow in onset, more marked than the attendant flaccid paresis, degenerative and often observed with fibrillary muscular contractions and increased myotatic irritability. It usually involves the small muscles of the hand, as the interossei, produces flattening of the thenar and hypothenar eminences and the claw-like hand, as seen in the Duchenne-Aran type. The radial supply is first affected, then the ulnar and median. Sometimes the scapulohumeral type is observed, especially with bulbar symptoms. The muscles of the trunk, and much less often of the legs, may be affected. From cervical compression of the pyramidal tracts, paraplegia results, with increased reflexes and spasticity, in 12 per cent. of cases. Other motor symptoms occasionally occur, as tremor, contractures, spasms, opisthotonos or choreiform movements. The gait is normal, spastic or paretic. Romberg's sign and ataxia result from involvement of the posterior columns or coincident tabes.

3. Trophic Symptoms.—These are frequent. (*a*) Phlegmons and *felons* develop, particularly on the fingers. They are painless, chronic, often recurrent and lead to necrosis of the bone, ankylosis of the joints, deformity or mutilation. (*b*) The *joints* suffer in over 10 per cent., especially in males (75 per cent.), probably due to loss of articular sensation (whereby the joints are used inappropriately) or to lack of the nutritional reflex. These arthropathies are of the hypertrophic form, with thickening and bony formation, like arthritis deformans or of the atrophic form with relaxation of ligaments or

flail joint. There is little exudation, no fever, no pain. On the whole they are similar to the tabetic joints but are more chronic and more often involve the upper extremities (shoulder 32 per cent., elbow 28 per cent., hand 16 per cent., hip 8 per cent., ankle 8 per cent., knee 6 per cent.). (*c*) *Painless bone necrosis* and spontaneous fractures may occur late in the disease; moderate scoliosis is frequent, usually confined to the dorsal spine, sometimes with kyphosis; it is probably due to trophic vertebral changes or possibly to atrophy of the back muscles; perforating ulcer, thick, friable nails and clubbed fingers are less common. Morvan (1883) described an affection as *paresie analgesique avec panaris,* endemic in Brittany, which is clearly syringomyelia. "Morvan's disease" is marked by trophic disorders, excessive or deficient secretion of sweat, œdema especially on the dorsum of the hands, erythema, urticaria, more rarely local asphyxia and herpes zoster.

4. Other Symptoms.—(*a*) The *reflexes* of the skin are usually normal; the abdominal, cremasteric and plantar reflexes are sometimes increased; the tendon jerks are variable, being often decreased in the upper and increased in the lower extremities. (*b*) The *sphincters* are not involved, or only late in the disease. Retention of urine and constipation are more frequent than incontinence. (*c*) *Bulbar or pontine symptoms* are not infrequent, are usually unilateral and appear in the later stages. They include facial hemiatrophy or paresis, paresis of the tongue with atrophy, partial dysphagia, paresis of the palate (nasal speech), paralysis of the vocal cords, of the external branches of the spinal accessory nerve (trapezius paralysis) and trigeminus (sensory symptoms), eye paralyses (abducens), unequal pupils, nystagmus, tinnitus, salivation, apoplectiform attacks, polyuria, glycosuria and vagus symptoms (disturbed pulse or respiration, vomiting and singultus). *Complications* include hysteria, paretic dementia, tabes, pachymeningitis, Graves' disease and epilepsy.

Diagnosis.—An almost positive diagnosis can be made from the three combined cardinal symptoms; (1) the dissociated anæsthesia (thermo-anæsthesia and analgesia with practically normal tactile sensation) is the earliest, most constant and widely distributed sign. Grasset has collected cases of twenty or more nervous diseases (functional, traumatic, neoplastic, specific) in which the dissociation of syringomyelia has been found; but it remains highly distinctive in conjunction with (2) *muscular atrophy* of the *upper extremities* with paresis and (3) *trophic disorders, felons, arthropathies* and scoliosis; to which three cardinal manifestations may be added the spastic paresis of the legs and bladder symptoms. Besides the classical form, various varieties are described by Schlesinger; (*a*) the latent form, (*b*) the motor form, simulating amyotrophic lateral sclerosis, (*c*) the sensory type, possibly resembling hysteria, (*d*) the trophic and (*e*) the tabetic types.

Differentiation.—*Progressive spinal muscular atrophy* and *amyotrophic lateral sclerosis* may resemble syringomyelia with poorly de-

veloped sensory symptoms (Clarke and Gull, 1862) but they possess very few sensory signs, no anæsthesia, no trophic disturbance and bulbar symptoms are bilateral. *Juvenile muscular dystrophy* may be simulated in the scapulohumeral type of syringomyelia. *Peripheral neuritis* rarely produces dissociated anæsthesia, but all variations of sensation are disturbed; the distribution of the paralysis is peripheral, not spinal or segmental, and there is the reaction of degeneration; neuritis of the upper brachial plexus (Duchenne-Erb paralysis) produces paralysis of the shoulder, arm and supinator longus, a combination not found in syringomyelia. *Focal disease* of the anterior and posterior gray substance, as multiple sclerosis, hemorrhage or myelitis may simulate syringomyelia, but their course is less chronic and there is less trophic disorder. The signs of irritation produced by tumor of the cord are lacking. Located in the dorsolumbar region, syringomyelia may closely simulate *tabes,* from the lancinating pains, crises and ataxia, but the Argyll-Robertson pupil is lacking and the sensory impairment is typical. The anæsthetic form of *leprosy* has caused difficulty but its anæsthesia is peripheral, not spinal, in type; in the lesions of the skin, ulcers and nasal secretion the Henssen bacillus is often found; if there are no lesions in the skin, thickness and tenderness of the nerves indicate leprosy.

Course and Prognosis.—The onset is gradual and the course chronically progressive for years or even decades. When recognized, the affection is advanced and recovery impossible. The first symptoms are pain and dissociated anæsthesia, followed by atrophy and paresis, with trophic changes in the advanced course. Death occurs from cystopyelitis, septicæmia and bulbar complications.

Treatment.—Treatment is unavailing, being confined to hygienic and symptomatic measures. The danger of self injury, as a result of the peculiar anæsthesia, should be borne in mind.

SYSTEM DISEASES.

These constitute a small group of very important diseases and involve *structures or systems having a common function,* thus differing from such indiscriminate or random disease as myelitis or multiple sclerosis. The essential change is a *primary slow degeneration of the nerve elements,* followed by *secondary proliferation of connective tissue* (sclerosis). The pathogenesis of these degenerations is not clear, although *hereditary weakness* causing nutritional failure at a certain age (the familial form) and the *selective action of toxins* in the blood acting on certain nervous structures have been thought causes.

I. System Disease of the Sensory Tract.

The sensory type of system disease is locomotor ataxia in which the peripheral sensory neurone is affected, in the posterior roots and columns, while the motor types are spastic paraplegia, involving the

upper motor neurone (pyramidal tracts) and muscular atrophy due to lesions in the lower neurone (anterior horns and roots).

Tabes Dorsalis, Locomotor Ataxia (Duchenne).—**Definition.**—A disease characterized (*a*) etiologically, by syphilis; (*b*) pathologically by lesions in the posterior spinal roots and posterior columns, in the cutaneous and muscular sensory and also the cerebral nerves; and (*c*) clinically by incoördination (ataxia), sensory symptoms (pains and defective sensation), loss of myotatic irritability (of the tendon reflexes), loss of the pupillary reflex to light and trophic disturbances.

It is the best known and most frequent chronic disease of the cord. Horn in 1827 partly described the affection, although Todd in 1847 gave the first accurate account, eliminated paralysis, and found the posterior columns diseased at autopsy. Stanley first (1840) referred the affection to the posterior columns and peripheral sensory nerves. Türck first examined the tissues microscopically and Romberg (1851), and especially Duchenne (1858), popularized the disease; it is still known in France as Duchenne's disease.

Etiology.—*Syphilis* is the essential causal factor (Fournier and Erb) in 75 per cent. (Gowers), 90 per cent. (Erb and Strümpell), or 100 per cent. (Möbius). In about 10 per cent. of cases the syphilitic history and stigmata are lacking, whence it is sometimes attributed to other causes, as trauma, heredity, concussion, exposure, toxins of soft chancres and ergotism; but it is most probable that these are only incidents and that syphilis is the sole cause. The following etiological data of tabes are practically those of syphilis: *age:* 50 per cent. in the thirtieth to fortieth year; 25 per cent. in the fortieth to fiftieth year; less than 25 per cent. in the twentieth to thirtieth year (Gowers); cases may occur in persons over fifty, under twenty, or even ten years of age, the latter of which are probably due to hereditary syphilis. Forty-seven juvenile cases were collected by Hirtz (1905); of these, the parents in thirteen instances had syphilis, tabes or dementia. *Sex:* as in syphilis and paretic dementia, ten cases occur in males to one in females. Syphilis is more difficult to diagnosticate in women and more often runs a latent course. Like syphilis, tabes is sometimes familial; Nonne (1904) saw tabes in a mother and two daughters. It prevails largely in cities and among the higher classes ("syphilization and civilization"). It is uncommon in the negro, though Hecht has seen cases. Tabes is a meta- or parasyphilitic, degenerative lesion occurring on an average six to twelve years after the chancre. Dalous (1904) collected 21 cases with active syphilis.

Pathology.—The *gross changes* are usually visible to the unaided eye; the posterior columns are smaller, outwardly flattened instead of rounded, and appear gray from loss of the medullary sheaths and translucent from primary loss of nerve fibers and secondary increase of connective tissue. The pia may be secondarily opaque and thickened. The degenerated areas are somewhat sunken on section. The

posterior horns and roots are always somewhat atrophied and gray or grayish-red in color. As to *minute changes,* the toxins (Strümpell) bring about a very *slow degeneration of the peripheral sensory neurone,* its fibers and the processes of the spinal ganglia cells, which themselves suffer much less and later obvious changes than their processes. These processes run in two directions: to the periphery and to the posterior roots and posterior columns. (*a*) The *peripheral spinal sensory nerves* are often degenerated (Westphal, Pierret, Déjérine), less in large trunks (sciatic or crural nerves) than in the peripheral filaments of the skin, joints and muscles which are important in the sensory, trophic and atactic symptoms of tabes; their white substance wastes and then their axis cylinders. The trophic centres for these fibers are largely in the root-ganglion, but there is some evidence that there are also some peripheral trophic cells. (*b*) The *fibers running from the ganglia to the posterior roots* are more early, regularly and obviously changed. In the cord the degenerated tracts stain deeply with carmine and include only structures derived from the posterior roots and their collaterals, all others escaping involvement. The posterior root fibers run (i) to the posterior columns, whence there occurs, low down in the cord, degeneration in Burdach's and then higher up in Goll's columns, into which the root fibers are pushed as new and higher root fibers enter the cord. The location of the degenerated fibers therefore differs at successive levels and the degeneration is an *ascending* one. In the *lumbar cord* the posterior columns are most intensively and extensively diseased, especially in their middle and posterior parts, nearest the posterior roots and surface of the cord. Early and marked degeneration is found in the fine fibers of Lissauer's zone which lies just back of the posterior horn. The anterior part of the post. ext. column and the central part of the post. int. column are slightly, if at all, diseased as they are not derived from the post. ganglia. In the *dorsal cord* the post. columns are degenerated save in the post. ext. part and in the ant. part. In the *cervical cord* the post. int. columns are diseased (representing the root fibers of the lumbodorsal cord) and also some areas in the post. ext. column where degenerated root fibers have just entered. There is some pathological as well as clinical variation in different cases; Marie holds that one set of sensory fibers after another is involved and that degeneration develops by segments. Tabetic changes are usually most developed in the lower cord, but in the rare "cervical tabes" the upper cord suffers severely and perhaps exclusively. (ii) From the post. roots a second series of fibers run to Clarke's column. They and their collaterals are degenerated but the cells of Clarke's column (and their ascending fibers to the cerebellum) usually remain intact. (iii) The post. horns degenerate, because they contain fine root fibers. (iv) The reflex collaterals to the ant. horns also degenerate.

As already stated, pronounced or early atrophy in the cells of the post. ganglia is rare. Degenerative changes may involve other analo-

gous neurones, as the *auditory, optic,* trigeminus, visceral, sympathetic or ocular nerves, or less often the peripheral motor nerves, the pyramidal tracts, the muscles themselves, the medulla and the brain cortex. In old cases, corpora amylacea are found.

The exact *pathogenesis* is disputed: (i) Obersteiner believes the initial changes are in the pia through which the dorsal nerve roots pass; (ii) Marie contends that there is a lymphangitis of the post. columns; (iii) Edinger believes the toxins of syphilis attack those nervous structures most in use and therefore most easily fatigued; these are the sensory tracts of the lumbar cord, pupil, bladder and intestine.

Symptoms.—The symptoms vary chiefly as to their intensity, duration and order of succession. In general *three stages* are described. (1) The *initial,* neuralgic or pre-atactic stage, lasting sometimes for years (usually 6 to 8, or even 10 to 20). The earliest symptoms are usually sensory and irritative, as *pains* which are lancinating or rheumatic, perhaps neuralgic or like migraine or paræsthesia. The *knee jerk* is *lost* in nearly all cases, usually very early and the *pupil reacts* to accommodation but *not to light.* Much less often optic atrophy, sexual, sphincter, ocular, articular or visceral symptoms first attract attention. (2) The *atactic stage* may last many years; muscular movements become incoördinate, especially in the legs, but sometimes, though later, in the arms. The tabetic gait develops, the patient sways when his eyes are closed (Romberg's sign), sensation is obtunded, especially the muscular sense, urinary and sexual disorders develop and in some cases diplopia. The gross muscular power is unimpaired. Trophic, joint, bone and skin lesions and visceral crises occur. (3) The *terminal stage,* improperly called paralytic, is much like that of other chronic affections of the cord. The patient is bed-ridden, helpless from extreme ataxia and rarely from motor weakness, and bed-sores, lesions in the medulla and cystitis lead to a fatal issue.

Special Symptoms in Detail.—1. *Motor Symptoms.*—(*a*) *Ataxia* (incoördination) is of such importance that the disease is named *locomotor ataxia.* It is present in 80 per cent. of cases, but may develop slowly, only after years or perhaps not at all; it is less common than formerly (Brissaud); it occurred in barely 10 per cent. of Babinsky's cases, who explains this decrease by his use of mercury. Ataxia is a disturbance of harmonic efficient muscular contraction and arises not only from tabes but also from lesions in the upper sensory neurones of the cord, medulla, cerebellum, pons and cerebrum. The cause of tabetic ataxia is probably disease of the afferent muscular fibers (muscular anæsthesia, Reynolds 1855), intensified by sensory impairment, disturbance in the reflex collaterals and sometimes by atrophy of the optic nerve. Duchenne first made the valuable distinction between ataxia and loss of muscular power which is preserved until late in the disease. Ataxia is frequently first observed by the patient when he walks in the dark or closes his eyes, as in washing the

face. When he closes his eyes or looks well upward or when the heels and toes are brought closely together, the body sways (*Romberg's sign*) and he perhaps falls, because vision is one of the sensory factors in equilibration. Later, incoördination occurs even when the eyes are open, and the patient can walk only with the support of a cane. It is often increased by removing the shoes, which support the feet. The ataxia is marked when the patient attempts to "about face," to walk backward (Althaus), to stand on his tiptoes (Rosenbach) or to stand with the knees half flexed (Brissaud). Ataxia is also seen when the patient, with the eyes closed, attempts to place his heel on the opposite knee or perform with one limb those movements made by the physician in another limb. The gait is atactic, the feet are lifted too high, are thrown too far forward and outward (*wide-legged gait*) and are brought suddenly and forcibly down in a "*stamping*" fashion. It has been called the "rooster gait." A tumbling cerebellar gait is less frequent, observed later in the disease and referable to involvement of the trunk or hips. Movement in tabetics is characterized by excessive excursion, rapidity, irregularity and uncertainty of execution. The arms usually escape or are involved less markedly and later, save in cervical or brachial tabes. The patient cannot execute finer movements, such as writing or buttoning the clothes. Ataxia is rare in the head, eyes, face and tongue, although in exceptional cases it has been observed in masticatory movements. In women marked ataxia is less frequent than simple awkwardness. (*b*) *Muscular power* may be normal even with extreme ataxia. The muscles are hypotonic and this loss of tone allows hyperextension and hyperflexion of the joints; it is due to loss of centripetal sensory conduction. There is a feeling of weakness in the legs in two-thirds of the cases. Attacks of transient muscular weakness are frequent (75 per cent.), as in the eye muscles. Paralysis, though usually transient, may be permanent from disease of the pyramidal tracts, ant. horns or peripheral nerves. (*c*) *Atrophy* is not common, but may affect the arm (type of Duchenne-Aran), the leg (the tabetic club foot, usually due to neuritis), or most rarely the face (hemiatrophy). (*d*) The *electrical reactions* are usually normal. Sometimes muscular contractures occur, due to abnormal sensation. Associated movements, in which one leg moves when its fellow moves, and spontaneous movements, taking place without volition or consciousness of them and due to the slight unnoticed reflexes, may occur.

2. *Sensory Symptoms.*—Sensory symptoms are prominent. In the majority of cases the disease begins with irritative sensory phenomena. (*a*) Spontaneous *pains* occur in 85 per cent., are sudden, paroxysmal, lightning-like, lancinating and often nocturnal. They are usually severe, but may be agonizing, though sometimes only dull, burning or like those of muscular rheumatism; they may be increased by damp and cold. They last from a few hours to a day or two. The legs are the most common seat of pain, then the back, trunk, arms especially in the ulnar distribution and sometimes the head (occipital

nerves) and face (trigeminus, whose ascending branch represents the sensory parts of most of the other cranial nerves); Collet found migraine-like attacks in 40 per cent.; their bilateral occurrence is very suggestive. The pains are more often deep than superficial and are more often irregular than exactly conforming to a nerve's course. The pain in the trunk may assume the "girdle form" (31 per cent.) over a narrow or broad zone. Sometimes the area affected shows local trophic changes, as herpes, ecchymoses or friable hair. (*b*) *Paræsthesiæ* are common, as creeping sensations (formication), "pins and needle" sensations, a sense of heat or especially of cold. They occur in the legs in 66 per cent. of cases. If in the arms, the ulnar distribution is especially involved (17 per cent.). The ulnars at the elbows are often insensitive to pressure (*Biernacki's sign*) and show lessened appreciation of pain, temperature and tactile impressions. In the face a perverted feeling may be noted (*"Hutchinson's mask"*). (*c*) In the early stages hyperæsthesia (probably from nerve irritation) may be noted. In 86 per cent. of developed cases *sensation is reduced;* it is most often of the spinal or segmental type, though sometimes peripheral. *Tactile sensation* is often disturbed, especially in the legs and soles of the feet. Hypæsthesia may be observed on the buttocks, scrotum, perinæum, penis (the cause of early decreased sexual power) or on the trunk as a zone (80 per cent.) which is hypæsthetic especially to light touch (Hitzig and Lähr). It is not usually found higher than the third rib, although there may be occasional occipital or trigeminal anæsthesia. If the arms are affected, it is mostly in the hands and palms. Sometimes a touch to one member is referred to another (allocheiria). Occasionally a single contact feels like several (polyæsthesia). *Perception* of pain is lessened in 50 per cent. of cases, occurring alone or with hypæsthesia; a short stab with a pin is often felt merely as a touch. *Pain conduction* is often retarded (60 per cent.); the sharp stick of a pin is felt at once as a touch, but the pain is perceived only after 7 to 15, even 35 seconds. When asked to state his sensations the patient says "now" when stuck and "ouch" as he feels the pain. Analgesia may be the cause of severe injuries, as in syringomyelia. Sometimes the testicles are insensitive to pressure, and inflammations such as pleurisy may run a painless course. The *sense of temperature* is rarely affected alone or completely and is usually associated with analgesia; tabetics are often oversensitive to cold. The *muscle sense* is disturbed not only as regards the location, but also as to active or passive movements of the muscles, especially of the legs. A change of angle of one or two degrees in the joints and tendons is noted by normal individuals, while in tabetics much wider movements are not perceived, especially those in the small distal joints; the toes may be moved unnoticed by the patient, perhaps in the later stages the fingers also.

3. *Reflexes.*—(*a*) The skin reflexes may be increased early in the disease. Later the plantar and gluteal reflexes are somewhat de-

creased. The abdominal and cremasteric may long remain normal. (*b*) Of the tendon reflexes the *knee jerk* is *lost* early (*Westphal's sign*); this is a most constant finding in tabes (98 to 100 per cent.) and is due to disease of the afferent nerves and collateral reflex paths in the cord. These reflexes should be examined by having the patient sit on the edge of a table with his eyes closed and excluding cerebral inhibition of the reflexes by having him pull, in an outward direction, the locked fingers (Jendrassik's reinforcement). The patellar reflexes are rarely present save in exceptional cases of cervical tabes. They are sometimes simulated by a reflex movement which occurs after tapping the patellar tendon but in this case the interval is longer before the reaction, and the same reaction may follow a simple pin prick. The knee jerk has been known to return after a cerebral hemiplegia. The *Achilles reflex* is often lost, though less frequently than the patellar (or equally frequently, according to Fränkel). The tendon reflexes of the arms are sometimes decreased.

4. *The Eyes and Special Senses.*—(*a*) The *pupils contract to accommodation but not to light* in 85 to 97 per cent. (Argyll-Robertson, 1869). In testing, the patient must look to a distance and one eye must be tested at a time lest accommodation be confused with contraction to light. The cause of the Argyll-Robertson pupil is not known, but it may depend upon a degeneration of fibers connecting the optic nerve with the oculomotor muscles (see PROGRESSIVE PARALYSIS OF INSANE). The tabetic pupil rarely dilates on pinching or sticking the skin of the neck (Erb), especially if the pupil is small. In about 50 per cent. it is small (spinal myosis); the pupils are often unequal in size and irregular in contour. (*b*) The *eye muscles* are affected in 50 per cent. of cases. Möbius holds that ocular paralyses are almost as important as the pupillary changes, especially when they are painless wherein they differ from the rheumatic and syphilitic forms; he holds that the majority of all eye paralyses in the adult are tabetic. Diplopia (in 16 per cent.) is common and usually unilateral. The order of frequency of involvement is the sixth, third (lev. palpebrarum) and fourth nerves. When one muscle is paralyzed the others are usually weak. The early form of paralysis is usually transitory; the late form is often permanent. Nuclear degeneration is the probable cause. (*c*) *Optic atrophy* (10 per cent.) is generally an early symptom. The disk is pale and shrunken, especially on its temporal side and at the edges, which are sharp. The process is always bilateral; central scotoma and actual neuritis are rare. The process usually progresses to blindness. When optic atrophy occurs early, ataxia may not appear; this is known as "tabes arrested by blindness." When it develops later it has no effect on the ataxia. (*d*) *Deafness* may develop gradually or suddenly, sometimes with vertigo or Ménière's complex. It may be permanent or transient. Auditory defects are present in 80 per cent. of cases. Atrophy of the auditory and olfactory nerves has been found.

5. *The Sphincters and Genital Sphere.*—(*a*) The *bladder* is fre-

quently affected (60 per cent.), possibly as the first symptom of the disease. The bladder is slowly or imperfectly evacuated, because its centripetal sensory fibers are diseased. There may be overflow, incontinence and in the later stages cystitis frequently results from decomposition of the residual urine, followed in turn by pyelitis, nephritis and septicæmia. (*b*) The *sphincter ani* is somewhat weak; there is rarely incontinence. Constipation is the rule. (*c*) The *sexual* appetite and power are reduced (66 per cent.) often early. Labor is sometimes painless.

6. *Trophic Disorders.*—The general nutrition sometimes suffers, but local trophic changes are much more common. (*a*) Trophic alterations in the *bones and joints,* described by Cruveilhier and Mitchell, were first thoroughly studied by Charcot, after whom they are named, Charcot's disease. They usually appear late and develop in 2 per cent. of cases. The onset of articular changes is usually gradual, sometimes acute with marked swelling or may seem to be traumatic. Two forms are observed; first and more frequently the atrophic form, especially in the hip and shoulder, in which the cartilages are eroded, the articular surfaces waste and crepitate, foreign bodies sometimes develop and the joint becomes too lax and dislocation of the hip or knee (genu recurvatum) results; second, the hypertrophic form with bony formation, ossification of the ligaments, and rigidity resembling arthritis deformans but more acute, extreme and effusive. The adjacent muscles atrophy—the arthropathic muscular atrophy. The large joints are usually involved, as the knee (45 per cent.), hip (20 per cent.), shoulder (11 per cent.), elbow (5 per cent.) or ankle (4 per cent.), but smaller articulations may be affected, as the tarsus (8 per cent.) or less often the jaw, spine, thumb, etc. The tabetic joint is *painless.* The cause is the same as in syringomyelia; the anæsthetic joints are moved inappropriately and the nutritional reflex, which normally depends on centripetal influences, is lacking (Marinesco and Serieux). The *tabetic foot* is characterized by thickness and roundness of its inner border and arch, flattening of the sole, decreased motility and pathologically by erosion and atrophy of its bones. It must be distinguished from the tabetic club foot due to muscular atrophy of the calf muscles with contractures. The *bones are friable* and break easily but painlessly, as first described by Weir Mitchell. Fracture is due to rarefying osteitis, which renders the medullary substance more porous, the cortex thinner and the inorganic base less in amount. If a callus develops it is often exuberant. The tendons sometimes rupture, probably from trophic changes. (*b*) The *perforating ulcer* of the foot (*mal perforant du pied*) is more frequent. It is "punched out" in appearance, and thickening precedes ulceration; it is usually painless, secretes but little pus and is due to nerve degeneration. Similar ulcerations resembling syphilis may occur in the nose, ear, buccal mucosa, palate or uvula. (*c*) In the *skin,* local sweating, ecchymoses, herpes, blebs, alteration of the hair or nails, gangrene and ichthyosis may occur.

Decubitus is not common save in the last stages, and it may not develop even after years in bed. There may be painless falling out of the sound teeth with analgesia of the gums or dental caries (changes in the fourth ventricle).

7. *Visceral Symptoms.*—The most important are the *painful crises* described by Delmarre but first correctly interpreted by Charcot. (*a*) *Gastric crises* occur in 5 per cent. of cases. There is severe, sometimes excruciating epigastric pain, which often radiates into the back, vomiting (with or without nausea) of food, then fluid, finally bile or even blood; this is often attended by restlessness, epigastric retraction, hiccough, fever and a slow or rapid and irregular pulse. A test meal gives varying results; hyperacidity is less frequent than hypacidity. The urine is alkaline, contains indican and the chlorides are decreased. The crisis lasts several hours to days (even 8 or 10) and may greatly reduce the general nutrition; it recurs and is sometimes the first evidence of tabes. The cause of the crisis is hard to explain; it is usually considered nervous, but Pal thinks it is due to increased arterial tension. (*b*) *Laryngeal crises* are next in frequency but are shorter in duration. They are due to spasm of the laryngeal muscles, and are often dangerous. In some instances only a hoarse cough like that of pertussis is noted; in others there is collapse or death (ictus laryngis). Anatomically, the ascending branch of the fifth nerve and the glossopharyngovagus nucleus or its branches are degenerated. (*c*) Other crises are far less common, as *intestinal* crises, which are characterized by colic, diarrhœa and sometimes rectal tenesmus; the author saw one case with severe constipation lasting 12 days, which was immediately relieved by morphine gr. ½; renal crises with scant urine, transient albuminuria or even hæmaturia, and less often nasal, pharyngeal, hepatic, cardiac, bronchial, vesical, urethral or vulvovaginal crises occur. (*d*) The *pulse* frequently reaches 100. Lachrymation, salivation, polyuria or glycosuria is sometimes noted.

8. *Cerebral Symptoms.*—Cerebral symptoms other than the eye changes noted above, are not frequent; they are unilateral atrophy of the tongue; paralysis of the vocal cords, especially of the abductors; vagus and accessorius symptoms other than those already noted, such as rapid pulse, palatal weakness or rarely paralysis affecting the trapezius and sternomastoid, which is probably neuritic; and very rarely facial or bulbar paralysis.

Complications.—These include hysteria, neurasthenia, Graves' disease, paralysis agitans; true cerebrospinal syphilis; other metasyphilitic maladies, as arteriosclerosis, aneurysm (15 per cent.), valvular disease, arteriosclerotic aortic regurgitation (5 per cent.) and especially *dementia paralytica,* which is an independent although closely correlated affection; myelitis; general or local muscular atrophy, particularly the symmetrical form (Déjérine) due to neuritis; cerebral thrombosis due to concomitant arterial disease; and apoplectiform seizures which occur as in paretic dementia.

Course and Prognosis.—The typical progress of the disease by stages

(*v. s.*) may be altered. According to Sarbó, pain is the first symptom in 68 per cent., while no other single early symptom is the first in more than 3 per cent. The disease in rare cases begins acutely. Not over 50 per cent. are progressive (Gowers). Optic atrophy or insanity may arrest tabes. In the early stages or in rudimentary forms there is a slight chance that the symptoms may become stationary, but not after ataxia fully develops. Tabes is incurable. Leube mentions an instance of a man who worked hard for forty years after the onset of tabes; tabetics may live for twenty years in the last stages of the disease. Crises, especially the gastric crises, alcoholism, traumatism, acute onset, intercurrent affections which may be unnoticed because of their painlessness, and affections of the bladder and kidney with sepsis are unfavorable prognostics. In 1,960 well-observed cases (out of 4,000) 40 per cent. followed a long course with exacerbations, 30 per cent. were steadily progressive, 19 per cent. halted for three years or more, 6 per cent. ran a severe rapid course and 5 per cent. almost recovered (Belugou and Faure, 1903).

Diagnosis.—The diagnosis is usually easily made and is based on the following signs in various combinations:—(*a*) The *Argyll-Robertson pupil,* which, according to Möbius, is indicative of tabes, paretic dementia or rare lesions in the corp. quadrigemina. (*b*) The most common and early symptom, *Westphal's sign,* loss of the patellar reflex (which most rarely fails in health, marasmus, old individuals or as an hereditary lesion). (*c*) *Lightning pains,* "tabetic neuralgia," girdle sensation, *gastric and other crises.* (*d*) *Sensory changes,* subjective and objective, including bladder disturbance. (*e*) *Optic atrophy.* (*f*) *Painless ocular paralysis,* particularly in the adult and of the nuclear form, producing ptosis and diplopia. (*g*) *Ataxia,* which usually does not develop early, with *Romberg's sign* and the *tabetic gait,* is practically pathognomonic after exclusion of peripheral neuritis.

In less than 1 per cent. of cases (Déjérine) the disease is located in the cervical region; in *cervical tabes* the sensory changes, pains and ataxia are most manifest in the arms or even in the face. The arm reflexes are sometimes abolished and the patellars may rarely be normal or increased. The characteristic pupils are present and gastric crises appear early and frequently. The diagnosis of *atypical tabes* may long remain uncertain, as in cases in which ataxia, Westphal's sign and the Argyll-Robertson pupil are absent, or in those with unusual symptoms, as marked motor weakness, atrophy or paralysis.

Differentiation.—In the earliest stage confusion with neurasthenia, hypochondriasis and nosophobia is possible, from common symptoms, as pains, paræsthesia, girdle sensation and weakness of the legs, but close continued observation determines the diagnosis; the tabes of Hippocrates was probably neurasthenia. One of the forms of *peripheral* (*multiple*) *neuritis* (*q. v.*), *viz.,* acute ataxia or pseudotabes, may require differentiation. *Cerebrospinal syphilis,* especially of the roots and the membranes, may sometimes cause confusion.

TABES.	*vs.*	SYPHILIS.
Gradual onset; very slow progress.		More rapid.
Blindness often complete; marked ophthalmoscopic changes. Hemianopsia not common; rarely central scotoma. Argyll-Robertson pupil.		Blindness rarely total; ophthalmoscopic changes may be few or none, yet with great disturbance of function. Amaurosis, etc., acute or subacute, "in installments"; central scotoma with free periphery. No Argyll-Robertson pupil.
Symptoms bilateral.		Often unilateral.
Oculomotor nerve symptoms may cause the greatest difficulty, since the closest relation (Erb) exists between the two diseases as regards this nerve, yet painless in tabes and nuclear in origin.		Oculomotor symptoms with headache (meningitis and nerve trunk involvement).
Crises and anæsthesia of the fifth nerve, laryngeal paralysis.		Crises, anæsthesia of the fifth nerve, etc., rare. Anæsthesia of the fifth is for all varieties of sensation, while tabetic anæsthesia is for pain.
Sensory nerves most involved.		Motor nerves mostly.
Paralysis very rare.		Paralysis frequent at some period.
Knee jerk absent.		Knee jerk variable, increased.
Ataxia.		Rare.
In syphilis and parasyphilitic disease (tabes), lumbar puncture shows predominance of mononuclear cells.		Remissions frequent with greater variability and brusqueness than in tabes.

Tabes and *paretic dementia* have a common etiology but in the latter the psychical alteration is characteristic. For differentiation from *Friedreich's ataxia,* see page 1072. In *cerebellar tumor,* the ataxia usually ceases when the eyes are closed and is not characterized by the excessive excursion or rapidity of tabes; in most cases the patellar reflexes are increased, there is no anæsthesia and signs of tumor are often present. In *multiple sclerosis* there may in exceptional cases be crises, pains and sensory disturbance, but the Argyll-Robertson pupil, ataxia, marked sensory and bladder disturbances are most rare. The reflexes are increased, there are nystagmus, scanning speech, intention tremor and central scotoma and functional oculomotor involvement is more frequent. *Paraplegias* are not difficult to differentiate, since true motor disturbance is rare or late in tabes; the reflexes are increased and spastic rigidity is marked, even in atactic paraplegia. *Tabetic pain* is often misinterpreted; pain as a rule suggests disease of the vertebræ and membranes rather than of the cord itself, but in vertebral disease or cord tumor the pain is fixed, and is often increased on pressure and movement. Tabes may be confused (but only on hasty or partial examinations) with *rheumatism,* intercostal or trigeminal neuralgia, and gastric, renal or other *visceral disease,*

Bilateral sciatica always suggests tabes, diabetes or nephritis. Strümpell describes, among workers in tobacco, a *nicotine tabes,* in which pain, pupillary alteration, atactic gait and absence of knee jerks are observed, but which presents a characteristic tremor and increased cutaneous reflexes in the legs.

Treatment.—Since the disease is incurable, no therapy is especially efficacious.

1. Etiological Treatment.—Antisyphilitic remedies are at first indicated, but their frequent and almost regular failure has led some writers to erroneously deny the causal connection between syphilis and tabes. Tabes is not active syphilis; it is a late degenerative parasyphilitic affection. Mercury and iodides are therefore advantageous chiefly in early cases or when gummatous changes co-exist with tabes. They apparently arrest some cases of optic atrophy. Mercury is thought by Erb to be beneficial if it is used carefully; it should be discontinued at once if untoward symptoms develop.

2. Hygienic Treatment.—Overwork, overexercise, worry, fatigue, trauma, exposure and excess in tobacco, alcohol or sexual indulgence should be avoided. Tabetics should not marry. The diet should be regulated, since dyspepsia promotes pain. If possible, the patient should live in a warm climate and at a moderate altitude.

3. Drug Treatment.—There is no specific remedy. Silver, strychnia and nitroglycerine give uncertain results. Gowers considers arsenic beneficial.

4. Balneotherapy.—Warm baths are injurious; lukewarm baths are best, but exert no specific influence on the disease. The same is true of (5) *electrotherapy* and (6) *massage.*

7. Symptomatic Therapy.—(*a*) If *pain* is superficial, sinapisms, chloroform liniments, cocaine (hypodermatically) or the galvanocautery should be tried; if moderate, acetanilide or cannabis indica, is beneficial, but morphia should be avoided as long as possible, because the habit is readily acquired. Peripheral faradization is often efficacious. Gowers recently described cases in which pain was a salient symptom and existed without ataxia or loss of the patellar reflexes; for this "tabetic neuralgia," differing from the ordinary pains of tabes, he recommends salicylates and aluminium chloride, gr. v–x t. i. d. (*b*) *Crises* resist milder therapy and usually necessitate the use of morphine hypodermics. Laryngeal crises often respond to nitroglycerine hypodermatically, chloroform by inhalation or cocaine locally. Stretching the spine and cord probably relieves pain; the patient sits on the floor with the legs extended; without other movement, the body is carefully pushed forward as far as possible. (*c*) For *vesical* affections, catheterization for residual urine is indicated, but under most rigid antisepsis; belladonna and faradization are often helpful. Cystitis is treated by lavage. (*d*) Secondary infection should be avoided. Corns should never be cut but always rubbed off.

8. Ataxia.—This may be relieved by H. G. Fränkel's systematic exercises, "a reëducation of the central nervous system by repeated

exercises," which teaches that carefully and persistently executed muscular movements, not forceful movements, are desirable. (The nerve stretching of Langenbuch and the suspension method of Mitchell are useless and even injurious.)

II. System Diseases of the Motor Tract.

The centrifugal or motor paths may be diseased (1) in the upper neurone, as in spastic spinal paralysis; (2) in both neurones, as amyotrophic lateral sclerosis; (3) in the peripheral neurone, as spinal muscular atrophy or its cerebral analogue, progressive bulbar paralysis; (4) in the peripheral motor nerve, as the neuritic muscular atrophy and finally (5) in the muscles themselves, as the muscular dystrophies. These forms are more or less correlated and some types blend, but separate descriptions are necessary to bring out the classical symptoms and physiology (the classification into those with and without disease of the spinal gray matter is more simple).

1. Spastic Spinal Paraplegia.—Lateral sclerosis, *tabes dorsal spasmodique,* was described first by Erb in 1875 and later by Türck and Charcot. While amyotrophic lateral sclerosis affects both neurones and the muscular atrophies the lower neurone in the cord, nerves or muscles, this malady concerns only the upper motor neurone, especially that part which is in the cord, although it has been traced at times to the cortex.

Etiology and Pathology.—The disease represents a clinical symptom complex rather than a pathological entity. The causal factors are uncertain, as syphilis, or occasionally an hereditary tendency. In 75 per cent. of cases it occurs between the twentieth and fortieth years. It was thought that there was a primary sclerosis of the lateral pyramidal tracts, especially in their lower parts, for which unequivocal confirmation is still lacking, for the cases of Erb, Charcot, Strümpell, Dreschfeld and Minkowski were not wholly confined to these tracts and there were some changes in other neurones, as in the post. columns.

Symptoms.—There are *three cardinal findings.* Motor weakness, which is usually paraplegic; muscular rigidity and increased reflexes. (*a*) *Motor weakness* is usually the first sign; it begins slowly and is evidenced by weakness, fatigue or stiffness in the flexors of the hips especially but also the knees and ankles. Every gradation from paresis to paralysis may be noted in different cases or in different stages. The arms are rarely first affected. Unilateral paresis, as of one arm and one leg, is very exceptional. Strümpell attributes the loss of finer synergistic movements to involvement of the lateral pyramidal tracts; he holds that the coarser movements for groups of muscles are possible, *via* the anterior pyramidal tracts. The muscles are large and well nourished. (*b*) *Muscular rigidity* (hypertonicity) results also from disease of the lat. pyram. tracts, which lessens the inhibition and is proportionate to the loss of power. The muscles

resist passive efforts at flexion, especially quick efforts, but yield to slow, gradual flexion and remain in the position given them; this is the "*lead pipe contraction*" of Weir Mitchell. The knees are rigidly extended, even when the patient sits on the edge of the bed; the great toe is strongly flexed dorsally; when one leg is lifted as the patient lies in bed the other comes with it; the specially tense adductors prevent abduction of the thighs. The muscles are stiff in the morning; painful spasms may occur during the night; hypertonicity may lead to contractures which are largely active in character and patients sometimes complain of the "tendons getting too short." In active movements rigidity is manifest; the gait is paretic and spastic, the knees are extended and closely apposed, the steps are short, the balls of the feet seem to cleave to the ground and the feet scuffle. (*c*) *Increase of the reflexes* is also due to lateral pyramidal sclerosis. The *knee jerks* are most *excessive;* ankle clonus and Babinsky's reflex are present; a strong clonic contraction of the quadriceps follows depression of the patella and the gait may be hopping; in short, this is the so-called "spinal epilepsy" (Brown-Séquard). The *arms* are often normal but may show weakness; there may be rigidity like that of a late hemiplegia, although less in degree, in which the arms hug the chest, the forearm is pronated and partly flexed and the fingers are flexed; there may be clonic spasm of the fingers and increased tendon and periosteal reflexes. In the *trunk* there sometimes are weakness in the back and painful contractions of the chest and back muscles with dull aching. The cranial nerves are rarely diseased, although the jaw-jerk may be increased. Sensation, the electrical reactions, the muscular nutrition and the eyes are usually normal. If there are sensory symptoms they are rarely objective; the sphincters are usually affected late.

Clinical Forms and Diagnosis.—Diagnosis concerns (*a*) the paresis, (*b*) hypertonicity, (*c*) increase in the reflexes, (*d*) gradual onset and (*e*) absence of sensory, trophic and other symptoms. Six clinical forms may be described. (1) There are rigidity and spasticity in the legs, arms and perhaps the face, sometimes with compulsory laughing or weeping, as in amyotrophic lateral sclerosis, but without muscular atrophy.

2. The *hereditary or familial form* (Erb, Bernhardt, Strümpell, Spiller) commences in persons between twenty and thirty years of age; the paresis develops late; this form is characterized anatomically by sclerosis of the (congenitally weak) lateral pyramidal tracts and also by sclerosis of the cerebellar and Goll's columns (more properly classified under combined system disease).

3. In *infantile forms* (see Cerebral Paralysis in Children), the rigidity is less; foot-clonus less common; tonic spasms are rare; athetoid movements, deficient mentality and epileptiform convulsions are common.

4. In the *syphilitic form of Erb* there are paresis and slight spasticity, plus urinary incontinence, retention and impotence.

5. There are *forms which are symptomatic of brain disease* (tumor, syphilis, paretic dementia, encephalitis, hydrocephalus), *cord disease* (tumor, myelitis, compression, syringomyelia, multiple sclerosis) and *hysteria* (which may closely simulate spastic paraplegia); hysteria has not the peculiar extensor spasm, loss of power is rarely complete and ankle clonus is rare, although a spurious half voluntary contraction of the calf muscles may be noted.

6. Westphal, Charcot, Schachnowitch, but especially Goldflam, have described *periodic familial paralysis,* which affects the legs and less often the arms, is flaccid and results in decrease of the patellar and absence of the plantar reflexes, decreased faradic irritability, sleepiness, but without sensory symptoms; it lasts one to three days. The prognosis is good.

Prognosis.—The disease may be stationary for years and its course is chronic. "It is perhaps the least dangerous to life of any chronic spinal lesion." Recovery or arrest is rare.

Treatment.—Much exercise, fatigue, electrotherapy and strychnia are to be avoided. Massage and warm baths relieve the spasticity. Otherwise the therapy is expectant.

2. Amyotrophic Lateral Sclerosis.—This was often called the *Maladie de Charcot,* because Charcot (1865) with Joffroy first described it.

Etiology.—It occurs mostly between the twenty-fifth and forty-fifth years, more frequently in males, but its exact causes are not known, although cold, trauma and overexertion are possible factors.

Pathology.—Changes may be found from the cerebral cortex to the muscles themselves, but the name amyotrophic lateral sclerosis, left to us by Charcot, designates the leading changes, which are, *amyotrophy,* muscular wasting, from atrophy of the anterior horns,—and *sclerosis of the lateral columns.* (*a*) The *lateral pyramidal tracts,* and to a lesser extent the anterior, show symmetrical primary degeneration of the nerve fibers, followed by interstitial overgrowth (secondary sclerosis) which is most marked in the cord, *i. e.,* the lower part of the upper neurone, but extends upward in some cases to the medulla, pons, crus or even to the very central convolutions, the ganglionic cells of which may be atrophied. Charcot's belief that the disease starts in the pyramidal tracts and is followed by changes in the anterior horns (*v. i.*) is not proven. In some cases similar but unimportant changes may be found in the cerebellar tracts or indeed in the posterior columns. (*b*) The *ganglionic cells of the anterior horn* are degenerated, so that the anterior motor part of the cord is distinctly smaller and flatter, particularly in the cervical region. Likewise the nerve fibers from the pyramidal fibers, anastomosing with the processes of the ant. horn, are degenerated. With these changes in the trophic centre of the second motor neurone the atrophic changes in the anterior roots, motor nerves and muscles are easily explained. Certain muscles are wasted to a varying degree and show

degeneration or complete atrophy, as in the purely spinal progressive muscular atrophy, next to be described. (*c*) The *bulbar nuclei,* with their nerves and muscles, show identical changes, for they are the analogues of the ant. horns.

Symptoms.—The symptoms, like the lesions, are strictly motor.

Stage 1 of Charcot.—The disease begins with weakness and atrophy of the *muscles* of the hand, first in one and then in the other; they are most marked in the thenar, hypothenar and interosseal muscles, and are followed by wasting and weakness in the *forearm extensors, deltoid* and *triceps,* while other muscles of the arm and forearm escape or are involved later. Sensory symptoms are absent. The decreased power of unwasted muscles is often manifest before they atrophy. Other symptoms of disease of the lower neurone appear, as *fibrillary contractions;* the *reaction of degeneration* is present in those muscles which are wholly atrophied, while in others the response is reduced proportionately to the atrophy; the nerve trunks respond normally. Evidences of involvement of the upper neurone also appear; paralysis often precedes the wasting and accentuates the weakness from ant. horn involvement; there is *increase of the reflexes* (which never occurs in muscular atrophy due to disease of the ant. horns alone), increased biceps, triceps and periosteal forearm reflexes and there are often *rigidity and contracture* in which the arm hugs the chest, the forearm is flexed and pronated, and the wrist and fingers are flexed. Sometimes the neck is rigid and even trismus is observed.

Stage 2.—In less than a year the *legs are involved;* indeed they show symptoms during the first stage. The legs are weak, especially in the flexors of the hip, knee and dorsal flexors of the foot. Spastic symptoms prevail. The legs resist passive movement, because of increased muscle tonus and the increased reflexes. The *gait is spastic* and *paretic* (see above, Spastic Spinal Paralysis) and walking finally becomes impossible because of the rigidity and the *twitching tetanic movements* of the *extensors.* The *patellar reflexes* are *exaggerated;* ankle clonus is frequent; when the patient attempts to flex the thigh while the examiner holds the ankle, there is a visible contraction of the tibialis anticus (the tibialis phenomenon); *Babinsky's reflex* is present (dorsal instead of plantar flexion of the great toe on stroking the sole); there are evidences, in short, of pyramidal disease, which dominate leg symptoms, because symptoms referable to the lower neurone, as atrophy, are much less frequent, less marked and less early in the legs than in the arms. In exceptional cases the leg symptoms develop first.

Stage 3.—This stage occurs in one or two years with bulbar symptoms, dysphagia, atrophy of the lips and tongue with fibrillation, as in primary bulbar paralysis (*q. v.*). On tapping the jaw a marked jerk is elicited, analogous to the increased reflexes in the arm and leg. A tonic tension of the facial muscles may occur, as in spastic spinal paralysis. Spasmodic laughing or weeping almost always occurs and is due to loss of inhibitory innervation. The mind is clear. Death

results from respiratory failure, aspiration pneumonia or intercurrent disease. In rare cases the bulbar symptoms antedate atrophy in the arms and spasticity in the legs.

Course and Prognosis.—In some cases the spinal, in others the atrophic, symptoms develop first and are dominant. The entire course is rapid compared with other diseases of the group, covers one to four years and is always fatal.

Diagnosis.—The involvement of both motor segments, muscle atrophy with increased reflexes, rigidity, absence usually of sphincter or sensory alteration (except rarely some pains) and the final bulbar symptoms make the disease easy of diagnosis.

Treatment.—The treatment is that of spinal muscular atrophy (*v. i.*), but avails little.

3. Progressive Spinal Muscular Atrophy.—Duchenne and Aran (1849–50) gave us the first full clinical description of this disease, while Clarke, Luys (1860) and then Charcot, discovered changes in the anterior horns.

Etiology.—The ultimate causes are not known. Seventy-five per cent. of cases occur in males, mostly between the twenty-fifth and forty-fifth years. Hereditary factors, which are often observed in the muscular dystrophies (*v. i.*) are infrequent, although Werding (1891), Hoffman (1894), Bruce, Osler and Thompson have described familial infantile forms. Hard work, mental distress, syphilis, trauma, acute infections and acute poliomyelitis are possible factors.

Pathology.—In the cord, especially in the cervical region, (*a*) *the ant. horns are small and flattened;* their ganglion cells are shrunken, deformed, their processes lost; they are wholly wasted in advanced cases. Any interstitial changes are wholly secondary. (*b*) The *anterior roots*, especially the cervical, are somewhat *degenerated* and often also the peripheral nerves, although changes in them are most difficult to distinguish. (*c*) The *muscles are small, stringy* and pale, having lost their normal redness. Microscopically their individual fibers are small, although a few normal or even hypertrophic fibers may be seen. Their transverse striation persists remarkably, and waxy, fatty or granular degeneration and longitudinal splitting are not common; sometimes the empty sheaths alone are seen. The muscle nuclei multiply, round cells may wander in, and there is usually increase of connective tissue and sometimes fatty deposit. Just where the process begins is disputed. No close distinction can be drawn between this disease and amyotrophic lateral sclerosis, although changes in the upper neurone are absent in spinal muscular atrophy. Gowers and v. Leyden consider them identical; Kahler and Pick held that both these diseases and bulbar paralysis are one affection.

Symptoms.—(*a*) The essential symptoms are those of the motor nerves and all others are accessory. The earliest symptom is *muscular atrophy* with *weakness* in *the upper extremity* (90 per cent.),

especially in the muscles of the (right) *thenar* and *hypothenar eminences.* The abductor and opponens pollicis are first involved, then the flexor brevis and adductor, the wasting of which brings closer together the metacarpi of the thumb and index finger (the "*ape hand*"). It is finally impossible to appose the thumb to the fingers. The interossei on the dorsum waste and leave marked furrows, producing the "claw hand," *main en griffe,* from contraction of antagonist muscles, the ext. digitorum communis, the tendons of which stand out prominently. The wasting lumbricales make the palm more hollow. Wasting as above described is known as the Duchenne-Aran type, in which "individual muscles" are selectively involved (Charcot). The onset and progress are always very gradual and the muscles become lax, soft, thin and flat. The atrophy long exceeds the paresis. The *forearm* may be next involved, first the extensors, especially those below the int. condyle and later the supinators and flexors. A deep depression between the ulna and radius eventually occurs. Later, often after several years, the *shoulder* muscles, the posterior and middle third of the deltoid, the biceps and brachialis waste and much later the triceps. Sometimes the wasting of the muscles of the shoulder and arm immediately follows the involvement of the hand, or perhaps the disease begins in the shoulder. The bones of the shoulder stand out clearly, covered only by the skin; these cases are "the living skeletons" seen in museums. The trunk muscles are involved in this order; trapezius in its middle and lower parts (rarely in its upper third, which is the *ultimum moriens* of Duchenne); pectoralis, latissimus dorsi, rhomboidei (causing the shoulder to droop) and extensors of the head (allowing the head to fall forward). Scoliosis may develop, lordosis is common in involvement of the extensors of the trunk and hip and the spine stands out distinctly in cases of extreme atrophy. The platysma and face almost always escape. In most instances the respiratory muscles, intercostals or even the diaphragm are involved, and this is a not infrequent source of danger to life. The abdominal muscles are sometimes weakened. In the leg the muscles are affected late and to a lesser degree, as the glutei, quadriceps and the peronei. In very exceptional cases the disease first develops in them. In the fully developed case there is extreme helplessness in which an added misery in the form of the sister-process, bulbar paralysis, may develop.

(*b*) In the wasted and weak muscles there are usually *fibrillary contractions;* they are involuntary twitchings, tremor- or even wave-like in character, which are sometimes incessant, and again are initiated only by cold or tapping; they show increased mechanical irritability. They may appear in muscles not yet wasted and are not to be confused with the similar neurasthenic fibrillation. (*c*) The *electrical reaction* depends on the degree of wasting. In the nerves it is normal. In the muscles there is usually only quantitative reduction and the voltaic excitability remains long after the faradic is lost; in the last stages there is a partial reaction of degeneration. (*d*)

The *reflexes* are *weakened* or abolished; they are roughly proportionate to the atrophy and due to disease in the motor side of the reflex arc. They are never increased. The sphincters are intact, but sexual power is often lost. (*e*) *Sensation* shows no objective change, and even subjective paræsthesia is rare; in some instances there are dull aching pains in the muscles or joints. (*f*) Vasomotor, secretory and trophic symptoms are lacking. The coolness of wasted parts is due to lack of motility.

Diagnosis.—The diagnosis is based on (*a*) the slow course; (*b*) the involvement of the muscles of the hand and its slow "individualization"; (*c*) fibrillation; (*d*) partial reduction in electrical irritability or partial reaction of degeneration; (*e*) absence of increased reflexes and (*f*) of any sensory signs.

Differentiation is necessary (1) from disease in which muscular atrophy is but one among several symptoms. (*a*) *Syringomyelia* is marked by its trophic characteristics, its analgesia, thermo-anæsthesia and less symmetrical wasting. (*b*) *Meningitis,* pachymeningitis, spondylitis, tumors and myelitis are manifested by anæsthesia, pain, tenderness and rigidity. (*c*) In *root neuritis* or *multiple neuritis* there is a clearer reaction of degeneration, more sensory signs and more tenderness in the nerve trunks. (*d*) The *occupation atrophies* are distinguished by the history and slight sensory disturbance.

2. *Other muscular atrophies.* (*a*) Subacute and *chronic poliomyelitis* causes paralysis first and atrophy afterward; it affects groups of muscles at once, its course is not gradual but rapid and fibrillary contractions are infrequent. (*b*) The *myopathic atrophies* (see p. 1070). (*c*) *Amyotrophic lateral sclerosis* is an allied, perhaps an identical affection; Charcot held that the atrophy is not "individualized," but occurs *en masse;* the distinguishing features are the increase of the reflexes, spasticity and a much more rapid course. Gowers has never seen a case of progressive spinal atrophy without anatomical change in the pyramidal tracts; in describing the two affections together, he distinguishes three varieties (i) extreme atonic atrophy, (ii) muscular spasm and weakness with little or no atrophy and (iii) moderate tonic atrophy with increased myotatic irritability. (*d*) *Arthritic muscular atrophy,* probably due to reflex influences, or direct extension of inflammation to the muscles or nerves, may be acute or chronic and involves chiefly the extensors in a moderate wasting; diffuse wasting suggests a nervous cause; the history or evidence of joint disease is distinctive. The electrical reactions are about normal. There is usually distinct increase in myotatic irritability and reflex action.

Course and Prognosis.—The onset between the twenty-fifth and forty-fifth years is gradual, the course is slowly progressive, remissions infrequent, arrest very rare and death usually results from intercurrent disease, respiratory affections (tuberculosis) or bulbar palsy. Gowers observed arrest but rarely and then chiefly in cases

marked by symmetry and synchronous involvement of both hands; arrest occurs, unhappily, chiefly late in the disease.

Treatment.—Drugs are of uncertain value. Gowers gives strychnia hypodermatically; he has seen 7 consecutive cases of arrest of which 6 were permanent; it has little effect by mouth. Arsenic and iodides are employed. Massage, active and passive gymnastics and electricity are always indicated but do not stay the disease.

4. Neuritic Muscular Atrophy.—Charcot-Marie type. Peroneal family type (Tooth). Neuritic type (Hoffmann).

Etiology.—It is often hereditary, even through five generations; it is usually familial. It occurs in the second half of childhood and rarely after the twentieth year. Sixty-six per cent. of cases are in males.

Pathology and Symptoms.—The pathological characteristic is *neuritis,* which Hoffman thought might ascend to the post. roots; secondary changes have been seen in Goll's column. Early atrophy occurs in the *small muscles of the feet* and may not attract attention until bilateral symmetrical peroneal paralysis and atrophy develop, together with paralysis in the long toe extensors, and club feet (*pes equinus* or *varo-equinus*). The calf muscles are the next to suffer and the thigh is involved still later. Then after years, paralysis and atrophy of the hands, as in progressive spinal atrophy, develop, with the "claw hand"; in involvement of the forearm, the extensors suffer more than the flexors. The shoulder, neck, trunk and face are seldom diseased. Fibrillation is infrequent; the reaction of degeneration is generally present, sensory manifestations are common, as pains or anæsthesia (especially plantar) and reflex action is decreased or abolished, though the patellar reflexes may long persist.

Diagnosis and Treatment.—The *diagnosis* is based on the hereditary or family history, the age, localization and signs of slow neuritis. *Treatment,* as in other forms of muscular atrophy, is unsuccessful.

5. Muscular Dystrophy.—The progressive muscular dystrophies (Erb) or myopathies (Charcot) are often classed with diseases of the muscles, which indeed show the sole constant pathological lesions. They are so closely related clinically to the less frequent spinal atrophies that they are here considered. Their status is provisional, since changes in the cord, nerve trunks and nerve filaments have been found in some cases of each type. In all types (*v. i.*), with few exceptions, there are certain common characteristics; (*a*) *heredity* is an obvious cause in 56 per cent. of cases; though this factor may be absent, a familial incidence is often observed; the muscular system seems deficient in nutrition, development or vitality. (*b*) It usually occurs in *childhood* or pubescence, rarely in adult life. (*c*) The *pathology* is practically the same in all types; the *muscles waste,* with or without hypertrophy or with increase of fat or interstitial growth (*d*) Being strictly a muscular affection, fibrillation and the reaction of degeneration are very rare. (*e*) In localization, certain muscles

are involved, as those of the face (orbicularis oculi et oris), shoulder (pectoralis, latiss. dorsi, serrat. magnus, rhomboidei, trapezius), arm (biceps, brachialis, supin. long., triceps), trunk (erector spinæ), glutei and thigh. Others are usually exempt, as the deltoid, supra- and infraspinatus, small muscles of the hands and feet, leg muscles (gastrocnemius) and those of the tongue and pharynx. There are three forms.

Pseudohypertrophic Muscular Paralysis.—Lipomatous muscular atrophy is the least frequent, but is the longest and best known type (Bell 1830, Partridge 1837, Meyron 1852, Duchenne 1861). It occurs in males (80 per cent.) and is less marked, less early in life and less fatal in women. It is often transmitted by healthy mothers, the males being impotent, and may be seen in five generations. It begins early (75 per cent. under ten years) and but rarely after twenty years of age.

FIG. 75.

Typical pseudohypertrophic muscular paralysis. (Dercum.)

1. *Impaired motility* is the earliest symptom. The weakest muscles are the invisible hip flexors. Weakness of the hip and knee extensors makes climbing of stairs difficult, causes the waddling gait (paresis of glutei) and interferes with rising from the floor, so that the child must rise by resting on all fours and then by "climbing his own legs with his hands" (Gowers). Paresis of the back muscles and hip extensors causes lumbar lordosis and abdominal protrusion. The ankle extensors also weaken. The child stands with legs far apart. The depressors of the shoulders are weakened early in the disease, later the shoulder muscles, then the biceps and triceps, but the hand remains normal for a long time, or is permanently unaffected. The weak muscles show nuclear increase, atrophy and division of their fibers, which are usually undersized, irregular in width, pale and narrow; fatty degeneration and loss of striation are rare. Increase of connective tissue is a later but most characteristic change.

2. Certain muscles show apparent increase in size (*pseudohypertrophy*),

which is due largely to early adipose deposit or rarely to actual muscular hypertrophy. The muscles are lax. The calf muscles are especially enlarged, then the glutei, knee extensors, or infraspinatus which contrasts strongly with the wasted latissimus and deltoid; the muscles of the face, neck and forearm seldom show enlargement.

3. As to *other symptoms,* the electrical irritability is lowered, but there is no reaction of degeneration. The deep reflexes decrease and disappear, from the muscular lesion. Mentality, sensation and the sphincters are normal. Contractures, such as the *pes equinus,* may occur late in the course. Cardiac hypertrophy was observed by Glotz in 60 per cent. of his cases. The course is progressive, the patient becomes bed-ridden, arrest is rare and death occurs after years from intercurrent disease or from respiratory inflammation.

Infantile Atrophic Form, with or without Facial Involvement.—There is usually no hypertrophy, but wasting occurs from the beginning. It may be combined with the first form, and the legs then show pseudohypertrophy and the arms simple atrophy. The pathology is that of pseudohypertrophic muscular paralysis, save that there is no hypertrophy, true or false; increased connective tissue characterizes this type of atrophy and also that of the juvenile form (see below).

The *face* may be involved, as was first observed by Duchenne. The lids droop and cannot be wholly closed, the forehead is smooth, the cheeks are flaccid and droop, the lips part and functionate poorly, as in whistling, the lower lip protrudes, the nasolabial fold disappears, the face becomes expressionless (the "*facies myopathica*"). Sometimes the buccinator muscles are implicated but rarely the masseters, tongue, palate, larynx, pharynx or eyeball. Simultaneously or more often later, the shoulder girdle wastes as in the first type; this is the *facio-scapulo-humeral* form of Landouzy and Déjérine; the latissimus and lower part of the pectoralis are usually first affected, but in the biceps, triceps and often in the supinator longus, the symmetrical wasting and paresis are more conspicuous. The forearm, save for the supinator longus, and the hand usually escape. The neck is seemingly lengthened (Brissaud), because of the drooping of the shoulders and clavicles. The leg muscles then waste, especially the hip flexors, knee extensors and less often the glutei; lordosis, the waddling gait and symptoms observed in the next type (*v. i.*) may develop. The order given may be altered, for facial wasting may follow that of the arms. The course is usually progressive, very chronic (ten to fifty years) and the disease in itself rarely causes death, which results from intercurrent infections, respiratory catarrh, tuberculosis, etc. In the second and third types the prognosis is more favorable than in the first form and the outlook is better when the face is unaffected.

The Juvenile Form of Erb.—This type begins after puberty, possibly later (twentieth to fortieth year) and is more frequent in women than the above forms. It begins more often in the shoulders and arms than in the legs. The pectoralis, latissimus, trapezius, serratus

mag., rhomboidei, sacrolumbalis, longiss. dorsi, supinator longus and later the triceps are wasted, while the sternomastoid, supra- and infraspinatus, lev. anguli scapulæ, coracobrachialis, teretes, deltoid and the hand and forearm (except the supin. long.) are almost always exempt. If we lift the patient with our hands beneath his shoulders he tends to "slip through"; this is due to "loose shoulders" (Erb). The scapulæ stand out prominently from weakness of the serrati. In the lower limbs the glutei, quadriceps and less often tibialis ant. and peronei become atrophied. The lordosis and gait are as above described.

Diagnosis of Myopathic Atrophies.—(*a*) *Atrophy* from *cerebral lesions* is less marked than myopathic atrophy and more often follows the marked loss of power. (*b*) *Spinal muscular atrophy* (according to Leube) is differentiated below.

(*c*) *Congenital spastic paraplegia* occurs in children with weak legs and contractures in the calves but the history, increased reflexes and a regressive rather than a progressive tendency easily distinguish it. (*d*) As to *peripheral neuritis,* the hereditary neurotic (neuritic) muscular atrophy (*q. v.*) is easily recognized. Multiple neuritis (*q. v.*) is usually toxic, more acute and shows sensory symptoms and local tenderness over the nerves and muscles. (*e*) In *congenital defects* in the pectoral muscles and trapezius, any progression is naturally absent.

Spinal Progressive Muscular Atrophy.	*vs.*	Myopathic Progressive Muscular Dystrophy.
Begins in small hand muscles.		In trunk, shoulder or pelvic girdle, leg and upper arm.
Extends upward (face free).		Progresses downward; face sometimes affected.
Muscular atrophy.		Frequently with hypertrophy.
Fibrillation the rule.		Very exceptional.
Tendon reflexes may be increased in the legs, if we include the amyotrophic lat. sclerosis.		Never increased.
Reaction of degeneration, partial, perhaps complete.		Practically never present.
Age; usually begins after thirtieth year.		Usually in childhood; rarely after twentieth year.
Heredity—rare.		Frequent.
Bulbar symptoms not uncommon.		Never present.

Treatment.—Electricity is recommended, but a current sufficiently strong to excite the wasted muscles causes too much pain. Massage, but especially reasonable exercise, is advisable, because the patients begin to fail when they cannot walk. In advanced cases the respiratory tract must be watched, since catarrh predisposes to tuberculosis.

Under diseases of the motor neurones, ophthalmoplegia, Landry's paralysis, bulbar palsy, myasthenia, acute poliomyelitis, etc., might also be described; these affections, however, are treated elsewhere for anatomical or differential reasons.

III. Combined System Diseases.

In 1867 Westphal described these affections in paretic dementia, but Kahler and Peck in 1877 brought to more general attention this group of cases with simultaneous disease in several systems of neurones. Von Leyden and Goldscheider consider them diffuse or even myelitic affections rather than true system diseases, of which, in their opinion, there are but two examples, tabes and progressive muscular atrophy.

1. Hereditary Ataxia, Friedreich's Ataxia (Friedreich, 1861).—**Etiology.**—This affection is hereditary in 33 per cent. and in the other instances it is usually familial, although isolated cases are seen. Most cases occur in boys. It is held by some to be a congenital hypoplasia of the pyramidal and other tracts, tending to degeneration (Friedreich, Schultze, Strümpell), though others regard it as a combined system disease (Kahler, Pick). Cerebellar atrophy has been held as causal (Senator, Mentzel) but this is more striking in the hereditary cerebellar ataxia, described below.

Pathology.—The cord or medulla and even the pons are small. The earliest and dominant change is atrophy with secondary *sclerosis of the post. roots and post. columns,* and in Goll's more than in Burdach's columns. Lissauer's "root zone" is less affected than in tabes, but more than in ataxic paraplegia. The *direct cerebellar tract* and Clarke's gray columns share clearly in the atrophy, as does the ant. lat. ascending tract of Gowers. The *lat. pyramidal tract* suffers in most cases, its inner portion being usually normal; the ant. pyramid. tract is less altered and principally in the cervical region. Chronic leptomeningitis, sometimes with annular sclerosis of the cord, is occasional.

Symptoms.—(*a*) The *cardinal sign and the one first observed is ataxia,* which is less rapid, excessive and stamping (as to gait) than tabetic ataxia, and more closely resembles cerebellar ataxia in its tumbling character and involvement of the trunk. Static ataxia appears later but to a marked degree, and the head oscillates as it does in multiple sclerosis or the arms show jerky movements. *Romberg's sign* is usually present and the subject leans over to watch his feet; this leads to some kyphosis. Testing the sense of location elicits ataxia, as in the knee-ankle test. The arms become atactic after a few years. *Nystagmus* develops in most cases, though it is a late symptom; it is elicited chiefly on lateral (or upward) movements and is atactic, as is also the *disturbance* of speech, which develops in three to ten years; the speech is monotonous, irregular, unclear,

slightly scanning, slow and strained; syllables are often elided, the tongue may tremble and a nasal twang may be noticed.

(*b*) Regarding the *reflexes,* the *knee jerks* are decreased early or are *abolished,* being present only in rare atypical cases. The skin, pupillary and sphincter reflexes are usually normal. In some (possibly syphilitic) cases the pupil reflex may disappear. (*c*) *Other signs: Club foot* (pes equino-varus) is common and is best seen in the dorsal decubitus; the foot is shortened and widened, the arch is exaggerated (pes cavus), the great toe is strongly extended dorsally. Scoliosis and kyphosis are common. Muscular paralysis or wasting is rare and late. Sensory signs are few; tactile and muscle sensation is sometimes blunted; the pain sense is but rarely impaired; paræsthesiæ, crises, lancinating pains and trophic, vasomotor and sensory disturbances are lacking. General or sexual development may be retarded at puberty. The pulse is often increased and hæmic murmurs may be heard. Cerebral symptoms, as mental dulness, develop late, if at all.

Course and Prognosis.—About 33 per cent. develop before the sixth year, 33 per cent. between the sixth and tenth and 33 per cent. after the tenth year. The atactic gait is the first sign, then speech is involved, possibly then the kyphosis and club foot develop, and later the arms are affected; the course is steadily progressive, possibly with remissions, and the patient becomes bed-ridden in twenty to thirty years. Death follows from acute infections, cystitis and sepsis.

Diagnosis.—The diagnosis is based (*a*) on the hereditary or familial etiology; (*b*) the age; (*c*) the ataxia; (*d*) absent knee jerks; and (*e*) disturbance of speech.

Differentiation.—1. In *tabes* there are also ataxia and absent knee jerks, but in hereditary ataxia the syphilitic stigmata or history, the immobile pupil, eye paralysis, optic atrophy, pains, crises and vesical, trophic and marked sensory disturbances are absent.

2. *Multiple sclerosis* (in which there are also speech alteration, nystagmus, ataxia and tremor) is differentiated by the spasticity, increased reflexes, optic atrophy, compulsory laughing and other psychical signs, apoplectiform insults, intentional tremor and slow, scanning speech.

3. In *cerebellar disease* the ataxia is similar, but in tumors there are optic neuritis, headache and vomiting. Marie, Londe and Nonne described a clinically similar affection, *hereditary cerebellar ataxia,* which is probably due to simple congenital atrophy of the cerebellum (middle lobe), degeneration in Clarke's columns, cerebellar tracts and posterior columns, is also hereditary or familial, but develops later, between the twentieth and thirtieth years. The ataxia is cerebellar. The *reflexes are increased* (which Patrick thinks is the sole differentiating point), often with spasticity; quite frequently there are *optic atrophy,* limitation of the visual field for objects and color, eye paralysis and sometimes an immobile pupil, nystagmus, choreiform movements and moderate sensory disturbance and speech disturbance,

which may be nasal and marked by unequal inspiration and expiration. The club foot, scoliosis and Romberg's sign are absent. Sanger Brown has reported 25 and J. H. Neff 13 cases.

4. *Cerebral diplegia* of children and ataxic paraplegia are distinguished by reflex increase and spasticity.

5. *"Amaurotic family idiocy"* (Sachs, Peterson, Hirsch) is characterized by paresis or paralysis, idiocy, optic atrophy, marasmus and a rapidly fatal course before the third year of life. At autopsy hypoplasia, microgyria, decreased pyramidal cells, tangential and other fibers are noted. Sachs observed 27 cases in Jews and Schaffer collected 86 cases. Vogt describes a juvenile form, after puberty.

Treatment.—Treatment is wholly symptomatic.

2. Ataxic Paraplegia (Gowers).—Most cases occur in males between thirty and forty; cold and concussion are possible causes; syphilis is a rare cause. *Pathologically,* there is a lateroposterior sclerosis; in the lat. column the degeneration is not wholly systematic, symmetrical or pyramidal; it may invade the mixed zone; in the post. column the process is not more intense in the lumbar cord and is less intense in the root zone than is the case in tabes. Sometimes the process is so diffuse that it suggests myelitis or arterial obstruction.

Symptoms.—The early symptoms are those of a gradual *spastic paraplegia* (muscular weakness, exaggerated reflexes, moderate rigidity), followed by *ataxia* (mostly in the legs, with Romberg's sign). The arms are involved later and to a less degree. In the last stages the ataxia is obscured by the spastic paraplegia; vesical or rectal tenesmus and muscle cramps are fairly common. Cranial involvement is infrequent, though sometimes suggested by slightly disturbed articulation, nystagmus, tremulous facial movements and increased jaw-jerk. The mind is usually clear. Sexual activity is often lessened and the sphincters are sometimes moderately disturbed. Dull sacral pain is sometimes observed.

Differentiation.—Differentiation is necessary (*a*) from *tabes.* Certain tabetic symptoms are absent, as the high-stepping and sudden stamping gait, the immobile pupils, optic atrophy, eye paralysis, lancinating pains, crises, other sensory disorders and absent knee jerks. (*b*) *Friedreich's ataxia* is distinguished by the age, heredity, speech and absence of patellar reflexes. (*c*) *Myelitis* is more acute, diffuse and later is regressive. (*d*) From simple *spastic paraplegia* it is recognized by the ataxia.

Prognosis and Treatment.—The prognosis and treatment are those of spastic spinal paraplegia.

3. Other Combined System Diseases.—Combined sclerosis may be (*a*) *exogenous,* as in pellagra (*q. v.*) with degeneration, and then sclerosis of the posterior lateral columns; as in ergotism (*q. v.*), the longest known variety, and in lathyrism (*q. v.*), or it may be (*b*) *endogenous,* as in multiple neuritis, diabetes, pernicious anæmia, icterus, leukæmia, brain tumor, plumbism or diphtheria. Some toxin

causes degeneration followed by sclerosis. It was found by Putnam in enfeebled middle-aged persons, especially in women.

Pathologically, there is posterolateral sclerosis with diffuse or discrete degenerative foci. All the columns may be affected. The most diffuse changes are in the cervical and the least in the lumbar cord and sometimes changes exist in the ant. horns and Clarke's columns. There are sensory *symptoms* (lancinating pains, paræsthesia, anæsthesia, ataxia), motor symptoms (paresis, paraplegia, choreiform movements) or reflex symptoms (the patellars being exaggerated or lost); optic neuritis and œdema are occasional findings.

DISEASES OF THE PERIPHERAL NERVES.

MONONEURITIS.

Definition.—Inflammation of a single nerve trunk.

Etiology.—(*a*) *Injuries, wounds,* fractures, dislocations, delivery, violent muscular contractions, as in blacksmiths; exhaustion or disturbed metabolism; anæsthesia (ether) paralyses, the arms being held over the head (in the Trendelenberg position), the legs bound in holders or when Esmarch's bandage is applied; ether hypodermatically, malpositions of the arm during sleep and the use of crutches are causes. (*b*) *Contiguous disease* (pleurisy, pelvic inflammation, compression by tumors or aneurysm) may produce neuritis. (*c*) *Cold* (rheumatic paralysis) causes inflammation in the sheath, whence extension to the nerve. (*d*) *Toxic causes,* as plumbism and alcoholism, and acute or chronic infections (*v. i.* Multiple Neuritis) are factors.

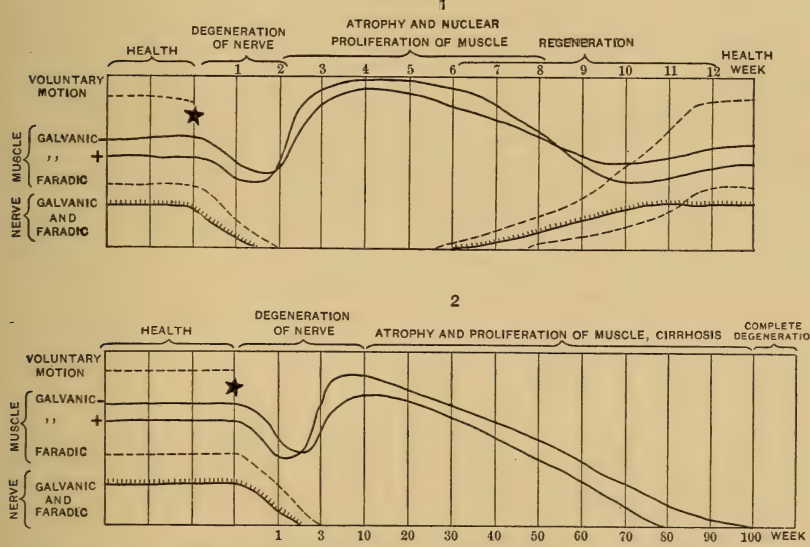
Pathology.—In *acute cases* the nerve is red, swollen, soft and sometimes the seat of punctate hemorrhages or extravasation of white cells in the sheath or between it and the nerve fibrils. In some cases inflammation involves the sheath chiefly (*perineuritis*) and in others the interstitial tissue (*interstitial neuritis*), in which the included nerve fibrils are readily compressed. The process is either disseminated or focal (where the nerve divides or enters the fascia or bone). In many cases the nerve fibrils are most affected (*parenchymatous neuritis*). It is often difficult to distinguish between inflammation and degeneration; in both processes there are nuclear multiplication and protoplasmic swelling in the neurilemma; this compresses the myelin sheath, which becomes varicose, granular, fragmented, shows nuclear increase and finally becomes emulsified. The axis cylinder, composed of thirty to fifty primitive fibrils, becomes granular, divided and may largely or wholly disappear. *Regeneration* is probably accomplished from the spinal or cerebral centres but also from the periphery.

Symptoms.—Symptoms vary with the intensity, extent and seat of the neuritis (see INDIVIDUAL NERVES). Constitutional disturbance may attend acute cases.

1. SENSORY SYMPTOMS.—The chief local symptom is *pain,* experienced largely in the sensory nerves, either locally or eccentrically over their area of distribution; it is increased by tension or movement, is usually intense and is sometimes increased at night. *Tenderness* is referable to involvement of the nervi nervorum (Weir Mitchell). Hyperæsthesia, hypæsthesia and paræsthesia may be noted, but complete anæsthesia is rare except for relatively small areas. Sometimes the swollen nerve or perhaps nodules (*n. nodosa*) may be felt.

2. MOTOR SYMPTOMS.—The *muscles* supplied are *tender* to touch, painful on movement, *paretic* or paralyzed, and atrophied (separation from their trophic centres or loss of reflex connections). They are narrower, less clearly striated, granular and finally indurated. Muscular contractures may result reflexly, especially in traumatic and professional or occupation neuritides. The *reaction of degeneration* is present. The normal nerve is stimulated by the faradic (induced) and by a stronger degree of the voltaic (constant) current; the faradic

FIG. 76.



1, paralysis with early return of motion; 2, incurable paralysis with complete degeneration. (Erb, modified by Church.)

produces a continuous muscular contraction and the voltaic produces a contraction when the circuit is made or broken,—the cathode closing contraction, which is first to appear and stronger than the anode closing contraction. In disease the following changes from normal occur (Church): (*a*) "The *muscle* responds weakly, sluggishly and

deliberately to faradism, and tends to maintain contraction after the current is withdrawn. This is the *modal change,* the most essential element in the reaction. (*b*) "The *nerve trunk* loses progressively and equally its responsiveness to both galvanism and faradism—a *quantitative change.* (*c*) "The *muscle* becomes much more excitable by galvanism and much less excitable by faradism; the latter reaction and the responses in the nerve trunk are completely lost after two or three weeks. This is the *qualitative change.* (*d*) "A *polar change* appears in the muscle about the second week when directly stimulated by galvanism; the muscle is not only more readily caused to contract by the constant current, but the normal mastering strength of the negative closing contraction over the positive has disappeared and the positive closing contraction is equal to or greater than the negative."

In the middle or *mixed form of Erb* there is a normal reaction in the nerves and increased voltaic response in the muscles.

3. Trophic Symptoms.—The skin is sometimes red, "glossy," œdematous or more rarely thickened. Increased secretion of sweat, eruptions as herpes, erythema or pemphigus, inflammation or adhesions in the joints and perforating ulcers are less common.

Neuritis may sometimes *ascend to the cord* or its membranes as in traumatic cases or in bladder disease. The *progressive hypertrophic interstitial neuritis of childhood* (Déjérine and Sottas, 1893) is a rare affection, in which there is anatomically sclerosis and hypertrophy of the nerves, ant. and post. nerve roots, and sclerosis of the post. columns as in tabes, and clinically, muscular atrophy, pains, anæsthesia, nystagmus, pupils immobile to light, and kyphoscoliosis.

Diagnosis.—Diagnosis depends on (*a*) localization of symptoms to a nerve trunk and its distribution; (*b*) pain, tenderness and objective sensory alteration; (*c*) muscular atrophy or paralysis; and (*d*) trophic changes. The early diffuse pain may simulate disease of the *bones or joints.* Chronic (sensory) forms may resemble *neuralgia,* in which the pain is paroxysmal, the tender points more localized, the initial tenderness less and objective sensory alteration absent. Disease of the cord or *nerve roots* usually paralyzes several muscles and affects sensation segmentally.

Prognosis.—The prognosis depends on the cause and intensity of the process. Acute cases last for weeks; chronic cases, for months. The outlook is usually good in traumatic cases and bad when they are due to contiguous suppuration.

Treatment.—The treatment is (*a*) that of the cause. (*b*) *Absolute rest* is indicated. Exertion increases just as it may initiate, neuritis. (*c*) *Pain.* The salicylates and iodides are of uncertain value. Local heat may relieve, but sometimes causes burns and ulcerations, for the anæsthetic skin may not perceive its intensity. Coal-tar products should be given in preference to morphine, cocaine or Schleich's infiltration anæsthesia, because habits may be contracted; opiates are necessary in severe cases. The positive pole of the voltaic current, just strong enough to be felt, may relieve pain. (*d*) *Paralysis* should

be treated only after the acute stage. The weak interrupted voltaic current (not the faradic) is indicated. Massage and later passive motion are beneficial largely because the veins from the nerves empty into the muscle veins and surround the muscle arteries.

MULTIPLE NEURITIS.

Multiple neuritis (polyneuritis, peripheral neuritis) was described by James Jackson (1822) and Ladd (1854), but the first accurate and pathological description was given by Dumeil in 1864, while Joffroy, 1879, Leyden, 1880 and Stewart, 1881, made important contributions.

Definition.—Neuritis (or nerve degeneration) due to the selective action of toxic substances on the less resistant peripheral nerves, causing (*a*) multiple, symmetrical, bilateral, flaccid or atrophic paralysis with (*b*) the reaction of degeneration, (*c*) sensory and (*d*) often trophic disturbances.

Etiology.—The causes are (*a*) *poisons; alcohol* is the most important; lead, arsenic (in beer), mercury, phosphorus, carbon monoxide, silver, aniline and ergot may also produce the disease. (*b*) The *infections*, acute, as *diphtheria,* typhoid, sepsis and also, occasionally, almost every active infection, and chronic, as *beri-beri, leprosy,* tuberculosis or syphilis, are etiological factors. (*c*) *Intoxications,* as auto-intoxications, *diabetes, nephritis, digestive disorders, cachexias* and pregnancy, may be causal. (*d*) *Other causes* include the seemingly primary cases. Cold and *rheumatism* may produce it; our present knowledge cannot explain their mode of action; probably these cases should be classified as infectious, for spontaneous multiple neuritis has occurred epidemically. Arteriosclerosis of the nerve vessels is a rarer cause. It may occur with tabes. It is a disease of adults (twentieth to fiftieth year); in children it may occur with poliomyelitis; Thomas and Greenbaum, 1907, collected 138 non-diphtheritic cases in children.

Pathology.—In *acute cases* the nerve may be red, swollen and the seat of punctate hemorrhage, exudation of leukocytes or albuminous exudation in the connective tissue, but usually *parenchymatous degeneration* predominates. It appears first and most markedly in the smaller nerves. As in mononeuritis, the axis cylinders waste and the medullary sheaths disintegrate. The symmetry and multiplicity of degeneration in the nerve endings are caused by the blood condition or toxins. Acute polymyositis of similar origin may accompany neuritis multiplex. In cases which are *chronic* from the beginning there is slow degeneration of the nerves. The connective tissue is increased in certain forms, as gouty or leprous neuritis. The toxic cause may also produce degeneration of the cord or brain and changes in the liver (alcoholic cirrhosis), kidneys (alcohol and lead), etc.

Symptoms.—The symptomatology varies considerably with the cause. In *acute cases* it may begin with chills, fever, delirium, head-

ache, diarrhœa, icterus, bronchitis, acute splenic tumor, albuminuria or other toxæmic symptoms; these are followed by prodromal numbness, tingling, pains, cramps or vasomotor symptoms, while in *chronic cases* the symptoms develop slowly.

1. Sensory Symptoms.—Sensory symptoms are usually *first to appear,* the last to disappear and are throughout the most vexing manifestations. Paræsthesia precedes pain and pain precedes paralysis. The muscles, nerve trunks and skin are the seat of *spontaneous pain, tenderness to pressure* and *pain on movement.* The palms of the hands and soles of the feet are especially hyperæsthetic in the tabetic, diabetic and diphtheritic forms. *Sensation* is often *blunted,* perverted or less often completely suspended; the most marked findings exist *below the elbows and knees,* although higher extension, even to the face, is possible. Sensory dulness may concern pain, temperature, tactile or muscular sensation. Some sensory disturbance is rarely absent. Delayed conduction may occur. The joints may be painful and swollen, especially in the hand, causing thickening and limitation of movement.

2. Motor Symptoms.—Motor symptoms occur chiefly in the *peripheral* distribution of the *spinal nerves;* they develop first and are most marked in the *extensors* of the *leg and foot, forearm and hand,* and produce the *ankle and wrist drop* (ext. popliteal and musculospiral nerves). The foot is also inverted. The paralysis is *flaccid,* being rarely attended by muscular spasm. If the paralysis is only moderately severe in the legs, the *"steppage"* gait is noted (Charcot) and the patient lifts the legs high to clear the pendant toes ("turkey-gobbler walk"). The paralysis later involves the flexors and the muscles of the arm and thigh. The muscles may show an early increased irritability to both the galvanic and faradic currents; this is followed by some or all of the elements of the *reaction of degeneration* (sometimes before paralysis appears, Popoff). The muscles rarely fail to respond to the galvanic current, even in the extreme cases. Then the *muscles atrophy,* chiefly *in distal parts, i. e.,* where the sensory and motor changes predominate. Other motor manifestations are cramps, tremor, fibrillation, ataxia and contractures. The patient either lies on the back with the members extended or on the side with flexor contractures, which draw the heels to the buttocks and the thighs to the abdomen. The trunk muscles may be affected as well as those of the neck, eyes, face, tongue or indeed those supplied by any cranial nerve; rapid pulse, palpitation, dyspnœa and vocal disturbance indicate involvement of the vagus; optic neuritis and amblyopia may occur. Involvement of the cranial and spinal nerves is observed in the neuritic form of Landry's paralysis. The tendon and skin *reflexes decrease and disappear* although initial increase or exceptional persistence has been noted. The organic reflexes are almost always preserved. Involvement of the bladder and rectum, which is very rare, is referable to the mental state or to neuritis of the pudendal plexus.

3. Trophic Symptoms.—The skin is cold, turgid, harsh, sometimes glossy; herpes, erythema, ulceration, bed-sores and trophic changes in the nails or hair are uncommon; œdema over the wrists and ankles, especially in alcoholics, is trophic or vascular in origin.

4. General Symptoms.—Mental symptoms and malnutrition may result from the antecedent toxæmia or cachexia, pain or disturbed circulation; Korsakow described the "neuritic psychosis," marked by amnesia for the recent past, confabulation, delirium and hallucinations. Recovery is the rule. Some visceral symptoms have been referred to involvement of the cardiac, sacral and pudendal plexuses, although mostly without positive evidence.

Course and Prognosis.—Whether the disease begins acutely or slowly, the common order of symptoms is paræsthesia, pain, paralysis, amyotrophy, trophic and mental symptoms. The distal parts are involved more early and intensely than the proximal. In most cases recovery requires about a year. Paralysis of the vagus may cause death. The *prognosis* depends in part on the cause. It is good in sensory types and in forms of slow evolution, when the sensorium, arms and trunk, cranial nerves and sphincters escape involvement, and when visceral disease, as liver cirrhosis, cord or brain complications, and œdema, are absent. The prognosis must regard the possibility of recurrence (recurrent type) and cover the progress of the disease, which is inevitably slow, despite treatment.

Diagnosis.—Early diagnosis is important, in order that etiological therapy may be instituted. The *cardinal signs* are (*a*) the motor and sensory symptoms, which correspond in function, peripheral distribution and symmetry; (*b*) the reaction of degeneration; (*c*) tenderness of the nerves and muscles; (*d*) loss of reflexes and (*e*) the etiology.

Diagnosis of Type.—(*a*) The *alcoholic* form is the most frequent; 70 per cent. of cases occur in women; the legs are often involved; there is the "steppage" gait; sensory and mental symptoms are most marked; the atactic form is not infrequent (*v. i.*); recurrence is probable. (*b*) When due to *lead poisoning,* the occupation, lead line, colic and constipation are characteristic; the arms alone are involved as a rule; there is little sensory participation; the involvement rarely extends; the wasting is conspicuous. (*c*) *Diphtheritic* form (*q. v.*). (*d*) In the *toxæmic* form there are fever and constitutional reaction; it begins in the legs or in all members simultaneously, involves equally the flexors and is attended by slight sensory symptoms (Gowers). (*e*) When due to *beri-beri* (*q. v.*), vagus symptoms, dropsy and cardiac failure are frequent. (*f*) *Leprous neuritis,* erythromelalgia and Dercum's disease are considered elsewhere.

Differentiation.—Atactic polyneuritis (pseudotabes, Déjérine) with little motor disturbance, may resemble locomotor ataxia:

Locomotor Ataxia.	*vs.*	Multiple Neuritis.
History or stigmata of syphilis.		Rare; history of infection or intoxication.

Locomotor Ataxia.	Multiple Neuritis.
Argyll-Robertson pupil.	Absent (loss of accommodation in diphtheria).
Optic atrophy, 10 per cent.	Rare, but toxic amaurosis is frequent.
Eye paralysis frequent.	Much more rare.
Early girdle sensation and lightning pains.	Very rare.
Muscular sense early and frequently disturbed; ataxia.	Rare.
Gait ataxic, stamping, striking heel first.	Gait due to motor more than sensory disturbance. Striking toe first.
Sensory symptoms predominate.	Motor predominate.
Paralysis, reaction of degeneration and amyotrophy rare.	In nearly all cases; rapid in development and symmetrical.
Nerve trunks often insensitive.	Tender, as are muscles.
Arthropathies, osteopathies, *mal perforant.*	Unknown; glossy skin more common.
Crises.	Almost unknown; toxæmic vomiting, etc.
Sphincters early and frequently involved.	Very rarely, if ever, and from delirium, etc.
Clinical evolution very slow.	Rapid.
Disease incurable.	Recovery usual, if not early death.

Poliomyelitis.	Multiple Neuritis.
Usually in children.	In adults.
More sudden onset.	Less acute.
Embraces whole limb; random distribution.	Peripheral and symmetrical.
Muscles functionating together. Reaction of degeneration in both, but strong galvanic current not required (during first months) to produce muscular contraction.	Dissimilar muscles involved. Muscles respond only to strong galvanic current.
Pain and tenderness most rare.	Common in nerves and muscles.
Sensory disturbance rare.	Usually (but not invariably) present.
Immediate tendency to regress, but always some deformity.	Tendency to progress. Total recovery the rule.

In *acute myelitis* the onset is acute, there are girdle pains, paraplegia, with corresponding anæsthesia, bed-sores, disturbance of the bladder and rectum; there is no tenderness of the nerves or muscles,

little or no ataxia and recovery is unusual. *Landry's paralysis* often cannot be distinguished (one form of Landry's disease is neuritic); sensory, trophic and sphincter changes are absent.

Treatment.—In the etiological therapy alcohol must always be considered and deception guarded against; it should be withdrawn at once. In sepsis, Gowers gives full doses of iron. For treatment of pain see NEURITIS. Absolute rest should be enforced. A full diet, rich in fats, should be given if the stomach can tolerate it; tonics should be given to maintain nutrition, but arsenic should be avoided. When the lower cranial nerves are affected, feeding by the nasal tube or by rectum, measures to avoid hypostasis, as in typhoid, and cardiac stimulants are indicated. To overcome contractures from ankle- or wrist-drop, splints or sand-bags should be employed when they do not cause excessive pain. The weight of the bedclothes on the feet may promote contractures, whence frames to lift the bedding should be used. Gentle massage and voltaism may be tolerated after the acute stage, may prevent contractures and may aid the return venous flow. Bromides and chloral are the most certain somnifacients, but chloral must be used with care in chronic alcoholism. Orthopedic measures are indicated in neglected contractures.

NEUROMA.

Two varieties of neuroma exist, the true and the false. The true neuroma may be medullated or non-medullated, though ganglion cells are exceptional (only 3 cases are recorded); they are usually multiple, sometimes very numerous (3,020 in one case) and have been found in instances on every cranial, spinal and sympathetic nerve. They may undergo sarcomatous degeneration. The *tubercula dolorosa* (Virchow) are small, painful, subcutaneous nodes on the trunk or extremities, the anatomy of which is not well understood. The *neuroma plexiforme* consists of twisted cords with nodes, is congenital and occurs largely in the distribution of the fifth nerve. The *false neuroma* is usually fibrous; glioma is rare; sarcoma or carcinoma may by contiguity invade adjacent trunks; neuromata have been found in the cerebral nerves in syphilis and in the peripheral nerves in leprosy. The nerve trunks suffer more in the false than in the true form. *Neuromata in amputation stumps* are frequent.

Symptoms and Treatment.—There may be no *symptoms;* local tenderness and local or projected pain are frequent. Reflex muscular spasm is common from nerve irritation. Mentality, sensation or motility may be affected. Pigmentation has occurred. Diagnosis is certain only when the growths are felt. *Treatment* is symptomatic for the exhausting pain or radical in localized growths. Recurrence is not infrequent.

DISEASES OF CRANIAL NERVES.

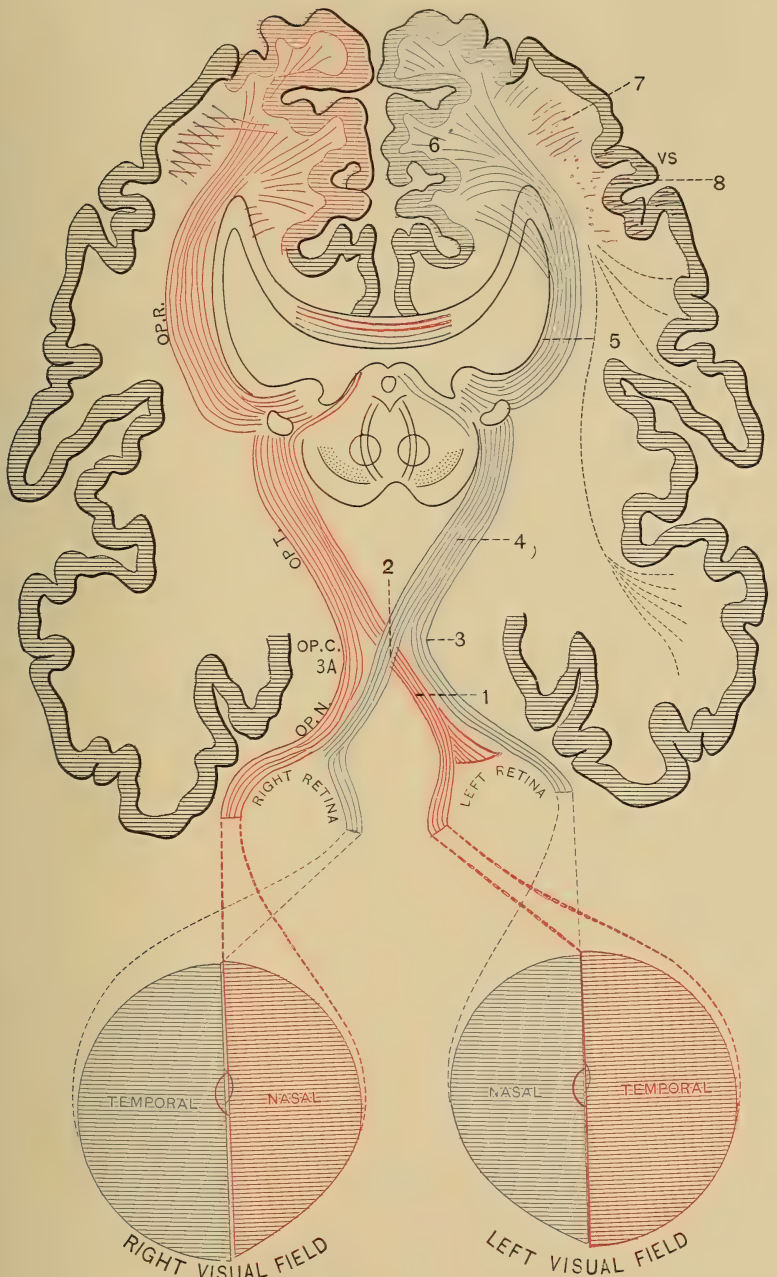
Olfactory Nerve.—The end nerves of smell are located on the two upper turbinate bodies and the upper septum; thence they run to the olfactory bulbs, which are really part of the brain. The central connections are not well understood. The centre is thought to be in the uncinate gyrus and the hippocampus, with relations to the optic thalamus, opposite internal capsule and cerebral cortex. The anterior nares perceive odors, the posterior, flavors.

Anosmia is loss of smell. Tests are made not with pungent substances, as ammonia, which irritates the fifth nerve, but with musk, asafœtida, cologne water and the essential oils. (*a*) Disease in the nasal mucosa is the most frequent cause; it may result from paralysis of the fifth nerve, arrest of secretion or atrophy. (*b*) Disease may result from congenital absence of the bulb, basal fracture or meningitis, tabes, foci in the opposite internal capsule, etc. (*c*) Central disease may be located in the hippocampus and uncinate convolution, as softening. The prognosis is usually unfavorable and treatment is ineffectual. *Hyperosmia,* or oversensitiveness of smell, occurs in insane and neurotic subjects; it may be cultivated, so that individuals are recognized by their odor. *Parosmia,* or perverted sensation, is rare and arises from irritation of the nerves or centres. Hallucinations of smell may occur. Imaginary odors, as of burning flesh, may constitute the epileptic aura.

Optic Nerve.—1. The Retina.—*Retinitis* occurs in general diseases, as nephritis (in 20 per cent.), especially in interstitial forms and late in the disease rather than early. The degenerative form is most common, the next is the hemorrhagic form, with "flame-like" hemorrhages along the vessels, and finally the inflammatory type, with swelling of the retina, serum exudation, cloudiness and disk blurring, which may be as marked as that of brain disease; the white foci are due to exudation, fatty change or sclerosis. Similar findings may occur in syphilis, anæmia, malaria, leukæmia, plumbism, diabetes, etc. (see PLATES VII and VIII). There are apparently primary cases.

2. Optic Nerve.—*Optic neuritis* (papillitis, choked disk) has been fully described and differentiated (see BRAIN TUMOR and PLATES VII and VIII). Tumors or inflammation in or back of the orbit may be causal. Errors in refraction must be excluded. In the *primary form* there is no initial inflammation. The edges of the disk are distinct, its arteries nearly normal and its color grayish; it may develop hereditarily in males at puberty; it occurs in tabes, paretic dementia, multiple sclerosis, syphilis and less often from exposure, excesses, diabetes, alcoholism, plumbism, the specific fevers, amaurotic family idiocy and familial cerebellar ataxia. The *form secondary to inflammation* commonly follows choked disk and has the same significance; the disk has an irregular contour, small arteries and is translucent. The symptoms are diminution in acuity of vision, field of vision, color perception and the pupillary reaction.

PLATE XXI



Visual Paths. (After Vialet.)

OP.N., optic nerve. OP.C., optic chiasm. OP.T., optic tract. OP.R., optic radiation. V.S., visual speech centre. A lesion at (1) causes total blindness in that eye; lesion at (2), bitemporal hemianopsia; at (3), unilateral nasal, and lesions at (3) and (3A), bilateral nasal hemianopsia; at (4), hemianopsia of both eyes and the hemianopsic pupillary reaction; at (5) or (6), hemianopsia of both eyes (pupillary reflexes being normal; at (7), amblyopia, especially of the opposite eye; at (8), word-blindness.

3. Chiasm.—Each half of the retina contains fibers from the corresponding occipital lobe (see PLATE XXI), that is, each right half receives fibers from the right and each left half from the left occipital lobe. The point of sharpest vision, the macula, receives overlapping fibers from both sides of the brain. The outer temporal portion of the field of vision is more limited than the inner nasal portion, because the nose cuts off light and it receives fewer nerve fibers. The larger number of fibers from each eye, *i. e.,* those from the nasal retinal half, decussate at the chiasm and lie in its center. Those from the temporal half are direct and lie at the side of the chiasm. While disease of one optic nerve causes blindness in the corresponding eye, lesions affecting the centre of the chiasm (decussating fibers to the nasal half of each retina) cause half blindness in the opposite (temporal) visual fields, *bitemporal hemianopsia,* since oblique rays passing the pupil fall on the opposite half of the retina. The macula receives fibers from both sides of the brain and escapes involvement. Temporal hemianopsia may result from tumors of the pituitary gland, pressure from the third ventricle, gummata and hemorrhage. The "oscillating" or varying bitemporal hemianopsia is often indicative of syphilis. The diagram illustrates effects of lesions of the lateral part of the chiasm (3), lesions affecting both sides of the chiasm (3 and 3A), as calcareous or aneurysmatic carotids; *binasal hemianopsia* is most exceptional; a large lesion might produce temporal hemianopsia in one eye and total blindness in the other; still more extensive disease might produce total blindness; these changes may be successive.

4. Optic Tract.—Just back of the chiasm the temporal fibers of the right and the nasal fibers of the left side form the right optic tract, *i. e.,* the right tract contains all fibers from the right half of each retina. The tract runs over the crus cerebri, where a lesion very often causes hemiplegia on the same side as the blind field. The fibers enter the geniculate bodies (in which 80 per cent. of the optic fibers end), opt. thalamus and ant. quadrigeminate body, from which gray ganglia the fibers pass by way of the posterior limb of the internal capsule and, by radiating fibers (optic radiation), to the visual centre in the posterior internal part of the occipital lobe, the cuneus. A lesion (at 5, in diagram) in the optic tract anywhere between the chiasm and cuneus produces *lateral or homonymous hemianopsia;* for instance, if it occurs in the right optic tract it produces loss of function in the temporal half of the right and nasal half of the left retina, so that the left half of each field of vision is lacking. Hemianopsia may be partial and may involve only a quadrant, or only a quadrant may remain after partial recovery. The unaffected fields may suffer some reduction. *Heteronymous hemianopsia* designates blindness of the right half of one field and the left half of the other or the converse. Disease in the gray ganglia or internal capsule may produce contralateral hemiplegia; in 50 per cent. of cases of hemianopsia this association is observed, and less frequently hemianæs-

thesia. Transient hemianopsia is sometimes seen in migraine. The optic tract is injured in tumors, multiple sclerosis, trauma, hemorrhage or softening.

5. Optic Centre (See CEREBRAL LOCALIZATION).—A lesion in the cuneus produces (*a*) *homonymous hemianopsia.* Sometimes only an upper quadrant is blind (disease in the upper part of the cuneus) or the lower quadrant is blind (disease in its lower part). Purely cortical hemianopsia is impossible, and Monakow held that the optic radiation must be involved; (*b*) *hemichromatopsia,* or homonymous color blindness or confusion, in which all colors seem gray, is probably due to lesion of the anterior superficial cuneal cortex. Other results are (*c*) *mind blindness,* or loss of visual memory; (*d*) *alexia* or word blindness; (*e*) *optic aphasia;* and (*f*) crossed *amblyopia,* concentric blurring or limitation of the visual fields for form and color; this is usually referred, however, to disease of the gyrus angularis, in which there is thought to be a higher centre in which the half fields are combined and the whole opposite field and part of the field of the same side are represented.

Diagnosis.—The eyes should be tested separately. Disease of *an optic nerve* causes loss of sight and pupillary reaction in the corresponding eye. *Central scotoma* (blindness in the centre of the visual field) indicates inflammation or hemorrhage in the central (axial) fibers of the nerve, usually back of the orbit; it is common in poisoning from tobacco and alcohol. *Peripheral limitation* of the visual fields indicates damage to fibers coursing in the periphery of the optic nerve and some damage to all the nerve fibers. *Sectorial blindness* indicates a pronounced but circumscribed affection of the nerve. *Bitemporal hemianopsia* always results from chiasm disease; unilateral temporal hemianopsia in one eye with complete blindness in the other has the same significance, but also implies that the process has extended laterally, possibly even to the optic tract. *Lateral homonymous hemianopsia* indicates disease of the optic tract between the chiasm and cuneus. If the geniculate (or quadrigeminate) bodies are the seat of the lesion, *Wernicke's hemianopsic pupillary reaction* is obtained, because the pupil reflex, while not at present perfectly understood, implies that the retina, the optic nerve, the centre and fibers of the third nerve and the iris are intact. A small beam of light in a dark room should be thrown on the hemianopsic half of the retina and the patient should look at a distance to eliminate accommodation. If the pupil does not react to light on the blind half, Wernicke's reaction indicates that the disease focus lies at or somewhere to one side of the geniculate (or quadrigeminate) bodies. If the lesion lies between these gray ganglia and the cortex, the reaction of Wernicke is absent. Transient hemianopsia is common in vascular lesions; if it lasts for weeks it will probably remain. In Sequin's case it was present for twenty-three years. It may occur in migraine and very rarely in hysteria. Its association with the crus, internal capsule or cortex symptoms which have been already considered is of

localizing importance. *Amblyopia and concentric limitation* in the fields, marked in one eye and less in the other, may result from (*a*) optic atrophy, with the absent pupillary reaction, and seen with the ophthalmoscope; (*b*) disease of one hemisphere, the ophthalmoscopic finding and pupillary reflex being normal (or (*c*) possibly functional disease).

Functional and Toxic Blindness.—(*a*) Hysterical blindness is transitory and rarely complete (the eye functionates normally though the visual centre neglects the impressions); it is often associated with loss or inversion of the color perception and often with hemianæsthesia. (*b*) *In amblyopia* from tobacco, vision is gradually impaired in both eyes, largely as central scotoma with invariable loss of perception for red and green colors; the fundus is usually normal but sometimes congested; recovery is usual, but atrophy may result. (*c*) *Toxic amaurosis*, usually transitory, may occur from uræmia, poisoning by lead, alcohol or quinine, syphilis, acute anæmia and irritation of the fifth nerve.

Ocular Paralysis; Third, Fourth and Sixth Nerves.—General Etiology.—(*a*) *Trauma* to the eye, orbit or base may cause ocular paralysis; it is characterized by a slow course and often imperfect recovery. (*b*) *Compression* by tumors in the orbit which often produce exophthalmos; tumors at the base, aneurysm and rarely hemorrhage into the nerve sheath may produce it. (*c*) *Neuritis* produced by the above factors, orbital cellulitis, meningitis, syphilis, cavernous thrombosis, alcoholism and rheumatism, which is made to cover many obscure cases, may be etiological factors. Neuritis may also result from acute infections, as diphtheria; exophthalmic goitre, plumbism and diabetes are rarer causes. (*d*) *Nuclear disease* is usually due to chronic wasting disease, as in tabes, multiple sclerosis or bulbar palsy, but it is sometimes acute from polio-encephalitis or vascular lesions. (*e*) *Supranuclear disease* (*v. i.* Conjugate Deviation) may be a cause. (*f*) In rare cases there is a myopathic form, as in muscular dystrophies.

Anatomy and Physiology of the Motor Nerves of the Eye.—The nerve supply is by the sympathetic (which through the ciliary ganglion dilates the pupil) and by the third, fourth and sixth cranial pairs, which arise from the middle ventricle, Sylvian aqueduct and fourth ventricle and are arranged as follows from before backward: (*a*) the third nerve nucleus with first the ciliary and behind it the pupillary centre, then its muscles in this order: the internal rectus, levator palpeb. superior, inferior rectus and inferior oblique; (*b*) fourth nerve nucleus for the superior oblique, lying close to the third nucleus, and (*c*) nucleus of the sixth nerve for the ext. rectus. Their connection with the motor cortex (and possibly with the occipital and parietal lobes) is through the internal capsule and corona radiata. The third nucleus is connected with the seventh; winking and movements of the eyelid are thus associated with the pupil reaction and accommodation. The action of the various muscles is indicated by

their names but convergence is supplemented by the sup. and inf. recti and the two oblique muscles. The inf. oblique turns the eye upward, the sup. oblique downward. The upper lid is raised by the levator palp. sup. (acting usually with the sup. rectus) and by Müller's muscle.

Paralysis of the Third Nerve.—This nerve emerges from the crus and follows the cavernous sinus, and therefore may be involved in disease of these structures. A complete paralysis involves all muscles save the external rectus and sup. oblique and results in drooping of the lid (ptosis), which the patient attempts to overcome by overaction of the frontalis. The eye can be moved only outward and a little down and inward. Accommodation is lost, the pupil is wide from the unopposed action of the sympathetic nerve and does not react to light. There is *double vision from divergent strabismus,* due to action of the unrestrained ext. rectus muscle. Paralysis is often partial, for the levator palp. sup. may escape involvement or be affected alone or with its associate (the sup. rectus), especially in bilateral nuclear disease. *Ptosis* may be (*a*) part of an ordinary oculomotor palsy; (*b*) part of a chronic ophthalmoplegia (*v. i.*); (*c*) reflex in rare cases of trigeminal irritation, as from disease of the teeth; (*d*) myopathic, as in muscular dystrophy; (*e*) a result of sympathetic paralysis, false or pseudoptosis (with a red, hot, œdematous skin, slight recession of the eyeball and contracted pupil); (*f*) an incurable congenital form, usually partial and bilateral; (*g*) a transient form, observed in the morning in women, usually with a good prognosis; (*h*) an hysterical form, usually bilateral, in various associations with the sup. rectus, inf. or int. rectus, the eyebrow being frequently lower on the side of ptosis.

Internal Oculomotor Palsy.—Internal oculomotor palsy includes cycloplegia and iridoplegia. (*a*) *Cycloplegia* (paralysis of the ciliary muscle) results in loss of accommodation. Near vision is poor, distant vision is good; extreme myopia or presbyopia make its detection difficult. Isolated cycloplegia in one eye may indicate disease of the lenticular ganglion, in both eyes it is nuclear, as in diphtheria or tabes. (*b*) *Iridoplegia* may assume three forms (Gowers). The first is loss of associated action, *accommodation iridoplegia,* in which there is no pupillary narrowing in the act of accommodation. It is due to the same causes as cycloplegia and is usually but not always associated with it. The second is *loss of the light reflex,* reflex iridoplegia, the Argyll-Robertson pupil, which is seen especially in tabes and paretic dementia (very seldom in softening, tumors, senile atrophy, hydrocephalus or congenitally). The reflex occurs through the optic nerve, optic tracts, corp. quadrigemina or corp. geniculata, anterior part of the oculomotor nucleus, probably the second branch of origin of the third nerve, its trunk, the ciliary ganglion and its nerves. The pupils are often equally affected and small (spinal myosis) but may be unequal (aniscoria, also observed in some normal persons). If the pupils are not small, cutaneous iridoplegia is not

present. The third form is *loss of the skin reflex,* the cutaneous iridoplegia of Erb, in which the normal dilatation of the pupil upon pinching, sticking or faradizing the skin of the neck is lost. It indicates, in general terms, disease in the cervical sympathetic nerve, in the fibers between it and the cervical cord, diseases of the cord, especially those affecting sensation, or of the centre, probably situated in the corp. quadrigemina just outside the light reflex centre in the trunks of the first and third nerves. The seat of lesion is probably nuclear, unless special localizing symptoms indicate some other localization. Specifically it is characteristic of tabes, paretic dementia or syphilis.

Recurrent or Periodic Palsy.—Recurrent or periodic palsy, to which Gubler (1860) and Möbius particularly drew attention, is a rare form, involving chiefly most (or possibly only one branch) of the third nerve, and rarely the sixth nerve. It occurs chiefly in women. At intervals of months to years, possibly throughout life, a recurring paralysis develops, lasts a few days or weeks and then disappears. Forms which begin with headache, pain and vomiting were called *ophthalmoplegic migraine* by Charcot. Its nature is unknown. It is probably peripheral in form (Mauthner), although Möbius thinks it is nuclear; von Monakow considers that its usual cause is tumor, meningitis or tubercle; the lesion affects the eye nerves (or centres) and also the descending branch of the fifth nerve; some few cases are seemingly hysterical.

Paralysis of the Fourth (Trochlear) Nerve.—The superior oblique muscle moves the eye downward and somewhat inward. Its paralysis is of little clinical importance. In attempts to converge downward the normal inward rotation of the eyeball is lacking.

Paralysis of the Sixth Nerve.—The eye cannot be moved outward, and is rotated inward by the unopposed internal rectus, marked *diplopia* resulting. This nerve is frequently involved, because of its long and exposed course. The muscles of the eye are involved in the following order of frequency: 1, ext. rectus; 2, sup. oblique; 3, inf. rectus; 4, sup. rectus; 5, int. rectus; 6, inf. oblique (von Gräfe).

Combined Eye Paralysis.—Convergence and accommodation are associated functionally and their centres lie adjacent, whence they are often lost together. The internal recti may fail to contract together but may move separately in lateral eye movements. *Conjugate deviation* may occur (see Brain Hemorrhage and Pons Localization); if a lesion occur above the sixth nucleus, the eyes cannot be directed toward the side of lesion and are, therefore, directed toward the paralyzed side; if the lesion is at the sixth nucleus the sixth nerve on that side is paralyzed, often also the seventh, and the opposite third nerve is paralyzed, not from lesion in the third nucleus but from the fact that each third nucleus receives afferent impulses from the contralateral sixth nucleus for associated eye movements.

Symptoms.—The *general symptoms of paralysis of the eye muscles* are five (Gowers):

1. *Limitation of movement* in the paralyzed muscle, followed by secondary contraction of the unresisted opponent. The habitual position of the head corresponds in every way to the physiological action of the paralyzed muscle (Landholt).

2. *Strabismus* (squint), causing lack of parallelism in the visual axes, which are divergent when the int. rectus and convergent when the ext. rectus is affected; this constitutes the *primary deviation.*

3. *Secondary deviation* is the extra movement of the sound eye in its efforts to see, due to its increased innervation, detected by testing with the diseased eye closed or by testing with ground glass over the sound one.

4. *Erroneous projection* in the paralyzed eye. Distance is normally judged by the position of the eyeballs and muscles and therefore when secondary deviation from excessive innervation of the sound muscles occurs, it gives the mind an impression of distance greater than is actually the case.

5. *Diplopia* (double vision) results from failure of the visual axes to correspond. The sound eye sees the true image, while the paralyzed eye sees the false one, which is less clear, since the image falls on a less sensitive retinal area. When the images are either near together or else widely separated the false one may be "neglected." Erroneous projection and diplopia always produce vertigo at first. If the false image is on the same side of the true image as the eye by which it is seen, the diplopia is simple or homonymous; if on the other side, it is crossed or heteronymous. The eyes are tested by holding red glass before one eye and testing vision with a strip of white paper. If the red image and covered eye are on the same side the diplopia is simple; if on opposite sides it is crossed. When the eyes converge it is simple, when they diverge it is crossed; *i. e.,* "when the prolonged visual axes cross, the diplopia is not crossed." Holding the test object stationary and the head being turned in one way or another, in some directions the images converge or perhaps blend, while in the opposite direction they separate. "The affected eye is that one in the direction of whose image the diplopia increases; the paralyzed muscle is the one which would give to the eye the direction of the false image" (Landholt). Monocular diplopia is almost invariably hysterical (unless there is disease of the eye media or error in curvature).

Treatment.—Treatment is often unsatisfactory from the etiological standpoint. Surgical interference is sometimes indicated. Syphilitic forms (*q. v.*) may respond to specific treatment, but often recur or become permanent. In subacute or acute forms counterirritation back of the ear by blisters, leeches or hot fomentations is indicated. Some find mercury useful in peripheral forms. Galvanism is the best form of electrotherapy, but may be painful and is seldom beneficial. Prisms and tenotomy are sometimes helpful.

Progressive Nuclear Ophthalmoplegia (Nuclear Palsy).—This disease was first described clinically by von Gräfe (1856) and later

pathologically by Hutchinson and Gowers. The etiology is not clear. Wernicke and Siemerling found syphilis causal in 17 per cent., which estimate is too low, according to Hutchinson. Lead, alcohol and toxæmia, as from diabetes, grippe or diphtheria, are possible causes. A few cases are hereditary or congenital. Most cases develop in males under thirty years of age. The *pathology* usually consists of degenerative nuclear atrophy resembling bulbar paralysis; sometimes slow compression or meningitis is found or much more rarely no anatomical alteration is detected, the so-called functional form.

Symptoms.—The symptoms are those of a slowly progressive paralysis of the eye muscles; ophthalmoplegia is called external when it involves the extrinsic muscles, internal when it involves the pupil, or total when both are concerned. The *eye muscles* are *successively affected,* but in no given order, until many or all are implicated. The *facies of Hutchinson* is peculiar, staring, from immobility of the eyeballs, or sleepy from partial bilateral ptosis. Diplopia is uncommon, or only an early symptom, because of the chronicity of the process. Associated muscles are often affected by groups, because of their successive nuclear involvement (*v. s.* Anatomy); as the pupil and convergence, one internal and the opposite external rectus, the two internal recti and the sup. rectus and the levator. Chronic ophthalmoplegia may be associated with the analogous *bulbar paralysis* or *progressive spinal muscular atrophy;* with *optic atrophy* or with *double facial paralysis* (the rare and obscure infantile or congenital oculofacial paralysis, without pupillary involvement, of which Möbius has collected 44 cases); it may be a part or a precursor of tabes, paralytic dementia or multiple sclerosis.

Diagnosis.—Nuclear involvement is probable when there is (*a*) partial and bilateral ptosis; (*b*) no involvement of the pupil; or (*c*) involvement of one or more conjugate groups. The lesion may be supranuclear, when the head and limbs are involved, exceptionally when there is isolated convergence paralysis or paralysis of up-and-down movements with ptosis. Sudden apoplectiform nuclear palsy from basilar thrombosis is usually asymmetrical, acute and often associated with hemiplegia. Wernicke's acute superior poliencephalitis is distinguished by its acuity and toxæmic symptoms. Ophthalmoplegia may result from syphilitic meningitis, polyneuritis, tumors (corp. quadrigem.) and orbital disease, but they are generally distinguished by their relatively acute course and concomitant symptoms.

Prognosis.—The disease is very chronic, possibly lasting twenty to forty years and is progressive. Arrest is possible, but can never be predicted. The disease itself causes little risk to life, but its associates are dangerous, as tabes or psychoses, which develop in 18 per cent.

Treatment.—Treatment is generally ineffectual. Iodides and mercury are useless in nuclear disease, but may be given if the localization is not definitely known. Strychnia and galvanism produce little result.

Sympathetic Paralysis.—This disease was first described by Hor-

ner; it produces *myosis,* due to paralysis of the dilator iridis, and slight *ptosis,* due to paresis of Müller's muscle. The ocular tension is decreased, the cheek wasted and the skin reddened and warmer than normal. *Irritation of the sympathetic* is characterized by wide pupils, widening of the interpalpebral space and by exophthalmos.

Ocular Muscular Spasms.—Ocular muscular spasms occur in conjugate deviation, irritation of their nerve trunks when compressed by tumor, and rarely in chorea, facial tic and spasmodic torticollis. In hysteria the eyes may be rolled upward and to one side, concealing the iris, or they are often fixed in convergence; they never diverge; there may appear to be ptosis, which is really spasm of the orbicularis. Convergent spasm may also occur in hypermetropia and divergent spasm in myopia. Isolated spasm of the levator palpebræ may occur in irritation of the cervical sympathetic nerve, in advanced years, in Graves' disease and in irritation of the fifth nerve. Secondary deviation (*v. s.*) is spasmodic, as is also *nystagmus,* which latter consists of rapid, rhythmic, clonic, involuntary oscillations, due to muscular spasm with alternating contraction of the opposing muscles. It is continuous, as distinguished from the "tremor of weakness" which occurs in paretic muscles on movement. It is usually horizontal, less often rotatory and but rarely vertical. Its physiology is not understood, but it is probably centric. The *causes* are (*a*) disease of the eye, as opacities and choroiditis; (*b*) it occurs very frequently in albinos, and (*c*) in miners, from working with the head down and eyes strained to one side. (*d*) It also occurs in numerous, usually degenerative nervous diseases; as a cardinal sign in multiple sclerosis and Friedreich's ataxia, as a common sign in tumor of the cerebellum, corp. quadrigem. and opt. thalamus, occasionally in meningeal inflammation or hemorrhage, sinus thrombosis, brain hemorrhage or softening and very rarely in hysteria.

Fifth Nerve (Trigeminus; Trifacial Nerve).—The nerve originates from its motor nucleus in the pons, from fibers (which are probably motor) descending from beneath the corpora quadrigemina, and from sensory fibers ascending from the medulla. It is connected with the motor cortex and with the cerebellum. The motor and sensory fibers leave the pons separately and join only after the sensory trunk has passed through the Gasserian ganglion. The Gasserian ganglion has three branches; (1) which passes to the orbit by the sphenoidal fissure, supplying the skin of the forehead, anterior scalp, upper lid, bridge and top of the nose; it is connected with the cervical sympathetic nerve; (2) which passes by the foramen ovale to the sphenomaxillary fossa to supply the upper and lower lids, cheek, fore part of the temples, side of the nose, upper teeth, gums, pharynx, tonsils, soft palate, uvula and roof of the mouth; it has connections with the facial nerve by the Vidian and with the sympathetic nerve; (3) which passes by the foramen rotundum and sphenomaxillary fossa supplying the rest of the temples, anterior and upper part of the

ear, auditory meatus, lower cheek near the mouth, lower lip, lower teeth, gums, chin, tongue, mucosa of the mouth, salivary glands, and as a motor nerve to the muscles of the lower jaw, temporals, masseters, pterygoids, mylohyoids and post. belly of the digastric muscle. The lingual branch gives off the chorda tympani which joins the facial nerve. From the otic ganglion arises the small superficial petrosal nerve which connects with the facial nerve and ends in the tympanic branches of the glossopharyngeal nerve.

Causes of Paralysis.—(*a*) Lesions of the cortex in very rare cases. (*b*) Focal disease in the pons, especially of the nerve-root fibers, as hemorrhage and tumors and much less often degenerative lesions, as tabes or bulbar paralysis. (*c*) Diseases of the base, as syphilis, meningitis, tumors, caries or aneurysms. (*d*) Affections involving any of the three branches, as of the first branch by cavernous thrombosis, pituitary tumor, aneurysm or orbital disease, or of the second and third branches by lesions in the sphenomaxillary fossa. (*e*) Trauma, puncture or bullet wounds, but rarely fractures, and (*f*) infrequently neuritis, unless it is secondary.

Symptoms.—1. Sensory Portion.—Anæsthesia, generally unilateral, is found in one or all branches, and tactile sensation is more involved than the sense of pain. It is often preceded by tenderness along the nerves, irritation or neuralgia. In complete cases the skin and mucosa of the mouth, nose and conjunctiva are anæsthetic. The tongue is furred on its anæsthetic half, because the food is not chewed on that side. The conjunctival reflex is abolished. The secretions are increased by irritation or decreased by paralysis. Smell is blunted and taste is lost (*v. i.*). Sometimes there is redness, cyanosis or swelling of the face or the teeth may be loosened. Two trophic manifestations are especially important. The first is neuroparalytic ophthalmia, which may run beyond simple inflammation of the eye into ulceration or complete loss of the bulb; it is due not simply to conjunctival anæsthesia, which may lead to inflammation because of foreign particles remaining unnoticed in the eye, but is rather due to irritation of the fifth nerve, especially of the Gasserian ganglion or nerve trunks anterior to it. (It has not occurred, however, in many cases in which the ganglion has been surgically removed.) It is rare in lesions of the pons. The second is herpes zoster, which is most common in disease of the first branch, results from irritation of the Gasserian ganglion or the nerves just anterior to it, is often preceded by pain and hyperæsthesia and sometimes leads to iritis or ophthalmitis. For the relation of trigeminal disease to facial hemiatrophy see below.

2. Motor Portion.—Placing the fingers on the temporal and masseter muscles, when the patient attempts to close the teeth, reveals their failure to contract. Paralysis of the ext. pterygoid is shown (*a*) by failure to move the jaw toward the sound side and (*b*) by deviation toward the paralyzed side, when the jaw is depressed. The muscles waste, but they are too deep to distinctly elicit the reaction of de-

generation, and the role of the tensor tympani and tensor palati in trigeminal paralysis is still in dispute.

3. Gustatory Portion.—The sense of taste covers only bitter, sweet, sour, salty and metallic substances, while flavors are recognized by the olfactory nerve. The tip and edges of the tongue detect saltiness or sourness; the base of the tongue, bitterness and sweetness. It is often stated that the fifth nerve supplies the anterior two-thirds of the tongue and the glossopharyngeal nerve the posterior third and the palate; but disease of the glossopharyngeal nerve alone does not destroy the sense of taste. In a study of 26 cases of extirpation of the Gasserian ganglion, Cushing decides that taste is only temporarily lost and that it does not travel by the fifth nerve. Disease of the lingual nerve (from the fifth), after the chorda tympani joins it, produces loss of taste (*ageusia*); disease of the lingual above the juncture or disease of the central part of the fifth in the pons does not result in loss of taste. It is thought that the gustatory fibers of the fifth run with the chorda to the seventh nerve which they leave by the petrosal nerve to again reach the fifth through Meckel's ganglion. It is interesting to note that anæsthesia of the tongue may result from facial neuritis and disease of the middle ear.

Diagnosis.—The diagnosis is usually easy when there are motor or sensory symptoms. Organic irritation characterized by pain alone may be difficult or impossible to differentiate from trifacial neuralgia, although Gowers holds that there is less radiation and more hyperæsthesia in organic irritation. *Localization:* When all branches are anæsthetic the disease probably lies at the base of the brain or in the Gasserian ganglion. In disease of the middle part of the pons the first division alone may be affected; in disease at its side all divisions are involved, often with implication of the third and sixth or less often of the seventh and eighth nerves. Cortical lesions are extremely uncommon.

Treatment.—Counterirritation over the occiput or mastoid is indicated by an inflammatory causation, but no irritant should be applied over the anæsthetic areas themselves, lest ulceration occur. Galvanization is said to be beneficial. Gelsemium, cocaine and morphine must at times be used because of severe pain, but the acquisition of drug habits must always be borne in mind. The eye should always be watched and boric acid and castor oil be dropped in several times daily. Antisyphilitic remedies should be tried in every doubtful case.

Masticatory Spasm.—The masticatory spasm of Romberg is not common. In the *tonic type* (trismus) the spasm is usually bilateral and the tense muscles are hard and may be tender. The cause may be tetanus, hysteria, epilepsy, tetany, tumor in the pons, cortical hemorrhage, or it may be reflex from trigeminal irritation, as from dental caries. The *clonic type* is rare and may be serial or intermittent; chattering of the teeth is an example. It may occur in chorea, epilepsy or hysteria. Grinding of the teeth occurs in paretic dementia,

meningitis or typhoidal states. In *diagnosis,* tumor, inflammatory irritation and disease of the temporomaxillary joint must be considered. *Treatment* consists of tonics in nervous subjects, galvanization, blisters in hysteria and the local use of morphine and cocaine.

Progressive Facial Hemiatrophy.—This affection, first described by Parry (1825), is rare, only 135 cases having been reported up to 1900. Its pathogenesis is not clearly understood, but it is classified under disease of the fifth nerve because the only autopsy on record, by Mendel, showed neuritis of the ascending root of the fifth nerve. Möbius holds that acute infections, as measles or scarlatina, are causal, because some virus enters through the skin or tonsils and results in atrophy. The essential change is a *facial hemiatrophy,* which develops most often in persons between ten and fifteen years of age, in girls (66 per cent.) and in about 60 per cent. of cases affects the left half of the face. In perhaps a half-dozen cases the atrophy was bilateral. There may be premonitory pains, hyper- or paræsthesias, but the cardinal sign is *atrophy of the skin,* which may show initial infiltration. The skin is decolorized and shrunken, its *fat disappears,* and later in the disease the *muscles waste* and the rough skin lies directly on the bone. The hair on the affected side loses its pigment and may fall out, the eyebrows and beard especially, sweat secretion is lessened or absent and the mouth, if involved, is drawn toward the side of lesion. The *bones* on the affected side *waste,* especially in young subjects, most notably the maxillæ, but even the smaller bones and nasal cartilages are involved. On the wasted side there may be no vasomotor reaction; sensation is usually normal, although pain or paræsthesia may be noted in the distribution of the trifacial nerve. The tongue, tonsils and palate are rarely involved. The course is *very chronic* and practically always progressive, although remission is possible. The outlook is hopeless and there is no treatment other than administration of thyroid extract and the injection of paraffin to lessen the facial deformity.

Facial Hemihypertrophy.—Facial hemihypertrophy, of which 13 cases only are on record, affects largely the cheek; Werner collected 9 congenital cases. Irritative neuritis is its suspected cause.

Seventh or Facial Nerve.—The face is represented in the lower Rolandic cortex, whence the fibers run through the corona, internal capsule, crus and pons (*q. v.*), where they decussate and enter the nucleus of the opposite side. The nucleus lies in the floor of the fourth ventricle inside of the ascending branch of the fifth nerve and receives fibers from the third nucleus above for the orbicularis of the eye (whereby ocular movements, winking and accommodation are coördinated) and fibers from the hypoglossus below for the orbicularis oris (whereby correlation of lingual and labial movements is affected). It curves around the sixth nucleus, emerging near the juncture of the pons and medulla, and enters the auditory meatus with the eighth nerve, bends sharply, and presents a ganglionic

swelling which receives the large superficial petrosal from the Vidian nerve, containing fibers of taste from the fifth nerve. These latter again leave the facial nerve by the chorda tympani. In the Fallopian canal the facial nerve gives off branches as follows: (*a*) a motor branch to the tympanic plexus; (*b*) a motor twig to the stapedius muscle; (*c*) the chorda tympani nerve with secretory branches to the salivary glands. Passing through the stylomastoid foramen, it supplies (i) a few sensory branches to the external ear; (ii) motor fibers to the external auricular muscles; (iii) motor fibers to the posterior part of the occipitofrontalis, and in the parotid gland, (iv) breaks up into branches which supply all the muscles of the face, the platysma, stylohyoid and post. belly of the digastric.

Peripheral Facial Paralysis.—Peripheral facial paralysis (mimetic facial paralysis, prosoplegia, Bell's paralysis, Bell, 1830) is the most frequent peripheral palsy.

Etiology.—1. *Neuritis* is the most common cause. Exposure to cold ("rheumatism") produces 73 per cent. of cases, especially in men between the twentieth and fortieth years, and produces a perineuritis which compresses the nerve trunk. Angina, gout, diabetes, diphtheria, typhoid, erysipelas, mumps, sepsis, etc., are occasional causes.

2. Disease of the petrous portion of the *temporal bone* (7 per cent.), especially caries and otitis media in childhood, may cause paralysis. The danger is proportionate to the extent of the bone disease, but infective matter may reach the nerve by way of the vessels; 88 per cent. of cases of disease of the labyrinth cause facial paralysis.

3. *Trauma* causes 6 per cent. of cases, as basal fracture by tearing, hemorrhage, inflammation or undue formation of callus; much more rarely meningeal or cerebellar hemorrhage at birth; the use of obstetrical forceps and trauma to the jaw or surgical operations on the parotid gland. Facial paralysis may occur in "head tetanus" (Rose).

4. If it results from lesions *at the base,* syphilis (3 per cent.) should always be considered when the cause is intracranial; syphilis rarely causes a peripheral facial paralysis. Aneurysm, tumor, meningitis and hemorrhage are next in frequency and nearly always involve simultaneously the closely contiguous eighth nerve.

5. *In the pons* the root fibers or nucleus may be affected, usually with the sixth nerve of the same, or the arm and leg of the opposite side. Degenerative affections, as tabes, bulbar paralysis, multiple sclerosis, and progressive muscular atrophy or acute poliencephalitis rarely invade the seventh nucleus.

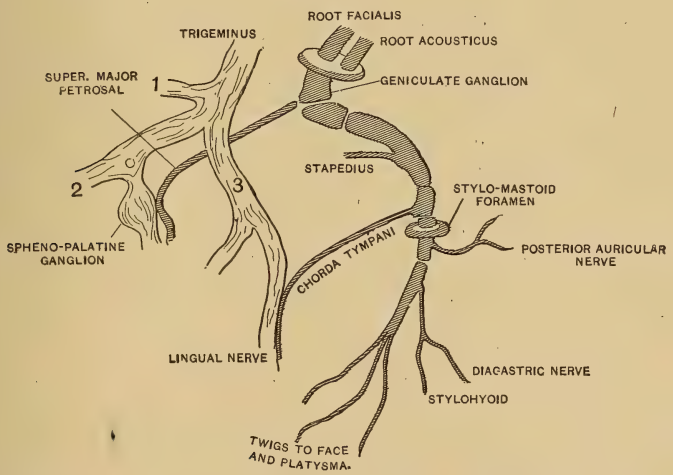
6. Other rarer factors are primary hemorrhage into the Fallopian canal, disease of the parotid or lymphatic glands and leukæmic infiltration. It is suggested by Neumann that disease of the lymph vessels at the point of exit of the facial nerve may produce paralysis.

Pathology.—Few autopsies have been made on simple cases. The nerve has been found severed, its sheath and cylinders destroyed, the nuclei in Schwann's sheath increased, the connective tissue increased and the muscles the seat of fatty change. It is thought that the essen-

tial and usual change is a *perineuritis* and that the exudation in the sheath compresses the nerve fibers, which then undergo parenchymatous degeneration. In some cases degeneration only has been seen and it is probable that slight swelling interferes with conduction. Facial neuritis differs from multiple neuritis in that alcoholism is a subordinate factor and the chief change is not in its peripheral twigs but in the trunk, usually within the Fallopian canal.

Symptoms.—The symptoms sometimes begin with prodromes, as vertigo, headache, tinnitus, pain or more often twitchings in the face, even fever or albuminuria, or sometimes without prodromes, in which cases one *half of the face* is rather suddenly paralyzed and *"completely immobile and toneless"* within two days. The forehead is smooth, the lids cannot close (lagophthalmos), the tears run over the cheek and when a strong effort is made to close the eyes the eye on the affected side is turned upward by the inf. oblique muscle (Bell's sign). The orbicularis at times escapes involvement, since it receives some fibers from the third nerve. Smell is impaired because of absence of tears, the nasal aperture is smaller and cannot be dilated, sniffing on the affected side is impossible and the nasolabial furrow is lost. The mouth is lower from paralysis of the levator anguli oris, it cannot be closed and the saliva runs out, although its total secretion is often lessened (*v. i.*). Labial enunciation is imperfect, puckering the

FIG. 77.



Relations of the fifth and seventh nerves and branches of the seventh.

lips, as though for whistling, is impossible and expectoration is impaired. The loss of wrinkles and expression on the affected side is more marked in adult and aged than in young subjects, especially about the eye, since years lessen the elasticity of the skin and the subcutaneous fat. Chewing is impaired by weakness of the buccinator and food accumulates between the teeth and the cheek. The unused

half of the tongue becomes furred. The tongue does not deviate on protrusion (but may seem to, on account of asymmetry of the mouth). The tongue is sometimes slightly depressed from weakness of the stylohyoid and digastric muscles. The platysma paralysis is elicited by asking the patient to depress the lower lip. The palate is sometimes described as sagging, the speech as nasal, the uvula as deviating towards the sound side, but the uvula and palate are often asymmetrical in health and there is a growing tendency to regard the spinal accessory as the motor nerve for these structures.

Taste is lost in disease of the nerve between the geniculate ganglion and the offset of the chorda; it is lost in half the cases of "exposure" neuritis; it is not impaired in disease of the pons or in disease outside the skull (see DIAGRAM). The chorda supplies the anterior two-thirds of the tongue and the glossopharyngeal the posterior third. Perverted taste (parageusia) results from irritation of Jacobson's nerve or the tympanic plexus. *Hearing* may be impaired. Deafness and tinnitus result mostly from coincident disease of the ear. When the stapedius is paralyzed, the unopposed tensor tympani (which arises from the fifth nerve) overacts and results in aural oversensitiveness especially to low notes (Lucae), which is termed hyperakusis or oxyokoia.

All facial reflexes are abolished. Wasting may be noticed in thin subjects and the *reaction of degeneration* appears in a week or two in severe cases. In cases of moderate severity the excitability may be first increased and then gradually decreased in seven to ten days. Sensation is usually normal, since the facial is an almost wholly motor nerve; pain, even anæsthesia in the ear and tenderness over the nerve trunk are sometimes observed; they are caused either by involvement of the fifth nerve or possibly because the facial nerve carries some sensory filaments. Accidental sympathetic œdema and sweating have been observed. Herpes is rare and indicates trifacial involvement. The saliva is decreased (the fibers for its secretion run with the chorda) and secretion of tears is lessened (the fibers from the facial nerve run to the fifth by the large petrosal nerve).

Diagnosis.—The diagnosis is made at a glance in fresh cases, while in older cases secondary spasm (*v. i.*) on the paralyzed side may at first cause confusion. The etiological diagnosis is important, as is also the topographical.

1. *Supranuclear paralysis,* paralysis in the upper neurone, from the cortex to the nucleus, is differentiated as follows:

CENTRAL OR SUPRANUCLEAR PALSY.	*vs.*	PERIPHERAL (NUCLEAR AND INFRANUCLEAR) PALSY.
Etiology; vascular disease, tumor, abscess.		Neuritis, ear disease, etc.
Distribution of paralysis; partial, lower facial involved or only slight or transient weakness of orbicularis palpebrarum.		*Complete;* orbicularis and occipitofrontalis involved. When eyes cannot be closed, probably peripheral or nuclear.

Central or Supranuclear Palsy.	*vs.*	Peripheral (Nuclear and Infranuclear) Palsy.
Reflexes persist and increased.		Lost. Paralysis flaccid.
Taste, auditory, salivary and sensory symptoms lacking.		Often present.
Pilocarpin increases salivary flow.		Does not increase it.
Voluntary motion lost, while emotional movement very often persists.		Both lost.
Degenerative reaction and wasting absent.		Present.
Cerebral symptoms present, general as headache, increased reflexes or focal as cortical epilepsy, hemiplegia, usually weak tongue, alternating paralysis (see crus and pons localization).		Absent.

In lesions of the pons all parts of the facial nerve are affected, wherein it differs from facial nerve disease in the internal capsule. In nuclear disease the orbicularis oris is less involved or escapes affection, its centre being in the hypoglossus nucleus.

2. *Localization in the peripheral type* (see Fig. 77) may be (*a*) in the *pons* (*q. v.*). General disease of the posterior pons also affects the sixth nerve about whose nucleus the seventh nerve curves. (*b*) In localization *at the base,* taste is not involved and deafness results from coincident disease of the eighth nerve. (*c*) If in the *Fallopian canal* taste is impaired, the salivary flow decreased and hearing often abnormally acute. (*d*) If *external to the skull* there is no involvement of taste, salivary flow or the stapedius.

Double facial paralysis is rare and usually indicates disease within the skull. It may occur in bulbar paralysis, disease of both sides of the pons or one focus in its centre, double otitis, especially syphilis, multiple neuritis, tetanus (of which J. H. Lloyd in 1905 collected 7 cases) or in a form observed in childhood with ocular paralysis (*q. v.*). Hysterical facial paralysis is extremely infrequent; organic facial paralysis may exist on one side and an hysterical paralysis on the other.

Course and Prognosis.—The onset is rapid and the disease is usually manifest during the first day. Its duration varies from a week to over a year, two to three months for moderate, and six to eight months for severe cases. Even in cases which have seemingly recovered and in those lasting over a month, some slight vestige of paralysis usually remains. The *prognosis* depends (1) on the cause; it is favorable in syphilis, birth trauma and polyneuritis; in ear disease it is better if the drum has not ruptured; it is unfavorable in tumor or nuclear disease; (2) on the electrical reaction. If after ten days the irritability is not reduced the outlook is good and recovery may be expected in about three weeks; if after fourteen days it is lowered but not lost, recovery occurs within two months; if after fourteen days the

irritability is lost, the prognosis is doubtful and the course is likely to cover months. Recurrence is infrequent, although five attacks are recorded. *Contracture* (secondary overaction) usually occurs in four to six months and progresses for eight to twelve months, when it lessens and disappears in mild but remains to some extent in severe cases. In old subjects it balances up the facial contour; in young subjects it produces deformity. It is thought to depend on an altered functional state of the nucleus.

Treatment.—(*a*) The *cause* should be reached, if possible; antisyphilitic remedies and ear-drainage have cured cases even of twenty-three years duration. (*b*) *Antirheumatic* drugs are inefficacious and the salicylates do not usually shorten the course. A mercurial purge, hot fomentations, which must be carefully maintained for two or three days, keeping the patient indoors, and the invariable application of a blister over the mastoid and one over the side of the neck are beneficial. (*c*) *Correction of deformity* is always necessary. The eye should be douched with boric acid, bandaged closed during sleep and the lids massaged, the cheek and mouth should be massaged upward and the placing of a hook in the angle of the mouth (sustained by tape over the ear or by adhesive plaster) prevents deformity to some extent. (*d*) *Electricity* should not be used for two or three weeks, and then the galvanic current should be given, for fifteen minutes each day, in strength just sufficient to produce muscular contraction; the application of the anode over the muscles is the least painful method. The faradic current causes pain. (*e*) For *contracture* little can be done, excepting massage and steaming the face. (*f*) In irreparable cases, surgical transplantation of the facial trunk into the spinal accessory nerve has been successfully performed. Spiller thinks anastomosis with the hypoglossus produces better results.

Facial and Mimetic Spasm (Tic Convulsif).—Etiology.—The causes are (*a*) organic disease in the cortex, pons or nerve trunk, which irritates (perhaps later paralyzes) the centre, nucleus or paths. Irritation of the fifth nerve is considered causal. (*b*) There is an idiopathic form, which occurs in persons between the fortieth and sixtieth years, most often in women with neuropathic or emotional tendencies (see HABIT SPASM under CHOREA). (*c*) There is also a reflex form which results from worms or uterine disease.

Symptoms.—The spasm begins paroxysmally on one side and is more often clonic than tonic. The *clonic form* affects the orbicularis palpebrarum (blepharospasm) and zygomatici most frequently, although the frontalis, chin depressors or all the facial muscles may be concerned in severe cases. Pain and paresis are absent save in progressive organic disease. The spasm is lessened by rest and increased by cold or emotion. It is usually unilateral, but may spread to the opposite side or exceptionally to the muscles of mastication or the tongue, pharynx, larynx, neck or arm. The *tonic form* is seen in photophobic disease of the eye, paralysis agitans, tetanus, tetany or hysteria.

Prognosis.—The prognosis is unfavorable after the first few months and the disease is then likely to last for years or for life.

Treatment.—Treatment is often useless and varies with the cause of the affection. Trigeminal irritation should be removed if possible. Arsenic has proved beneficial in some cases, but nervines and antispasmodics generally are useless. Galvanization, if tender points are found, has cured a few cases (Berger). Weir Mitchell has recommended freezing of the cheek with ethyl chloride. Stretching of the facial nerve usually affords relief, which is generally but temporary.

The Eighth or Auditory Nerve.—The eighth nerve in the old nomenclature was the portio mollis of the seventh pair, the facial nerve being the portio dura; as the names imply, the auditory is softer than the facial nerve and therefore less resistant to pressure. It is purely sensory and consists of two distinct roots, the cochlear and vestibular. At the point where the roots separate is found a group of cells, analogous to the spinal ganglia, from which arises Wrisberg's nerve, an accessory structure with vasomotor and salivary secreting fibers.

The *cochlear nerve* is the true auditory nerve and its nuclei are found in the floor of the fourth ventricle. It is connected through the tegmentum of the crus and internal capsule with the contralateral and to a less degree with the homolateral temporosphenoidal lobe. It is distributed to or arises from the cochlea and ganglionic cells of the organ of Corti. (*a*) *Cortical disease* (see Brain Localization) may in rare cases cause word deafness, as may (*b*) *disease of the auditory tracts* by lesions of the lateral lemniscus, the post. corp. quadrigemina, internal capsule or less often of the pons. Nuclear disease is almost unknown. Neuromata are rare, but occur on the auditory trunk oftener than on any other cranial nerve. The nerve trunk may degenerate in tabes. (*c*) *Lesions at the base* may follow trauma and meningeal inflammation, hemorrhage or syphilis. (*d*) Most auditory nerve diseases result from *disease of the internal ear,* either primary or secondary to disease of the middle ear; these affections include acute or chronic inflammation, syphilis, degeneration or hemorrhage in the labyrinth. There are three prominent *symptoms:* (*a*) *Nervous deafness may result.* Deaf-mutism is congenital in 80 per cent. and is acquired in early life in 20 per cent. of cases. Deafness from occlusion of the meatus or from disease of the middle ear is excluded by Rinne's test; a vibrating tuning-fork or watch, if held over the meatus, is not heard in these affections, but is heard when its base is placed on the mastoid which conducts vibrations to the internal ear. In disease of the internal ear, bone-conduction is lacking. Differentiation between deafness due to disease of the internal ear and that due to bone disease is only made by means of the associated symptoms. The *treatment* of nervous deafness is etiological. Iodides are sometimes beneficial, as are full doses of pilocarpine. Electricity is useless. (*b*) *Auditory hyperæsthesia* may result from central or peripheral disease. In genuine hyperæsthesia (hyperacusis), sounds

are heard by the patient which other individuals may not hear; this occurs in hysteria, epilepsy, migraine, meningitis, acute mania and at times after use of caffeine, alcohol or opium. The spurious hyperacusis results from paralysis of the stapedius. In dysæsthesia (dysacusis) ordinary sounds cause disproportionate sensations or discomfort; this is common in headache and cerebral disorders. (*c*) *Tinnitus aurium* designates subjective distressing and intractable sensations, as ringing, roaring or whistling in the ears. In 80 per cent. of cases it is caused by disease of the internal ear. It may also occur in any disease of the middle ear, when there is cerumen or foreign bodies in the external meatus, or occlusion of the Eustachian tube, when the individual is subject to long exposure to loud sounds, as in boiler factories, and in the neuroses (hysteria, neurasthenia, migraine, epilepsy), aneurysm, anæmia, gout or cinchonism. Sounds of centric origin are usually continuous and sometimes elaborate. Wax or bilateral labyrinthine disease may also cause continuous tinnitus, while in anæmia or aneurysm, these sensations are intermittent (Gowers). Hearing may be hyperacute, deranged or lost. Spasm of the palate or musculature of the Eustachian tube may produce a most annoying snapping sound, which is at times audible to bystanders. While nothing is usually heard on examination, in children or less often in adults we may hear a systolic brain murmur (see Brain Tumor). *Treatment* depends on the cause. Bromides are most useful, combined with a few drops of the tr. belladonna or cannabis indica. Quinine was recommended by Charcot and Politzer and salicylates by Gowers, but they seldom give relief. Full doses of nitroglycerine relieve certain cases and counter-irritation over the mastoid sometimes mitigates tinnitus.

The vestibular nerve arises from the medulla, supplies the vestibule and semicircular canals, subserves sensation of space and equilibration and has cerebral and cerebellar connections.

Ménière's Disease, Auditory Vertigo (Vertigo ab aure læsa).—Paul Ménière in 1861 described a condition consisting of vertigo, tinnitus, vomiting and sometimes deafness, developing with apoplectiform suddenness and due to acute labyrinthine disease. The lesion is usually a slightly irritative disease of the vestibular nerve, which supplies the organs of equilibration, the semicircular canals. According to Gowers 80 per cent. occur in persons between the thirtieth and sixtieth years, 66 per cent. in males, and 90 per cent. of all cases of vertigo not due to epilepsy or organic brain disease are caused by lesions of the labyrinth. Fränkl-Hochwart thus classifies Ménière's complex, which may be elaborated as follows: (*a*) *Apoplectiform cases,* which occur in healthy ears, of which few more than 30 cases are recorded, due to labyrinthine hemorrhage or to trauma; leukæmia and arteriosclerosis are promoting factors and it is thought that acute angioneurotic transudation of lymph may occur in the canals. (*b*) Cases developing in *ears already acutely or chronically diseased;* the internal ear is the usual seat of disease, but the external or middle

ear may be affected or the branches of the eighth nerve, perhaps by tumors, syphilis, tabes, paretic dementia, gout, cold, facial paralysis or drugs, such as quinine and the salicylates. (*c*) Cases caused by *external influences* or procedures, as pressure on the drum by violent douching of the ear; (*d*) *pseudo-Ménière's disease,* which occurs in the neuroses, as epilepsy, hysteria, neurasthenia, migraine or sea-sickness.

Symptoms.—The symptoms begin gradually or with a sudden stroke and are paroxysmal; the attacks may be separated by weeks or months or perhaps occur daily. (*a*) The *vertigo* is subjective or objective, perhaps combined; the patient cannot stand; he is likely to fall backward or if standing is possible is ataxic. These symptoms indicate lesion in the *vestibular nerve;* there is occasionally momentary loss of consciousness; some slight vertigo may persist between paroxysms. (*b*) *Nausea and vomiting* are usual, save in the lighter forms and are often attended by headache, sometimes collapse, pallor and clammy skin and rarely by death. (*c*) The *tinnitus* of a roaring, throbbing character and (*d*) *nervous deafness,* which is usually not absolute, indicate disease of the *cochlear nerve.* Nystagmus, double vision and ataxia are occasional symptoms and result from the relations between the labyrinth, cerebellum and cerebrum. The *prognosis* is always uncertain. Acute or progressive disease is unfavorable. The vertigo may cease when complete destruction of the nerve results in deafness. Some cases recover.

Diagnosis.—Aural vertigo constitutes 90 per cent. of vertigo. Differentiation is as follows: (*a*) *Gastric disease* may cause vomiting or *vertigo* (*a stomacho læso, Trousseau*) but does not produce tinnitus or deafness. Gastric vertigo was once thought to cause 80 per cent. of cases of vertigo; now, but 5 per cent. are attributed to it (Gowers). Many of these cases are neurotic. (*b*) *Epilepsy,* especially *petit mal,* may produce vertigo, very rarely tinnitus or deafness and much oftener loss of consciousness. (*c*) *Cardiac disease,* as aortic regurgitation or arteriosclerosis, may cause vertigo but not the other symptoms. (*d*) *Organic brain affections,* as tumor, have other distinctive signs, as headache or focal lesions. Confusion is possible with arteriosclerotic vertigo, and especially with coincident accidental deafness. (*e*) *Ocular vertigo* ceases on closing the eyes. Gerlier (1887) described a form which is endemic in France and Switzerland and is characterized by vertigo, great depression, paretic weakness of the neck and limbs, ptosis, strabismus, diplopia and retinal hyperæmia, hemorrhage or œdema. A similar form occurs in Japan. Recovery occurs in one to four months.

Treatment.—Labyrinthine disease is little influenced by drugs, except when syphilis and gout are its causes. Quinine, salicylates and nitroglycerine should be given (see TINNITUS). *Bromides* are most useful in daily doses of one dram. Galvanization with the anode over the ear and cathode to the neck has seemingly helped some cases, as has blistering of the mastoid. Disease of the pharynx, Eustachian

tube and accumulation of wax must be recognized and treated. Removal of the malleus and incus with mobilization of the stapes relieves a few cases.

Ninth or Glossopharyngeal Nerve.—This nerve is almost inseparably connected in its course and functions with the vagus and the internal part of the spinal accessory nerve; their nuclei blend, their sensory and motor filaments supply the digestive tract from the spinal accessory nerve and send motor branches to the lungs, larynx and heart. Compared with disease of the first eight cranial nerves, affections of these visceral nerves are infrequent. Of all the cranial nerves, the ninth is least understood and is almost never the seat of isolated disease. The causes of its disease are essentially the same as those of the vagus (*v. i.*); its nuclei suffer in bulbar palsy and its roots in basal disease.

It is distributed as follows: (*a*) its sensory fibers supply the back of the tongue, pharynx, soft palate, tonsil, upper pharynx, Eustachian tube and tympanic cavity. It is probably the nerve of nausea. (*b*) Its motor fibers supply the upper pharynx and possibly the palate. (*c*) Taste fibers are usually thought to be distributed to the posterior third of the tongue and palate, but they are not found in the nerve root and are considered to pass through the fifth nerve to the brain. Increased flow of saliva and loss of taste on the root of the tongue may result from disease of the middle ear due to relations between the nerve of Jacobson (ninth) and the branches of the fifth nerve. The nerve is thought to regulate and inhibit the contractions of the pharynx, which become frequent (spasm) in disease or on section of the nerve.

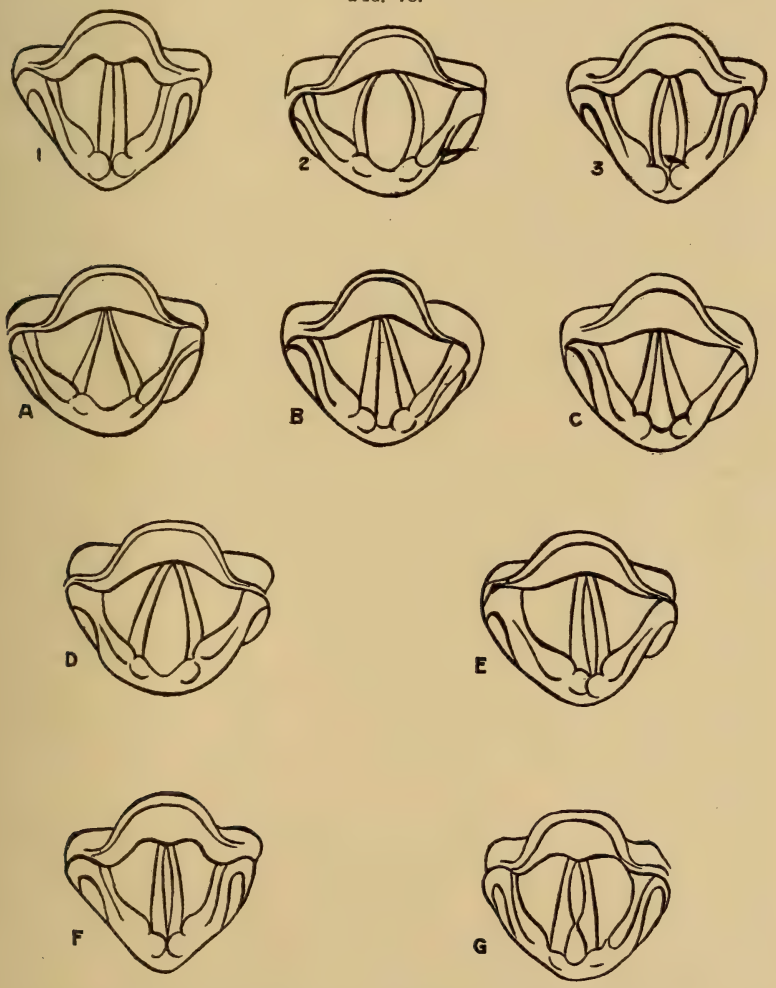
Tenth or Vagus Nerve.—This nerve arises in the medulla above the ninth, below the eleventh and outside of the twelfth nuclei. With the internal part of the accessory nerve it has an enormously diffuse and important visceral distribution. It is a mixed nerve, the motor fibers originating from the nuclei and the sensory fibers from the jugular and plexiform ganglia. It is generally maintained that the vagus carries the sensory and the accessory, motor fibers.

Etiology.—Affections of the vagus result from: (*a*) Nuclear disease, as degenerative bulbar palsy or acute vascular lesions, which usually also involve other nuclei; (*b*) involvement of the root and trunk by meningitis, syphilis, caries, trauma, tumor, aneurysm of the arch or its branches, carcinoma, œsophageal growths, tuberculous or other adenopathies, pleural (right-sided especially) or pericardial effusions or adhesions, mediastinitis, goitres, operations on the neck or rarely neuroma and neuritis from diphtheria or other infections, tabes or plumbism. Cortical, especially functional, causes may occasion symptoms. Irritative and paralytic manifestations occur in numerous combinations and will be treated topographically.

Pharyngeal Branches.—The pharyngeal plexus consists of branches

from the ninth and tenth nerves. In *paralysis,* sensation and the pharyngeal reflex are lost; bilateral disease causes dysphagia. The stomach tube can be introduced without sensation or spasm. Pulpy foods are better swallowed than dry foods or fluids. Food is likely to enter the larynx and when the palate is involved there is regurgitation into the nose and nasal speech results. The innervation of the palate is not definitely known, but it is probably supplied by the accessory nerve. *Spasm* is not frequent, is always functional, is transient or recurrent, does not allow of introduction of the stomach tube and

FIG. 78.



THE LARYNGEAL PARALYSES. (From Church's Nervous Diseases.)

1, normal phonation; 2, normal deep inspiration; 3, normal cadaveric position; A, bilateral adductor palsy; attempted phonation; B, left adductor palsy; attempted phonation; C, bilateral abductor paralysis; deep inspiration; D, left abductor paralysis; deep inspiration; affected cord in cadaveric postion; E, left abductor paralysis; phonation; affected cord in cadaveric position; right cord crossing median line; F, bilateral thyro-arytenoid paralysis; G, bilateral thyro-arytenoid paralysis and paralysis of arytenoideus, giving an hour-glass opening.

includes the globus hystericus, œsophagismus, pharyngismus, false and genuine hydrophobia and the pharyngeal crises of tabes. The œsophagus is rarely affected except in diseases of the centres or trunk.

Laryngeal Branches.—There are two laryngeal nerves, the superior laryngeal and the recurrent laryngeal.

The *superior laryngeal* is (*a*) the sensory nerve for all parts of the larynx above the vocal cords, (*b*) the motor nerve for but one larynx muscle, the cricothyroid, which modulates the voice by regulating tension in the cords, and (*c*) it is the motor nerve for the depressors of the epiglottis, the thyro-epiglottic and ary-epiglottic muscles, paralysis of which causes the epiglottis to stand upright, thereby risking entrance of food into the larynx and aspiration pneumonia.

The *recurrent laryngeal* curves around the aortic arch on the left and the subclavian artery on the right side; (*a*) it is the sensory nerve to the mucosa of the larynx below the cords and the trachea and (*b*) is the motor nerve to every intrinsic larynx muscle except the cricothyroid. The physiologically complicated larynx muscles have three main movements: (*a*) to render the cords tense (this is accomplished by the cricothyroids and to a less extent by the thyro-arytenoids); (*b*) to adduct the cords, which is done chiefly by the lateral crico-arytenoids (also by the thyro-arytenoids and arytenoids); (*c*) to abduct the cords, which is done by the post. crico-arytenoid; it is especially important because it is the only special abductor; it is very frequently and almost invariably first involved in incomplete recurrent paralysis.

Treatment.—Treatment depends wholly on the cause, which is usually peripheral. In nuclear types there is no therapy. Suggestion, or electrotherapy, is valuable in hysterical forms. The faradic current may be used with the positive pole to the forehead and the negative to the larynx externally or internally. Other measures include laryngeal gymnastics, in which the patient repeatedly attempts to speak, intralaryngeal insufflations of alum, the administration of strychnia hypodermatically and Ollivier's procedure, in which the thyroid cartilage is compressed between the index-finger and thumb during inspiration and expiration, while the subject attempts to phonate. Tracheotomy and feeding by the nasal catheter are sometimes indicated.

Anæsthesia and Hyperæsthesia of the Larynx.—*Anæsthesia* of the larynx is rare, but may occur uni- or bilaterally in disease of the sup. laryngeal nerve, the vagus roots, its nucleus or in the hemianæsthesia of hysteria or organic brain disease. In nuclear disease it is partial; in affections of the nucleus, root and trunk the reflexes are lost, but they are present in supranuclear and functional affections. *Hyperæsthesia* is rarer, being observed in neurotics as a tickling, tendency to cough or spasm; neuralgia and crises also occur.

Laryngeal Spasm.—The most common form of laryngeal spasm is spasm of the adductors, which are concerned in reflexes inimical to

TABLE FOR DIFFERENTIATION OF LARYNGEAL PARALYSIS (elaborated from Gowers and Archibald Church).

FORM OF PARALYSIS.	NERVE AND MUSCLES AFFECTED: FUNCTION.	CAUSES.	SYMPTOMS.	SIGNS BY LARYNGOSCOPY.
Total bilateral palsy.	Recurrent nerves; all muscles involved or all save cricothyroids.	Nuclear disease, disease of trunk or of recurrent nerve.	Voice lost; cough impossible; stridor only on deep inspiration.	Both cords moderately abducted, motionless in cadaveric position, neither receding on deep inspiration nor approximating in phonation. FIG. 3.
Total unilateral palsy.	Recurrent nerve; ditto.	Ditto. (In general, paralysis of one cord always suggests pressure on the recurrent.)	Voice low-pitched and hoarse; cough impossible; stridor absent or only on deep inspiration.	Paralyzed cord as above; sound cord moving freely, even beyond median line.
Bilateral abductor palsy.	Recurrent nerves; both posterior crico-arytenoids, abductors.	Central disease, toxæmia, pressure or disease of trunk or recurrent nerves. (In general, marked bilateral cord paralysis always suggests centric disease.)	Voice little changed; cough possible; extreme inspiratory stridor, long inspiration, marked laryngeal excursion, cyanosis, even death; expiration free whereby differentiate from bronchial stenosis even without laryngoscopy.	Glottis is chink-like, cords near together, not separated on inspiration but even drawn closer by unopposed adductors. FIG. C.
Unilateral abductor palsy.	Recurrent; one posterior crico-arytenoid.	Same; especially aneurysm, injury, etc., to one recurrent laryngeal nerve.	Symptoms inconclusive, little affection of voice or power to cough.	The same, but unilateral. FIG. D and E.
Bilateral adductor palsy.	Recurrent; both lateral crico-arytenoids and arytenoids; adductor muscles.	Hysteria (women under thirty years), overuse of voice, catarrh of larynx, anæmia.	Voice lost, but coughing, laughing, even singing are possible, hence called phonetic paralysis by Türck; no stridor or dyspnœa; often with anæsthesia of larynx and pharynx (hysteria), etc., hysterical aphonia.	Cords normal in position but not brought together on phonation; normal movement on breathing. FIG. A.
Unilateral adductor palsy.	Recurrent; one lateral crico-arytenoid and arytenoid.	Toxæmia, lead, diphtheria, cerebral disease, syphilis, tuberculosis.	Voice hoarse; coughing or laughing imperfect; sometimes dysphagia.	Ditto; but unilateral. FIG. B.
Paralysis of cords proper.	Recurrent; thyro-arytenoids; tensors and adductors of cords.	Very frequently involved from overuse of voice; hysteria; catarrh.	Loss falsetto notes; uncertainty of phonation; often with adductor or arytenoid paresis.	Cords atonic and concave. FIG. F. When thyro-arytenoids and arytenoids are involved, the space between cords is hour-glass in form.
Bilateral paralysis of tensors.	Superior laryngeal nerve; crico-thyroids, the tensors and modulators of cords.	Cold, straining of voice, diphtheria.	Hoarseness; high notes imperfectly taken; voice weak.	Cord higher on paralyzed side, but hard to see with mirror.

entrance of foreign bodies into the larynx; it may result from centric, local (laryngitis with croup-like cough at night) or reflex irritation from distant or neighboring foci, as elongated uvula, goitre or hypertrophy of the pharyngeal tonsil. It includes *laryngismus stridulus,* which occurs in children under three years of age, especially in boys, and was previously referred to the thymus gland, but is due to rickets in 66 per cent. of cases and also to tetany, and reflexly to alimentary disorders; it comes on at night or on awakening in the morning, with croup-like cough, and presents all the symptoms of acute suffocation, as inspiratory and expiratory stridor, pallor, cyanosis, wide pupils, protruding eyes, low diaphragm, involuntary evacuations, centric vasomotor and cardiac disturbances and convulsions either generalized or of the infantile carpopedal type. The attacks last from a few seconds to one-half or one minute and are sometimes attributed to loss of temper when they follow a reproof. In the adult, laryngeal spasm occurs in tabes, hysteria, tetany, hydrophobia, epilepsy (the initial cry of which arises from spasm), asthma or migraine. The attacks are recognized by the negative laryngoscopic findings, paroxysmal occurrence and the stridor being manifest in both inspiration and expiration. Partial spasm occurs in some types of stuttering, in clergymen's sore throat and in explosive, unmodulated speech.

Nervous cough is present in the explosive hysterical type, in the metallic ovarian form in young girls and in the barking variety in young masturbators.

Treatment.—Treatment of spasm includes removal of the cause, relief of local irritation, as affections of the ear (which has branches from the vagus) or pharynx, correction of hygiene, administration of tonics, as cod-liver oil in children, and such immediate measures as thrusting a finger into the throat, dashing water in the face, hydrotherapy and for the attack chloral and bromides by enema, local application of cocaine, inhalation of chloroform and in adults nitrite of amyl and morphine.

Pulmonary Branches.—The vagus and the sympathetic form the pulmonary plexus. Afferent fibers convey sensory impressions to the respiratory centre; these include accelerating fibers which predominate, and inhibitory fibers, stimulation of which (sup. laryngeal nerve) arrests breathing. Afferent impulses are also carried to the vasomotor centre. Efferent motor impulses are conveyed to the bronchi. Division of the vagus causes rapid congestion of the lungs with hemorrhage (also observed in acute lesion of the pons) and slower but deeper breathing; stimulation of the divided end of the nerve accelerates breathing even to its tetanic arrest. Bronchopneumonic foci are not trophic but aspiration pneumonia from paralysis of the palate and larynx. The pulmonary twigs are probably concerned in Cheyne-Stokes' breathing, bronchial asthma and hiccough.

Cardiac Branches.—The vagus and sympathetic nerves constitute the cardiac plexus. (*a*) Motor fibers from the spinal accessory nerve

inhibit and coördinate cardiac action, which is sometimes under the control of the will; the heart may at times be slowed by pressure on the neck; marked irritation of the vagus slows or even arrests the heart. Division may cause sudden death, though this is unusual in unilateral lesions. Paralysis of these fibers allows the sympathetic accelerator fibers to dominate, producing arrhythmia and tachycardia. Digitalis is ineffective in paralysis of the vagus. (*b*) The sensory fibers are concerned in the cardiac neuroses (*q. v.*). (*c*) These are probably trophic fibers, as Eichhorst's experiments show fatty myocardial degeneration on division of the vagi.

Gastric Branches.—(*a*) Motor branches supply part of the stomach; they cause nausea and are concerned in reflex, direct or centric vomiting, as in brain lesions, wherein the vomiting usually occurs without nausea. (*b*) Sensory branches; the vagus is the nerve of hunger and thirst, and its disease often causes anorexia and loss of thirst. Centric disease causes the gastric crises of tabes and cortical functional disease produces the epigastric aura of epilepsy and perhaps gastralgia in the neuroses. Vertigo probably causes vomiting through the vagus. Intestinal and splenic symptoms are lacking in affections of the vagus, although the nerve accelerates peristalsis.

Eleventh or Spinal Accessory Nerve (external portion).—The internal, minor part runs with the vagus, while the major, external segment is really a spinal motor nerve, arising from the lateral portion and ant. horns of the cord between the sixth cervical segment and the medulla. It courses upward through the foramen magnum into the cranium and leaves it with some of the true cranial nerves. It is the chief supply of the sternomastoid muscle and the upper part of the trapezius, the lower two parts of which are usually supplied by the cervical and upper dorsal nerves.

Paralysis.—Paralysis is rarely caused by cortical disease, because the muscles have bilateral representation. The usual causes are bulbar paralysis, lesions near the foramen magnum, meningitis (then usually with involvement of the inner accessory branches and the twelfth nerve), syphilis, trauma, tabes, syringomyelia, progressive muscular atrophy, caries or tumor of the spine, or neuritis. The symptoms are paralysis of (*a*) the sternomastoid, in which there is impaired rotation of the face to the side opposite the lesion; in old cases contracture turns the face toward the paralyzed side. (*b*) That part of the trapezius from the occiput to the acromion is paralyzed, showing a concave instead of a straight line, best seen in deep inspiration or shrugging the shoulders. (Paralysis of the second portion causes imperfect elevation of the arm, some drooping and winging out of the scapula, the lower part of which is drawn toward the spine by the rhomboids and lev. anguli scapulæ; paralysis of the third portion results in weakness in the adduction of the scapula to the spine.) If it is bilateral as in chronic meningitis of childhood or progressive muscular atrophy the head falls forward or back-

ward as the sternomastoids or trapezii, respectively, are affected. In peripheral disease there is wasting and the reaction of degeneration.

Accessory Spasm, Spasmodic Torticollis or Wryneck.—Etiology.—It occurs in women in 66 per cent. of cases (Gowers); Osler finds it largely in males between 30 and 50 years of age. A neurotic tendency, trauma, overexertion, acute infections, alcoholism, plumbism, cervical caries, brain tumor and diseases of the eye and ear (torticollis ab oculo læso, ab aure læsa) are among its uncertain causes.

Symptoms.—The symptoms vary as the affection is clonic or tonic, unilateral or bilateral, and also with the number of muscles involved. One sternomastoid is generally involved, especially in tonic spasm, in which the occiput is turned to the diseased side, the face to the opposite side and somewhat upward, and the ear brought nearer the clavicle. The upper part of the trapezius comes next in frequency of involvement and is affected equally by clonic and tonic spasm. The affected shoulder is raised and slightly rotated, the scapula drawn toward the spine and the head backward. The spine is often convex toward the diseased side. The splenius is half as frequently affected (Gowers), the head being drawn backward, the face a little to the affected side and upward. Less often the scalenus, platysma, rectus and obliquus capitis are affected. The muscles in tonic spasm are hard, prominent and sometimes hypertrophied. Clonic spasm disappears during sleep, but is increased by fatigue and emotion; it may also invade the face, neck, eyeballs, vocal cords or the arm. There may be pain, fatigue and rarely tender spots from compression of the brachial plexus by the scaleni. Bilateral clonic or tonic spasm of the trapezius draws the head backward (retrocollic spasm) and always involves the frontalis muscle.

Course and Prognosis.—The onset is gradual, the disease is usually stationary or progressive in its course, rarely recurrent; recovery is infrequent. The outlook is generally grave in well-developed types.

Diagnosis.—(*a*) In *congenital or fixed wryneck* there is no active spasm. One sternomastoid (but rarely the trapezius) shows atrophy, induration and cicatrization, which Stromeyer considers due to traction on the neck with injury during delivery. Seventy per cent. of cases occur in left occipito-anterior presentations. The disease escapes notice for years because of the shortness of the child's neck. Wilks always found facial asymmetry. Golding-Bird considers the affection a contracture from a central lesion. Tenotomy cures it. (*b*) *Hysterical torticollis* occurs in subjects under thirty years of age; other stigmata usually develop. Bompaire and Brissaud describe a *mental torticollis*, clearly a psychical phenomenon. (*c*) Torticollis may occur *in children* from dentition to puberty; clonic spasms, especially in the deep muscles, lead to nodding, salaam movements, spasmus nutans, nictitatio spastica. The eyes may show nystagmus. The affection may develop from reflex dental or digestive disorders or from organic cerebral disease. It is sometimes habit spasm, hysteria or even part of an epilepsy or idiocy.

Treatment.—Treatment is first etiological. Nervines, sedatives and electricity seldom produce permanent results. Morphine is a dangerous agent, although its local hypodermatic use has led to recovery. Massage and deep compression are occasionally helpful. In chronic cases resection of the accessory nerve is indicated. If many muscles are involved the third and fourth cervical nerves must also be resected. Successful results are reported by W. W. Keen and Noble Smith.

Twelfth or Hypoglossal Nerve.—This is purely a motor nerve. *Paralysis of the tongue is* (*a*) *rarely cortical;* supranuclear glossoplegia from lesions between its centre in the lower part of the ant. ascending and the third frontal convolutions, almost always occurs with hemiplegia and paralysis of the lower facial fibers; there is no wasting or reaction of degeneration. The root of the tongue is higher (hyoglossus paralysis) and on protrusion the tongue deviates to the paralyzed side, being pushed over by the sound genioglossus. (*b*) *Nuclear paralysis* occurs often in bulbar paralysis, sometimes in tabes or paretic dementia, and much less frequently in acute inflammation or softening in the medulla. Nuclear lesions are almost invariably bilateral since the nuclei are closely contiguous, and show the reaction of degeneration and wasting, the mucosa being wrinkled over the atrophic half. Wasting may be more marked than paralysis. Fibrillation is frequent, as in paretic dementia, from cortical and nuclear changes. In bilateral glossoplegia the tongue cannot be protruded, there is impairment of the first act of swallowing, in chewing and articulation, all of which are but slightly marked in unilateral paralysis. (*c*) *Infranuclear paralysis* results from lesions near the surface of the medulla, which may produce unilateral paralysis of the tongue, palate and vocal cord. Contralateral hemiplegia may occur. The lesion may affect one or both nerves. Syphilis, trauma or tumors are causal; there are recorded only 40 cases of peripheral, isolated hypoglossal paralysis (Panski, 1903). Wasting, the degenerative reaction and fibrillation are present. *Treatment* is that of other paralyses.

Spasm.—Spasm is rare either in the tonic or clonic form. It occurs in chorea, facial spasm, certain types of stuttering, epilepsy (in which the tongue, protruded by the genioglossus, is bitten), hysteria (in which the tongue is rarely bitten), certain psychoses, meningitis, neurotic spasm in speakers (aphthongia) or musicians. The prognosis varies with the cause and in general is favorable.

DISEASES OF THE SPINAL NERVES.

The spinal nerves differ from the cerebral in that they contain sensory, motor and vasomotor fibers. Disease or injury therefore suspends motility and sensation, causes vasomotor paralysis, trophic changes or wasting, glossy skin, the reaction of degeneration, atrophy and contracture.

Phrenic Nerve.—Paralysis.—(*a*) Disease or trauma of the vertebræ, cord, membranes or of the third, fourth and fifth cervical nerves of origin may cause it. In disease of the cord other paralysis is always present. (*b*) Its trunk is well protected but may be injured in its course or damaged by aneurysm, tumors or pleurisy. Neuritis sometimes occurs in multiple neuritis or degenerative lesions, as tabes or progressive muscular atrophy.

Symptoms.—The diaphragm is paralyzed, usually on both sides. The abdomen retracts on inspiration and bulges on expiration, the converse of its normal movements. Litten's diaphragmatic sign is absent and there is no inspiratory descent of the liver or spleen. Blowing, coughing and pressing at stool are impossible. Costal breathing is exaggerated, the accessory muscles are strongly in play, dyspnœa and cyanosis are present, especially on exertion, and coincident bronchitis may cause most alarming symptoms, allowing stagnation of mucus and basal congestion. The nerve may be tender in the neck.

Diagnosis and Treatment.—Costal hysterical breathing may at first cause confusion, as may inflammation and degeneration of the diaphragm from diaphragmatic pleurisy. The *outlook* is always grave. It is more favorable in syphilis, lead or diphtheritic neuritis than in alcoholic forms. *Treatment* is that of the cause but is usually futile. Faradic phrenic stimulation is sometimes beneficial, as in apparently asphyxiated new-born infants.

Phrenic Spasm.—1. *Clonic spasm,* singultus or hiccup is a form in which the diaphragm spasmodically contracts with a sound due to sudden closure of the glottis, sometimes 50 to 100 times a minute. It is probable that the vagus and respiratory centres are involved, for dyspnœa, dysphagia or dysarthria may occur. Clonic spasm occurs (*a*) in inflammation, either thoracic (pleurisy, pericarditis) or abdominal (peritonitis, appendicitis, obstruction); (*b*) in irritation, œsophageal, mediastinal, hepatic or gastric, directly or reflexly; (*c*) in nervous affections, as centric disease or the neuroses; and (*d*) in toxæmia or uræmia. *Treatment* is that of the cause. Gastric lavage; apomorphine; circular constriction of the lower chest with the head bent forward; attempts to sneeze, cough, breathe deeply or press down with the glottis closed; faradization, freezing, blistering the neck and epigastrium; asafœtida, chloral or bromides; and narcotics and inhalations of chloroform or amyl nitrite may be beneficial, although the affection is often intractable.

2. *Tonic phrenic spasm* is rarer. The causes are tetany, epilepsy, tetanus, rheumatism and hysteria. The diagnosis is easy. The lower chest and upper abdomen are distended and often sensitive; the upper chest moves rapidly and forcibly, with the accessory respiratory muscles in full play; there is no respiratory excursion, the lungs are acutely distended, the abdominal viscera are luxated downward, there is dyspnœa, and cyanosis and death may result. *Treatment* consists of the application of heat, blisters and the faradic current to

the lower chest and the use of chloroform inhalations and morphia internally. Analogous spasm may occur in the respiratory muscles, as yawning, sneezing, coughing, laughing and weeping spasms, possibly from vagus disease or from the neuroses, psychoses, multiple sclerosis, bulbar paralysis or disease of the ear, nose or stomach.

The Posterior Thoracic Nerve.—This nerve is often affected in its long course, from trauma, muscular strains in overhead work (the nerve originates in the scalenus medius) and from carrying loads on the shoulder—whence its occurrence in men in 90 per cent. of cases; it is also involved in muscular atrophies and in neuritis. The results are impaired fixation of the scapula, movement of the arm and inspiration. The scapula wings out when the arm is moved forward and stands less obliquely than normal, its outer part moving forward, while the lower angle is drawn in by the rhomboids and trapezius. The sole sensory change is pain in the neck and shoulder. The course is tedious and rest, especially of the scaleni, is the prime indication.

Suprascapular Nerve.—It originates in the fourth, fifth and sixth cervical nerves and is affected in disease of the plexus or alone in trauma, shoulder dislocations or falls on the hands. The supraspinatus and infraspinatus are paralyzed, giving some weakness in fixation of the humerus, as uncertainty in writing, turning of the lower scapular angle up and in, some anæsthesia over the scapula, frequently shoulder girdle pain and often involving the **Circumflex Nerve**, which arises from the fifth, sixth and seventh cervical nerves of the posterior cord of the brachial plexus. Deltoid paralysis (dislocation, crutch paralysis, plexus disease) produces inability to deviate laterally the arm, changes in the shoulder, which becomes angular, sometimes adhesions in the joint, probably trophic from its articular branch and anæsthesia over the lower part of the deltoid. It may be confused with primary joint disease, in which, however, the scapula follows the arm movements. Paralysis of the teres minor is unimportant.

Musculospiral Nerve.—Arising from all the roots in the plexus except the first dorsal and curving around the humerus, it is the most exposed and most frequently injured nerve in the arm or body; injuries result from fracture, callus, blows, muscular exertion, carrying heavy weights, tying the arms to the side or behind the back, sleeping with the arm over the back of a chair, especially in alcoholics, lifting the arms during surgical anæsthesia and Esmarch's constrictor; less often injury results from cold or lead palsy. *Musculospiral (radial) paralysis* involves (*a*) the extensors of the upper extremity, *i. e.,* the *triceps,* so that the elbow cannot be extended, and the supinators; the triceps sometimes escapes affection; the supinator longus is not involved in lead palsy or traumatism below the lower third of the humerus and in centric disease it escapes because its centre is with the flexors. The *extensors of the wrist* are involved, producing marked atrophy of the back of the forearm and the characteristic wrist drop; the *extensors of the thumb and fingers* are affected and

become semiflexed and can only be extended by the interossei (ulnar nerve) after the first phalanges are passively extended. Changes in the synovial sac or bones of the dorsal wrist produce the painless carpal tumor of Gubler, from involvement of branches to the wrist or more probably from the mere mechanical action of flexion (see LEAD INTOXICATION). (*b*) *Sensory disturbance,* which is slight compared with the palsy, may rarely cause anæsthesia over the deltoid and over the back of the forearm or more often over the radial side of the dorsum of the hand, thumb, index and half of the middle finger. Most cases recover with rest and immobilization. Flexor contracture can be avoided by careful splinting.

Median Nerve.—The median nerve originates from the outer and inner cords of the plexus and contains fibers from all its cervical roots. Isolated paralysis is not common. It results from trauma, fractures, stabs, carrying weights in the bend of the elbow and sleeping on the arm. (*a*) *Motor symptoms* are conspicuous. Both pronators are involved, besides all the flexors of the forearm, wrist and fingers except the flexor carpi ulnaris and the ulnar part of the deep flexor of the third and fourth fingers. In the hand, the palmaris longus, the abductor, opponens and short flexor of the thumb (resulting in flattening of the thenar prominence and the "ape hand" from the index finger and thumb lying parallel) and the two radial lumbricales are affected. The second phalanges cannot be flexed on the first (save in the two ulnar fingers); flexion of the first phalanges by the interossei is possible; metacarpophalangeal subluxation may result from the unopposed extensor action of the interossei on the second and third phalanges. The paralyzed muscles may waste and trophic manifestations are quite common. (*b*) *Sensory symptoms* are often slight. Anæsthesia, if present, is greatest in the palm, front of the thumb, the first two fingers and half of the third.

Ulnar Nerve.—Its origin is the last cervical and first dorsal nerves; its superficial course at the inner condyle, forearm and wrist, exposes it to injury, as by dislocation of the elbow, cuts and pressure on the elbow during sleep and work. Neuritis occasionally causes paralysis. It must be distinguished from spinal disease, which ascends from the dorsal to the cervical region; this form is usually bilateral and is preceded by ulnar anæsthesia (Biernacki) in tabes and paretic dementia. The *flexor carpi ulnaris* is paralyzed (the hand deviates to the radial side in attempts at flexion), and also the ulnar half of the *deep flexors* of the *fingers,* the muscles of the little finger, palmaris brevis, the interossei, the third and fourth lumbricals, the adductor and inner head of the short flexor of the thumb (the thumb being rotated toward the palm). The first phalanges cannot be flexed nor the others extended, although this is less marked in the first and second fingers, in which the lumbricals are supplied by the median nerve. The *"claw hand"* results, although it is less marked than in spinal muscular atrophy; the hypothenar eminence, palm and interosseous spaces become thin. *Sensory loss* is limited to the back and front of the ulnar

side of the hand, the dorsum of two and a half fingers and the front of one and a half fingers on the ulnar side.

Combined Paralysis of the Arm Nerves.—This affection, described by Erb (1884), is very common and is due to affections of the vertebræ, cord or nerve roots, to tumors, trauma or cicatrices of the neck and wrenches or forcible delivery in obstetrics. Several nerves are involved, as the musculospiral and ulnar in fracture of the humerus, or in subcoracoid luxation; in Erb's case the circumflex, suprascapular and musculocutaneous nerves, fifth and sixth cervical roots, with some branches from the fourth, were imbedded in a cicatrix and paralyses of the deltoid, teres minor, spinati, biceps, brachialis and supinators, with few sensory signs resulted. Excision of the scar, resection and nerve suture resulted in recovery. Erb found one spot between the scaleni where all these nerves could be stimulated and could be simultaneously affected. Neuritis ascending from the median nerve may cause ulnar and musculo-cutaneous or other paralysis. Obstetrical paralysis usually recovers, while adult forms depending on their etiology are often protracted or permanent. Flaubert (1827) described a combined paralysis, which usually bears Klumpke's (1885) name, in which paralysis of the first dorsal and eighth cervical roots (ulnar and median paralysis) also causes narrowing of the pupil and the interpalpebral fissure.

Brachial Neuritis.—This affection was first clearly described by Gowers as a perineuritis of the plexus or nerve roots (radicular neuritis), occurring largely in gouty or rheumatic subjects over fifty years of age, but at times due to compression or trauma. The author saw one case which occurred in the right arm of a busy surgeon. A cervical rib may cause compression symptoms; Reisman (1904) collected 46 cases of which 24 were operated on with good results. It is marked by great pain, the first, most severe and most enduring symptom. It is constant, with paroxysmal exacerbations, is increased by the least movement and is experienced over the plexus, nerves or referred to the wrist or scapula. Associated with it are tenderness over the trunks or plexus, hyperæsthesia of the skin, some muscular wasting, mostly in radicular forms and somewhat less often anæsthesia, anæsthesia dolorosa, œdema, glossy skin and trophic alterations in the joints. Neuralgia, angina pectoris and aneurysm may cause diagnostic difficulties. Remak holds that reported recoveries of acute poliomyelitis in children (Kennedy's temporary paralysis) are only plexus neuritis. The *outlook* is usually favorable, but a long course of from a month to more than a year, relapses and even some wasting, cramps, rheumatic pains and joint adhesions must be considered in framing the prognosis, despite early and careful treatment, as in reduction of shoulder dislocations. Treatment, as in neuritis, consists of rest by immobilization, injection of cocaine in severe cases and treatment of the underlying trauma, gout or rheumatism.

Nerves of the Trunk.—These are rarely involved except in lesions of the spine or cord. The *neuritic pain in Pott's disease* or cord

tumor and the *girdle pain of tabes* are of great localizing value. It has been shown that *herpes zoster* is due to inflammation in the spinal root ganglia (Campbell and Head).

Nerves of the Lower Extremities.—These nerves, except in sciatica, are less frequently affected than those of the arm.

The Lumbar Plexus.—The lumbar plexus (the first three and a half lumbar roots) may suffer in abdominal tumors, glandular enlargements, parturition, psoas abscess, pelvic inflammations, spinal meningitis, vertebral disease or in neuritis ascendens. The symptoms are *paralysis* of the cremasters, *flexors and adductors* of the *hip* and *extensors of the knee;* all the hip flexors are involved in plexus disease only; great pain in neuritic forms is noted in the ilio-inguinal, iliohypogastric and genitocrural areas. *Anæsthesia* exists over the lower abdomen, front and sides of thigh and inner side of leg and foot and adjacent sides of first and second toes.

Anterior Crural Nerve.—Lesions in the abdomen cause paralysis of the iliacus (not the psoas), the hip flexors and knee extensors, with wasting, loss of knee jerk and anæsthesia over the front and sides of the thigh and the inner side of the leg and foot. It has occurred in 1.7 per cent. of labor cases and may result from stab wounds of the thigh, in which case the iliacus escapes involvement. Neuritis of its *external cutaneous branch* (multiple neuritis, alcoholism, nicotinism or gout) may cause isolated anæsthesia over the lower outer half of the thigh, paræsthesia or marked hyperæsthesia; the last condition, known as *meralgia,* was described by Bernhardt and Roth and is usually neuritis but may be due to pressure from varicose veins. It is noted, chiefly, on standing or walking. In 1900 Musser and Sailer collected 99 cases (74 per cent. in men).

Pressure on the cord at the fourth lumbar roots produces the same result as a partial lesion of the ant. crural nerve, as paralysis of knee extensors, loss of knee-jerk and anæsthesia of anterior part of thigh, but foot clonus is present. Disease of the anterior horns on one side may exceptionally occur in adults, but its wider prevalence and sensory exemption are usually sufficiently distinctive. Caudal lesions are usually bilateral.

Obturator Nerve.—This nerve is rarely alone involved save during parturition. Its paralysis affects the thigh adductors, so that the knees cannot be crossed, and the pectineus and ext. obturator, so that impairment of outward rotation of the thigh results. Sensory symptoms concern the hip and knee joints, which sometimes show trophic disturbance. The pain of hip disease is referred to the knee by the obturator nerve.

Superior Gluteal Nerve.—This arises from the lumbosacral cord, being intermediate between the lumbar and sacral plexuses. Its rare isolated involvement results in loss of thigh abduction and circumduction (gluteus medius and minimus).

The Sacral Plexus.—The sacral plexus (half of the fourth, the fifth lumbar and the first four sacral nerves) supplies motion to the

extensors and rotators of the hip, flexors of the knee and all the foot muscles, and sensation to the gluteal region, the back of the thigh, outside and back of the leg and most of the foot by the gluteal nerves and the two sciatic nerves. The *lesser sciatic* is affected only in disease of the plexus; it is often involved in pelvic inflammation and tumors, parturition, traction in leg deliveries, plexus neuritis and diseases of the hip.

Great Sciatic Nerve.—This is the largest, the most exposed throughout its course and hence the most frequently diseased nerve of the lower extremity. Paralysis of the gluteus maximus interferes with rising more than with walking. A lesion near the sciatic notch paralyzes the extensors of the hip, the flexors of the leg and all muscles below the knee. If the lesion lies below the upper third of the thigh the flexors of the leg may escape involvement and walking is still possible, even though all muscles below the knee are paralyzed, if the ant. crural nerve for the hip flexors and knee extensors is intact. Sensation is lessened over the outer half of the leg, most of the dorsum of the foot and all of the sole. Wasting is marked over the back of the thigh and perforating ulcer of the foot may develop. It has two *branches:* (*a*) the *external popliteal* or *peroneal nerve,* involvement of which causes paralysis of the tibialis anticus, peronei, long toe extensors, extensor brev. digitorum; the ankle cannot be flexed dorsally, the first phalanges of the toes cannot be extended, adduction and lifting the inner edge of the foot is difficult and abduction and lifting its outer edge is impossible. Foot-drop results and contracture assumes the equinovarus type. This nerve is the analogue of the musculospiral in the arm. Wasting is often observed and the anæsthesia covers the anterior outer half of the leg and the dorsum of the foot. (*b*) *Internal popliteal nerve,* involvement of which affects the popliteus, tibialis posticus, long flexors of toes and all the foot muscles except the extensors; plantar flexion, abduction of the foot, inability to stand on tiptoe, talipes calcaneus and the claw foot from overextension of the proximal and overflexion of the second and third phalanges result. Sensory alterations are variable; anæsthesia may be observed on the outer lower posterior part of the leg and on the sole of the foot. Its plantar branches are rarely affected alone; (i) paralysis of the internal plantar nerve, which is analogous to the median; anæsthesia on the inner part of the sole and the plantar surface of the three inner toes and one-half of the fourth; paralysis of the short toe flexors, plantar muscles of great toe (except the abductor) and the two inner lumbricals; the big toe is overextended, "the hammer toe"; (ii) paralysis of the external plantar nerve, the analogue of the ulnar, produces anæsthesia of the outer half of the sole, little toe and one-half of the fourth, paralysis of the accessory flexor, small toe muscles, all the interossei, two outer lumbricals and great toe adductor; the results are serious, walking being impaired by flexion of the second and third and extension of the first phalanges.

Caudal Lesions.—(See SPINAL LOCALIZATION.)

Sciatic Neuritis.—Sciatica, Ischialgia, Malum Cotunnii (Cotugno, 1764).—Sciatica is, after facial neuritis, the most frequent isolated form and is rarely simply neuralgic.

Etiology.—1. General Causes.—(*a*) Sex and age; 88 per cent. of cases occur in men; 16 per cent. in persons between twenty and thirty, 31 per cent. between thirty and forty, 25 per cent. between forty and fifty and 18 per cent. between fifty and sixty years of age. It is very rare in children. (*b*) Acute infections, rheumatism and grippe are causative. (*c*) Intoxications; gout, fascial rheumatism (sometimes by extension from lumbago) and diabetes are factors. (*d*) Anæmias and neuroses are less significant than in neuralgia.

2. Local Causes.—These include (*a*) spinal affections, tumor and caudal neuritis; (*b*) abdominal and pelvic diseases, as aneurysm, tumors or inflammation; (*c*) cold; exposure and dampness, favored by certain occupations; (*d*) trauma to the spine, pelvis, hip or peripheral course of the sciatic trunk or branches, as from the use of forceps or pressure of the head in labor, fractures, dislocations, falls, protracted sitting or riding, use of the sewing machine, marching, vigorous contractions of the leg flexors, peripheral pressure by popliteal aneurysm, tumors, callus or varicosities.

Pathology.—In the infrequent autopsies or in surgical stretching of the nerve its sheath shows perineuritis with œdema and in some cases increase of connective tissue or varicosities of the sheath vessels, especially in the middle thigh. Sometimes interstitial neuritis is found.

Symptoms.—The symptoms are unilateral and chiefly sensory, as *pain and tenderness* over the sciatic roots, trunk and branches. (*a*) The onset of *pain* is usually gradual but is sudden and febrile in some rheumatic cases. It is usually constant, with exacerbations which in some cases are agonizing. The pain descends more often than it ascends; it sometimes radiates to the buttocks and crural region, even into the other leg. Gowers attributes the local pain to perineuritis and the distant pain to interstitial neuritis. Any movement which increases sciatic tension increases or renews the pain (Lasègue's phenomenon); pain is increased or initiated by flexing the hip and extending the knee, walking, stooping or defecation. The patient seeks positions which spare stretching of the nerve and its compression by the muscles; he may lie on the opposite side with the thigh extended, knee flexed and foot extended, or on his back with hip and knee flexed and foot extended; in standing, he rests on the sound leg and in protracted cases scoliosis develops (ischias scoliotica, Albert, 1886), in which the lumbar convexity is toward the diseased side, heterologous scoliosis, which usually disappears with the disease; a homologous scoliosis (with the lumbar concavity toward the affected side) may result in case the muscles of that side become spasmodic. (*b*) *Tenderness* may be detected over the entire course of the nerve or in certain locations only; (i) over the lumbar region; (ii) near the sacrum and post. sup. iliac spine; (iii) at the lower gluteal fold

where it leaves the sciatic notch; (iv) between the tuber ischii and trochanter; (v) especially over the middle of the *posterior surface of the thigh;* and (vi) in other lower areas of distribution over the popliteal space, head of fibula, calf, internal and external malleoli and sole. The enlarged nerve may occasionally be felt. Pain is often experienced in the hip and sciatic notch when pressure is made in the popliteal space. (*c*) Other symptoms are less frequent, as vasomotor changes, herpes, muscular cramps, twitchings, fibrillation or wavy movements (myokymia); there is usually some diminution in the electrical excitability, but there is rarely a marked degenerative reaction; muscular atrophy may occur from disuse or neuritic atrophy; sciatic paralysis may occur; hypæsthesia, paræsthesia, or in severe cases anæsthesia, may develop in the posterior part of the thigh, leg or foot; very rarely there is polyuria or glycosuria. The patellar reflex is usually normal, the Achilles reflex is often decreased or absent and Gibson found the cremasteric usually increased.

Diagnosis.—(*a*) The etiological factor should be sought. (*b*) The pelvis should always be examined in women and the rectum in both sexes, because compression produces more distant than local pain; the urine should always be examined for sugar and albumin; and free catharsis and bowel lavage should always be given. (*c*) Bilateral sciatic pain suggests disease of the cord, especially tabes, a lesion of the cauda, in both of which there is no sciatic tenderness, or it prompts examination for diabetes. (*d*) Coxitis, sacro-iliac or psoas disease present appropriate local signs, without however Lasègue's sign or sciatic tenderness, both of which are also absent in (*e*) hysteria. (*f*) Muscular rheumatism is irregular and is chiefly confused with pain over the sciatic branches.

Prognosis.—The prognosis is generally good, in regard to the ultimate outcome. It depends on (*a*) whether the affection is primary, as from cold, or secondary, as from disease of the cord or pelvic neoplasm; (*b*) the amount of rest which the patient accepts; (*c*) the intensity of the neuritis, which may extend to the lumbosacral plexus; and (*d*) the chance (in 33 per cent.) of relapse. The average course is from two to eight weeks, but cases may last over a year, indeed one to three or exceptionally six to thirty years.

Treatment.—(*a*) Treatment is that of the *cause;* the salicylates, in some, but unfortunately few, cases afford relief; gouty or syphilitic taints may be benefited by colchicum or antisyphilitic remedies, which are often given as a last resort in other cases. (*b*) *Rest in bed* is the most important indication. In this, extension or Weir Mitchell's long splint is often beneficial. (*c*) *Counterirritation* by blisters over the nerve's course or by touching the tender or painful points with the hot iron, may give temporary though rarely lasting relief. (*d*) *Analgesics and narcotics.* Acetanilide has little effect. Gelsemium (flextr. ℥ij) at intervals of two to four hours, not infrequently gives relief, though it is necessary to watch for pulse weakness and cutaneous tingling. Codeine and atropine should be given

before resorting to morphine; morphine is often inevitable, but always dangerous. Chronic morphinism may be promoted by severe pain, the long course and the tendency to relapse. Hypodermics of cocaine are very beneficial, though this method is attended by the same danger. Simple hypodermics of water are sometimes valuable and the author has seen good results from deep hypodermatic use of Schleich's solution given in quite large amounts over sensitive points. Lange advises the use of 160 c.c. of 0.1 per cent. eucain solution into the muscle and nerve at the sacrosciatic foramen. Injections of chloroform and ether should be avoided because of the possibility of sloughing and also of accentuating the neuritis. (*c*) *Acupuncture,* the old Chinese treatment, is recommended by Gibson; it resulted in complete relief in 56 per cent. of his cases, much improvement in 32 per cent., slight benefit in 10 per cent. and failure in but 2 per cent. (*f*) *Massage* may be given. Negro has obtained results from frequent and severe local rubbing and rolling, over the nerve trunk. (*g*) *Electrotherapy* may aggravate the pain and is at least of uncertain value. Galvanism is reported to have helped some cases. (*h*) *Nerve stretching* may be effected by extension by weights, although it is rarely tolerated by the patient in personal experience. The direct exposure of the nerve has justly fallen into disuse, for the same results attend simple stretching, which should be performed under anæsthesia. The hip should be strongly flexed, the knee extended, the foot dorsally flexed and the pelvis firmly immobilized. Ordinary care prevents luxation of the hip. The results were excellent in half of twenty personal cases and were seemingly curative in seven.

THE NEUROSES.

HYSTERIA.

Definition.—The neuroses are nervous affections, provisionally isolated and often designated "functional," since no anatomical basis has yet been discovered. Hysteria is difficult to define. Möbius considers it "a state in which ideas control the body and produce morbid changes in its functions." The school of Charcot has established the psychogenic nature of hysteria. It is a cerebral, cortical condition, in which the sensory and motor centres are affected, the perception, association and recollection of ideas are disturbed and trophic and vasomotor disorders occur. As its name implies, hysteria was long thought to be due to the uterus; Sydenham first called it a nervous affection.

Etiology.—(*a*) *Age:* It is most frequent at puberty and adolescence (fifteen to twenty-five years). It is occasionally seen in children (six

to ten years or even earlier) and rarely in middle or advanced life. (*b*) *Sex:* Women are ten times as frequently affected as men, although in the lower French classes it is more frequent in males. (*c*) *Race:* The Jewish, Latin and Slavic races are most frequently affected. (*d*) *Heredity:* Charcot and most of the modern French school hold that this is the sole cause, other factors being only *agents provocateurs.* Parental intoxications, toxæmias, constitutional diseases, as gout, diabetes, arthritis deformans and a neurotic taint are important in the transmission of a congenitally weak nervous system. (*e*) *Acquired hysteria* may be due (i) to various intoxications, notably lead, alcohol and drugs; (ii) to infections, as typhoid, malaria, diphtheria and syphilis; (iii) to constitutional maladies, as gout, diabetes, chlorosis, secondary anæmias or cancer; (iv) to exhaustion from mental or physical overwork or excesses; (v) to emotional disturbance of any kind; (vi) to trauma, especially factors involving psychical shock. (vii) It may occur as an associate with chronic nervous disease, epilepsy, syringomyelia, tabes, brain tumor and multiple sclerosis. (viii) Imitation is an important factor in schools, hospitals and at religious revivals; endemics occasionally occur now as they did in the middle ages. (Refer to Zola's *Lourdes.*) Reference to the sexual organs as causes and to reflex hysteria is purposely omitted, because they have no etiological bearing. The factors enumerated under acquired hysteria are, according to the French school, merely incidental causes, making manifest the latent hereditary disposition.

Symptoms.—There is no typical clinical picture and the disease is much more commonly polysymptomatic than monosymptomatic. French writers distinguish two main groups of symptoms, the *stigmata* and *accidents.*

Stigmata.—The stigmata are usually but not necessarily persistent symptoms.

1. *Sensory Stigmata.*—Sensory symptoms are present in 85 per cent., are more frequent than the motor and are often combined with them. (*a*) Hysterical *anæsthesia* in some form is very seldom absent. It must often be looked for, but the fact should not be too obviously suggested to the patient. It varies in *degree and form.* It may be absolute, affecting appreciation of touch, pain, heat and cold. *Analgesia* is its most common form and, in decreasing order of frequency, there may be complete anæsthesia, hypæsthesia, loss of tactile sense, loss of all sensation save to the faradic current or loss of faradic response alone. In the mucosæ the same deficit frequently occurs and sometimes the bones, ligaments, joints and muscles are likewise anæsthetic or analgesic. The *distribution* varies. In rare cases there is universal cutaneous and muscular anæsthesia; the following are more frequent: Hysterical *hemianæsthesia,* which involves precisely one-half of the skin, often the accessible mucosæ and the special senses; *segmentary* or geometrical anæsthesia, which affects one limb or part of it, is variously described as "mitten or stocking" anæsthesia and is frequently associated with motor or special sense dis-

turbance in the part affected, as paralysis, aphonia, amblyopia and deafness; and *insular* anæsthesia, which affects small areas, and is therefore frequently overlooked.

Characteristics.—The anæsthesia is psychical, is associated with the functions of the part affected and does not follow the area of distribution of the peripheral nerves or the spinal segments; it is not noted by the patient, at least until suggested; fine movements may be conducted with the aid of the eyes, as the using of a pen; it may be localized because of injury or disease in the part. Most of the reflexes usually altered by organic disease remain normal, as the pupillary, lachrymal, epigastric, cremasteric, patellar and organic reflexes, while the pharyngeal in 90 per cent. of cases is lost, as well as the plantar (though the knee reflex remains); the anæsthetic areas may last during life, but they vary, move even under examination (Patrick) or disappear from emotion, strongly aroused attention, drugs or on application of various indifferent substances, as metal or wood (*transferring* it to the opposite side).

Hysterical hyperæsthesia and *hyperalgesia* are very common in localized areas, but never in universal or hemiplegic distribution. They occur spontaneously or from attention directed to a part or organ, as by traumatism. A joint may be affected with or without contracture (Brodie's joints) and this often leads to great and unfortunate errors. There is sometimes hypæsthesia in the sphere of any of the special senses. The feeling of a nail in the top of the head (clavus hystericus), sensitive spots in the mammæ, epigastrium, groins or spine are the most common forms and may occur in the midst of anæsthetic areas. The so-called ovaralgia is found more often on the left side and is cutaneous; it has no relation to the location or to disease of the ovary; it occurs also in men and in women normal as to the pelvis. Because hysterical attacks have been initiated and sometimes stopped by pressure over this and other sensitive areas they have been called *hysterogenic* points or *zones,* but attacks are only so produced in patients already hysterical and the hysterical seizure on pressure results only from some associated memory on the part of the patient; they are often the result of suggestion. The *special senses. Taste* and *smell* may be dulled or hypersensitive, and *hearing* may be blunted, or more rarely complete central deafness results, which is usually associated with anæsthesia in and about the auditory meatus. Complete *loss of vision* is both rare and transitory. Hemiopia and scotomata are almost always organic and not hysterical. Partial stigmata are more common. In most cases there is *concentric contraction of the visual field,* which is usually bilateral, but unequal on the two sides and generally greater on the side of the cutaneous defects. Concentric *contraction of the field for colors* (dyschromatopsia) is more characteristic, even pathognomonic. In the normal eye, blue has the largest field, then yellow, orange, red, green and violet; in hysteria the red field is often wider than the blue and is the last color lost. Achromatopsia designates the com-

plete loss of color perception. Errors in accommodation (always excluding abnormal corneal curvatures) may cause double images in one eye—the other being closed—(monocular diplopia), triple images (polyopia) or large or small images (macropsia, micropsia). In hysterical *amblyopia* impairment of vision in the affected eye is decreased when the other eye is opened.

2. *Motor Stigmata.*—These are much less conspicuous but foreshadow the motor accidents described below. Voluntary movements are weaker, more clumsy, slower, less coördinate than normal and there is a slight tendency toward contractures.

3. *Mental Stigmata.*—These are in certain cases so pronounced as to justify the term psychosis. Hysteria is always a psychical affection. The most conspicuous alienation is impairment of memory (*amnesia*), which concerns memory in the ordinarily accepted sense, as memory for persons, things, words, hysterical paroxysms experienced, etc., or concerns memory and associations for speech, writing, walking or standing. The amnesia, like paralysis and anæsthesia, is functional and like them returns, save in rare cases. *Mental power* is distinctly decreased, *will power is lost* (aboulia) or lessened and the patient becomes the ready victim of impulses, impressions, suggestions, fixed ideas and misconceptions both on his own part and on that of others who incorrectly consider him deceitful, vain or erotic.

Accidents.—The accidents of hysteria are the incidental attacks, also called *seizures or paroxysms.* They are usually transient or intermittent, but may become fixed and are then ranked as stigmata.

1. *Motor Accidents.*—(*a*) *Convulsions* occur in 54 per cent. of all and 84 per cent. of female hysteria. They are much less frequently the hysteria major (*grande attaque* of Charcot) than the partial, modified type (hysteria minor). The *"grand attack"* (Charcot, Richer) comprises (i) a prodromal stage, with mental depression or exaltation, palpitation, vasomotor symptoms, nausea, polyuria, an aura beginning in the epigastrium and culminating in feelings of choking (globus hystericus), vertigo and unconsciousness; (ii) a period of epileptiform convulsions (so-called hystero-epilepsy) in which, in exceptional cases, there may be biting of the tongue or even the cry of epilepsy; the face is at first pale, then red; the teeth are ground; the convulsion is most pronounced on the anæsthetic side, toward which the face turns; the tonic spasm lasts less than two minutes and is often characterized by stupor, stertor and slow wide movements of supination, circumduction and overextension, for the trunk and body are tetanically rigid; then the clonic spasm develops, with sobbing respiration, audible swallowing, abdominal rumbling and small clonic movements. Resolution follows slowly. (iii) There is a period of "clownism," characterized by contortions and grand movements, as opisthotonos (*arc de cercle*), bowing movements, violent struggling, screams, strikes and bites. (iv) There is next a period of "passional attitudes," in which the subject mimics terror, rage, joy and love; this is followed by (v) a stage of delirium with

hallucinations, perhaps of animals (zoöpsia). The entire attack covers fifteen to thirty minutes and frequent repetitions constitute the "status hystericus."

The *partial attack* contains some of the elements of the major type, and infinite variations occur; in the vertiginous type there is vertigo and sometimes other features of Ménière's disease; the globus type is extremely common; the epileptiform variety closely resembles epilepsy; there are also tetanic, emotional and syncopal types; trance-like sleep, with catalepsy in which the limbs retain any attitude given them, with sleep, which is apparent only, for the subject later recalls everything; there may be attacks of somnambulism, in which patients run miles or even undertake distant travel without recollection of the transpired interval. (*b*) *Paralysis* is an accentuation of the stigmatic muscular weakness. In over 50 per cent. it develops after convulsive paroxysms; again it follows emotional causes, traumatism or organic brain disease. It develops suddenly or gradually; it assumes the paraplegic five times as often as the hemiplegic form; the monoplegic variety is less frequent; the triplegic, diplegic (quadriplegic) or crossed forms are most common. They are rarely complete; often automatic (gesticulatory) movements may be seen; the antagonistic muscles are also involved, thus, in attempted flexion of the knee the knee extensors contract; remissions and relapses may occur; the paralyzed part may be limp or contractured; the paralyses may be ephemeral or life-long in duration; paralysis may be "ideal," *i. e.*, only for standing or walking. Vasomotor changes and œdema are rare; Weir Mitchell described a hemiœdema; the tendon, skin and electrical reactions are usually normal, although the tendon reflexes may be increased. *Hysterical hemiplegia* occurs three times as frequently on the left as on the right side. Its differentiation from organic hemiplegia is based on Babinski's table:

Organic Hemiplegia	*vs.*	Hysterical Hemiplegia.
Coma: often complete.		Rarely complete (automatic movements often persist).
Reflexes: tendon and bone reflexes abolished at first; later increased. Ankle clonus.		Rarely abolished; no clonus (or only a spurious ankle clonus due to contraction of the calf muscles, Gowers).
Cutaneous reflexes (abdominal and cremasteric) early lessened or abolished; Babinski toe sign; lachrymal reflex abolished.		Skin reflexes normal; no toe sign. Never abolished (Spiller).
Paralysis: (1) Unilateral; (2) not systematized; face, arm and leg are weak during bilateral synergetic movements. (3) Involves subconscious as well as conscious voluntary movements, producing the platysma sign (lessening of its contraction on the hemiplegic side, *e. g.* on opening the mouth forcibly), and the com-		(1) Not always unilateral; if face involved, usually on both sides. (2) Paralysis sometimes systematized; and hemiplegic side functionates perfectly in bilateral synergetic movements. (3) Subconscious voluntary movements are not involved; absence of the platysma sign and that of combined flexion of the thigh

bined flexion of the thigh and trunk (when the patient attempts to sit up). (4) Paralysis slowly improves some, but is not variable.	and trunk. (4) Variable paralysis, improvement alternating with relapses. Absolute aphasia with ability to write (Charcot).
The *tongue* deviates slightly toward the paralyzed side.	Very slightly, very greatly or even toward the sound side.
Lower muscular tone, as lowering of the eyebrow or exaggerated flexion of the forearm, especially in early cases.	None. Asymmetry of the face is due to spasm, and exaggerated flexion of the forearm is absent.
Contracture cannot be voluntarily reproduced; at first flaccid paralysis, then contracture.	May be reproduced; variable paralysis, now persistently flaccid, now spastic from onset; flaccidity with increased reflexes highly characteristic.
Hemianæsthesia: rarely total; mostly of stereognostic and muscle sense; in general rare; coarser; more lasting when present; often with bilateral hemianopsia.	Often total, involving special senses, mostly involving pain and pressure sense; frequent; variable in duration and location; usually with concentric contraction of visual field, and sometimes with crossed amblyopia.

In hysterical *paraplegia* there are usually no trophic changes, no bed-sores, no electrical alteration, no incontinence of urine or fæces and rarely retention of urine; concomitant sensory changes are significant, but many errors in diagnosis are made. Hysterical *monoplegia* usually occurs with anæsthesia of even greater extent and segmental distribution; it is lawless and illogical, viewed from the standpoint of organic disease. In *astasia-abasia* (Charcot and Blocq) the patient is unable to stand or walk, although the muscles concerned may be efficient in other movements. The condition is essentially hysteria, although it was thought to result from neurasthenia and hypochondriasis. Lesser degrees are called *dystasia* or *dysbasia.* The term stasobasophobia is self-descriptive. Möbius' akinesia algera is similar. (*c*) *Hysterical contractures* precede, succeed or alternate with paralysis, or occur alone, but, as Church well says, they are "rigid palsies"; they usually persist during sleep and yield to ether anæsthesia, save in severe protracted cases with great fibrous transformation and irremediable deformity; they develop and terminate suddenly or gradually; their causes of onset and distribution are as in paralysis. In the arms, contracture of the flexors predominates; in the legs, that of the extensors (club foot). Various spinal deformities may develop, most frequently from pseudosciatica. Weir Mitchell has seen the muscles so contracted as to resemble actual muscle tumors. It is still stated that the *face* always escapes paralysis, but its lower portion may be weak; more often, however, it is contracted early, even when paralyzed; hemispasmus glossolabialis and trismus have been observed. Spastic or pseudo-ptosis may resemble the paralytic form, but spasticity, the lowered or more level eyebrow and the concentric or parallel folds in the lid are distinctive of contracture. The ocular muscles are seldom involved; voluntary

movement may be impossible, but associated and reflex movements are normal; convergent squint may occur, but never the divergent type nor contracture of a single rectus or oblique muscle; pain suggests hysteria. The pupils are usually said to be normal, but Karplus maintains that the light reflex is lost during convulsive seizures. Organic disease of the *hip* may be simulated; local hysterical œdema may occur. Brodie (1827) first drew attention to this group of cases, and later Duchenne. Many avoidable diagnostic errors are constantly made. (*d*) There may be *rhythmical spasms,* tremor or tic. Rhythmical spasms, chorea major, the saltatoric chorea (*v. i.*) and local manifestations, as barking cough or the various tics, may occur. Hysterical tremor is wholly a "rest tremor"; that is, it occurs without voluntary movement. It may resemble the senile, Basedow or lead tremors or that of paralysis agitans or may be combined with them. It rarely simulates the intention tremor of multiple sclerosis.

2. *Sensory Accidents.*—These are frequent and are often severe and painful. Headache may occur; it is often dull or may be severe; it may be superficial or deep; it may resemble trigeminal neuralgia, migraine or the headache of organic disease, as tumor. Meningitis (hysterical pseudomeningitis) may be closely counterfeited. Spinal or vertebral pain is frequent and sometimes suggests tumor or caries. One and three-tenths per cent. of cases of sciatica are said to be hysterical. Joint disease and various visceral lesions (*v. i.*) may be closely mimicked. Dieulafoy (1905) collated 59 cases of hysterical blindness, in 5 of which this accident was the only symptom.

3. *Visceral Accidents.*—(*a*) *Cardiac* accidents include tachycardia, palpitation and pseudo-angina pectoris. (*b*) *Respiratory* accidents: *Aphonia,* usually attended by normal capacity to cough and often also to sing, has been described under paralysis of the vocal cords. It is usually due to an "idea," as in a motor aphasia which allows the patient to write. Stammering, cough and cries may be provoked by local nasopharyngeal disease. Dyspnœa, resulting from paralysis of the diaphragm, and laryngeal spasm have already been considered. Rapid breathing (tachypnœa) also occurs. The combination of dyspnœa, aphonia and paralysis of the diaphragm constitutes Briquet's syndrome. Hæmoptysis may occur, usually in the course of phthisis, but sometimes without it, possibly as a vasomotor derangement, since the blood is thin; one fatal case is on record. (*c*) *Digestive* accidents: Hysterical anorexia results from cortical conception, stomach pain, dysphagia from spasm or simple sensory perversion; it may endure for weeks and cause extreme inanition or even death. Hysterical (pseudo-) hydrophobia is an analogous aversion to water. Vomiting is sometimes simulated by protracted globus or œsophageal spasm, in which the food is simply regurgitated. Vomiting itself may be occasional or attacks may last weeks or months, often with remarkably little malnutrition; it is rarely fæcal but substances injected by rectum may be vomited in a few hours; or again vomitus may be blood-tinged, possibly from vasomotor disturbance or more often from

actual disease, as round ulcer. Ulcer may also be simulated by pain and tenderness, which are surprisingly well confined not to one spot, but to the exact outlines of the stomach. The vomitus contains vicariously large amounts of urea when the urine is suppressed. Eructations are frequent and so much gas escapes that one must conclude that air has been swallowed; this also probably causes tympanites, in which swallowed air reaches the gut through the incompetent pylorus (Ebstein). Phantom tumors or spurious pregnancy (pseudocyesis) are explained by Gowers as protrusion of the distended bowel by tonic contracture of the diaphragm. Peristaltic unrest, diarrhœa on eating, enteritis membranacea, spastic constipation, simulation of peritonitis, rectal stricture and intestinal obstruction also occur. (*d*) *Genito-urinary* accidents: Anuria in some cases has existed for ten days without uræmia. Renal pain is not frequent, but hyperæsthesia of the bladder is common. The urine is often greatly increased, with low specific gravity and solids. During attacks of the major type the solids are reduced, the urea one-third and phosphates one-half; the phosphates, which normally have a proportion of 3 of the alkaline to 1 of the earthy phosphates, show nearly an equal proportion of each. Depression of the genital functions is more common than exaltation; anæsthesia is frequent; vaginismus may be observed. Lower has found that the hysterical ovarian and other genital pains have no relation to local pelvic disease, a rather gratifying gynæcological observation.

Hysterical fever is a disputed topic. It is an established fact that severe forms may suffer a rise to 105 or 110° or higher. With visceral manifestations this may lead to a diagnosis of meningitis or peritonitis; but on the other hand the possibility of simulation by the patient rubbing the thermometer, or more important still, the possibility of confusion with tuberculosis or typhoid, must always be kept in mind.

4. *Vasomotor and Trophic Accidents.*—These are rare. Erythema is the most common. Gangrene, falling out of the hair or nails, Raynaud's local asphyxia, cutaneous hemorrhages (crucifixion stigmata, stigmata diaboli), bloody sweats, lachrymation, hæmoptysis, hæmatemesis, sudden tender swellings in the mammæ and angioneurotic œdema, which is sometimes blue in color, have been recorded in exceptional cases.

Course and Prognosis.—The course is always chronic. If we look on the disease as congenital the prognosis is poor as to total recovery, especially with fixed psychical alteration and major manifestations. Jolly remarks that complete recovery is as rare as death, which rather overstates the facts. In mild forms practical recovery may follow, but relapses may occur on disproportionately slight provocation. The outlook is better in infantile and adolescent forms than in those of middle or advanced life. The stigmata may be ephemeral or lifelong. Marriage may prove beneficial in light cases, but, with maternity, it is injurious in the severe types; marriage must never be regarded as a therapeutic agent.

Diagnosis.—The stigmata, mental, motor and sensory, are highly characteristic. The accidents are either unequivocally hysterical or prove hysterical after consideration of their associations or history. The real danger lies in overlooking concomitant disease (*a*) nervous, as multiple sclerosis, brain tumor, paretic dementia; (*b*) postinfectious hysteria, as hysteria plus neuritis; (*c*) lung, heart and other visceral diseases, all of which, as Sydenham remarked, may resemble hysteria. Simulation does not include the common borrowing or mimicry of symptoms by hysterics; simulation of hysterical stigmata and accidents is practically impossible.

Treatment.—1. Prophylaxis.—This is possible when the earliest manifestations occur in childhood. Home treatment is frequently impracticable because of the family tendency. The etiological factors must be studied, especially the hygiene and the hours of sleep. Useful occupations, as out-of-door gardening, are preferable to less wholesome and more artificial plans of distraction. Cultivation of the cardinal virtues, courage and self-control, is sought. Servants who teach fear of the dark and tell fairy stories should be watched. Self-control should be insisted upon in adult cases.

2. General Treatment of the Cause.—As the disease depends on disturbed cortical relations between the psychical and material processes, the principal treatment must be psychical. The "fixed idea" must be grasped and eradicated, which is best accomplished in ordinary circumstances by isolation of the patient from home or from other psychical or emotional circumstances surrounding the inception of the disease. Then the undue susceptibility incident to the disease may be turned to therapeutic advantage by the self-confidence of the physician and by his covert suggestions. The Weir Mitchell rest-cure may result in great benefit, and later "vague hints regarding the curative power of nature," and fresh air and sunlight may captivate the patient. Symptoms must not be suggested to the subject, as anæsthesia or pelvic disease, and examinations, especially of the pelvis, should be omitted. Hysterics should not be allowed to associate with each other for obvious reasons. This general plan is psychotherapy, which of course might include hypnotherapy, as advocated by the Nancy school, Bernheim and others. Hypnotism is generally dangerous in its ultimate and often in its immediate effects; though the definition of hypnotism may be a matter of difference or of dispute, its danger is shown by the fact that Strümpell considers it an "artificial hysteria," and Féré defines it as a "transformation of hysteria." "It is easier to make a sound person hysterical by hypnosis than to cure an hysterical one by it" (Liebermeister). Treatment is sometimes frustrated by the patient's vanity. Successful treatment requires unusual tact, profound knowledge of human nature, strong sympathies (but dominance of the patient) and enormous optimism. Of course certain single qualities, in an ignorant quack, may effect cures.

3. Special or Symptomatic Treatment.—(*a*) In relieving *pain* the physician should first acknowledge its existence and intensity in

order to soothe the patient and should then apply blisters, liniments or the galvanic current with the anode on the painful area. Ovaralgia in most cases should be ignored; in exceptional cases pelvic surgery has given relief; Lower's position has already been mentioned (*v. s.*). (*b*) *Convulsions* are treated by dashing cold water into the face, by suggestive measures or, if severe, by etherization. Bromides are of little value, valerian is sometimes beneficial and turpentine is recommended by Gowers as most efficient, 10 drops of the oil being given until slight vesical symptoms appear. Pouring water into the mouth or nose, which causes coughing, or Hare's method of holding a towel over the nose and mouth for 15 to 20 seconds, may also serve. (*c*) *Paralysis* and *contracture* should receive early treatment, for late neglected cases may necessitate surgical intervention, as tenotomy. Massage, faradism, blisters and fixation in new postures are indicated and especially should their significance be minimized and the patient be assured that they are not organic. In two cases the author has seen the paralysis disappear at once on administration of asafœtida in an effervescing mixture. (*d*) *Anæsthesia* should be ignored. (*e*) *Aphonia* is treated by intralaryngeal faradization. Laryngeal and other respiratory spasms usually answer to energetic traction on the tongue and anæsthesia or tracheotomy is rarely necessary. (*f*) For *insomnia,* the patient should retire at an absolutely regular hour; nervines should be given rather than bromides, and hot milk should be very slowly sipped. (*g*) *Dysphagia,* vomiting and anorexia may be treated by rectal feeding or by forced feeding through the stomach tube, which is the better method. Constipation is often obstinate and its mixed spastic and paretic character is shown by the fæces which are like those of sheep. Free administration of olive oil by mouth and rectum, cold abdominal compresses and endorectal galvanization are usually effective.

NEURASTHENIA.

Definition.—Neurasthenia is a diffuse neurosis, which affects the entire nervous system, but chiefly the brain; it is characterized (*a*) by morbid irritability of the nervous system with a tendency to its rapid exhaustion ("irritable weakness"), and (*b*) particularly by psychical, motor, sensory, vasomotor, secretory and vegetative disturbances in function. The nervous tissue responds unduly to stimuli, its reaction time is short and ready exhaustion results. It seems probable that the slight cellular and protoplasmic changes in nerve cells, noted by Hodge to follow fatigue, may have some bearing on its pathology. The affection as described and named by Beard in 1869, whose description was first received with some skepticism and ridicule abroad, later gained universal recognition. It is possible that the disease is more frequent than formerly because of the more strenuous life and methods of education. The difference between "nervousness" and neurasthenia is only quantitative and while nervousness is no disease in itself it is also characterized by a short reaction time (irritability) and precipitate fatigue (weakness).

Etiology.—Neurasthenia is the most common neurosis, constituting 35 per cent. of all nervous diseases and fully 50 per cent. of all neuroses. (*a*) A *neuropathic tendency* is observed in 50 per cent. of cases, especially among Hebrews whose proneness to nervous diseases is referred by Erb to inbreeding and desire of gain. Other neuroses, as hysteria, epilepsy or migraine, the psychoses and parental infirmities or intoxications may be obtained in the family history. (*b*) *Age and sex:* It is a disease of adults (75 per cent. of cases occur in persons between the twentieth and fiftieth year). It is a question whether men are more often neurasthenic than women; we may say women are more disposed, men more exposed, to the disease by their excesses in work, sexual activity and alcoholism. (*c*) *Psychical* causes include sorrow and care; hard mental or physical work rarely produces neurasthenia. Worry is a prolific factor; it is the irritable weakness of work. Hard work is injurious when combined with excesses in tobacco, alcohol, coffee, drugs or venery. Certain occupations entailing responsibility or emotional factors predispose to nervous weakness, as in railway or telegraph employes, board-of-trade men, teachers, actors, artists and musicians, but even then it is questionable whether the work itself is most injurious. Trauma operates chiefly through psychical causes, as in the traumatic neuroses (*q. v.*). (*d*) *Intoxications,* as from the stimulants above mentioned, from drug habits, lead or arsenic poisoning, acute infections, especially typhoid or grippe, from chronic affections, notably malaria and syphilis, and gout, anæmia or diabetes may be indirect factors. (*e*) *Sexual life:* Masturbation, excessive coitus and coitus interruptus, sometimes promote neurasthenia, but their importance is enormously overestimated. Masturbation probably produces less direct physical than psychical injury, for it leads to brooding over its possible results. Continence is never injurious, as sexual indulgence is rather a habit than a necessity. In neurasthenic women gynecological complaints are more often subjective, secondary and neurasthenic than objective, primary and organic. The same statement usually holds true in male neurasthenics with chronic urethritis or prostatitis. Pregnancy and lactation are sometimes apparent causes. (*f*) *Organic diseases* may possibly be factors, but it is questionable to what extent enteroptosis, chronic gastric or other somatic lesions promote neurasthenic symptoms.

Symptoms.—A clinical description holding for all cases is impossible. A patient presents himself for the treatment of some single visceral complaint or describes symptoms in his head, heart, lung, stomach, sexual or other organs. The various types, which are often separately described, will be considered in the general picture, for pure forms are rare; they are the cerebral (*cerebrasthenia*), spinal (*myelasthenia*), cerebrospinal (*the general type*), the *sympathetic* or vasomotor and the *visceral* (cardiac, sexual, gastric) types. Some varieties concern the course, as (*a*) the *acute form* from simple nervous exhaustion in normal individuals, or in those with neurotic stig-

mata; (*b*) the *subacute* or chronic acquired form, and (*c*) the chronic *constitutional* neurasthenia, which is usually hereditary.

1. Cerebral Symptoms.—These are most important, because the disease is a psychoneurosis. Headache is almost invariable, is more often dull than acute and produces a sense of pressure ("lead cap" headache) or intracranial paræsthesia and in localization is most often occipital. Vertigo is frequent. *Insomnia* is extremely common, but rarely absolute, which always suggests organic brain disease; sleep is slow in onset, the patient being kept awake by his fear of sleeplessness, by a rapidly moving circle of uncontrollable memory pictures or by indigestion or palpitation; sleep, when once attained, is not resting or is too short and the patient awakes in the night or early morning. It is broken by dreams, startings or sometimes emissions. In mild cases distinct *alterations in temperament* and character develop, as *loss of courage* and confidence, depression, *irritability,* undue response to emotional stimulation, as on seeing sad plays or hearing hard-luck stories, slight ethical blunting, tendency towards extremes and development of quiet self-concentration or obtrusive egotism. The brain tires readily on mental or bodily exertion, especially as the irritable patient is often prodigal of time and energy. It is remarkable how in some cases the morning depression may disappear before an afternoon or evening excitement, but this is notably followed by reaction. Psychical processes are disturbed; perception is blunted and memory is weakened or often temporarily lost (dys-, para-, amnesia), as shown by the copious notes of his symptoms which the patient produces in the physician's office (*"l' homme aux petits papiers,"* as Charcot called him); association and combination are difficult, as in thought, composition, judgment or conclusions, whence the speech is often slow and disjointed, names are forgotten and the patient rereads addresses, runs repeatedly to see if the door was closed, etc. Compulsory conceptions may lead to homicide or suicide in highly psychopathic types. Fear, being an attribute of the weak, is common enough, and may in marked types assume the form of various phobias, as the fear of open places (agarophobia), of enclosures (claustrophobia), rivers (potomophobia), high places (acrophobia), stars (asterophobia), railways (siderdromophobia), dirt (mysophobia), of high things falling (batophobia), of standing (stasophobia), or walking (basophobia), of darkness (nyctophobia), of a single thing (monophobia), of all things (pantophobia), of places (topophobia), of men (anthrophobia) or of disease (nosophobia). The *special senses.* Fibrillation of the lids and wide, variable, sensitive and unequal (3 per cent.) pupils are frequent, although permanent irregularity is almost always organic. Asthenopia is very common, especially in hypermetropia from weakness of the ciliary and int. recti muscles. The field of vision may be concentrically limited but this is rarely marked or permanent, and Förster's shifting type may be found, in which objects brought from without into the field are better seen than those moved from within the field outward. Hyper-

æsthesia of the retina and ear, ringing in the ears, Ménière's complex and disturbance of taste or smell are not common.

2. Spinal Symptoms.—These include the so-called "spinal irritation." Many symptoms ranked as spinal are essentially cerebral. In women they are more common and are localized chiefly to the upper dorsal and coccygeal regions; in men they are lumbodorsal and most frequent in the sexual type. *Sensory* symptoms are practically constant. Hyperæsthesia is more common in the bodies of the muscles than in their attachments or in the skin; paræsthesia is frequent, as tinglings, girdle sensation or even itching. Paralgesia in the form of backache (rhachialgia) is as frequent as headache and the pain may radiate into the various nerve plexuses; it is very often sacral; the spine may be exquisitely sensitive to pressure. Paralgesia is common in the skin about the hair roots, in the trunk and extremities. Neuralgia is rare, although it sometimes radiates from the spine into the limbs. Anæsthesia is no part of neurasthenia. Of the *motor* symptoms, muscular weakness or myasthenia is the rule. It is probably spinal and cortical in origin and is especially conspicuous in the morning. The muscles often show fibrillary contractions and a *tremor* (85 per cent.) which is as fine and frequent as that of alcoholism or Graves' disease. Muscular fidgets, starts, cramps and increased irritability of the nerve trunks, as in tetany, are also observed. Actual ataxia is most rare, although suggested by the writing. The akinesia algera (Möbius) or the abasia-astasia of Blocq, as in hysteria or the psychoses, is the result of a phobia or a fixed conception. One of Erb's patients remained in bed for fourteen years. The skin, tendon and periosteal reflexes are increased.

3. Vasomotor Symptoms.—These produce many central, visceral and peripheral manifestations. Mosso proved that during brain-work the cerebral vessels normally dilate while those of the arm contract. According to Anjel this does not occur in neurasthenia. The manometer in normal cases reveals no essential variation during work, while in neurasthenia marked manometric variations occur (Weber). These findings are valuable because they explain many otherwise vague findings. Neurasthenics often suffer from cold and wear more clothes than is necessary; their skin is cold, even cyanotic or locally asphyxiated. On the other hand, the skin may be flushed and hot. Cold and clammy sweats over the forehead, hands and feet develop without exertion. Salivation or a dry mouth, polyuria, pseudo-angina pectoris (vasomotor changes in the cardiac and brachial plexuses), the wide temporal vessels, the epigastric pulsation, circumscribed œdema, urticaria and dermographism are but vasomotor manifestations.

4. Visceral Manifestations.—(*a*) Some of the *cardiovascular* signs and symptoms have already been treated. The arteries are relaxed and often throb, the pulse may be slightly of the "water-hammer" type and capillary pulsation is often apparent in the lips or under the nails. Palpitation and precordial anxiety or dyspnœa

are frequent. The pulse is frequently faster and may increase 10 or 20 beats on pressure over some sensitive area (*v. i.* Traumatic Neuroses). Many neurasthenics seek consultation for cardiac disease. Thomeyer describes arrhythmia on leaning forward, orthostatic tachycardia (on standing) and inspiratory acceleration and expiratory retardation of the pulse. (*b*) *Gastric symptoms* (in 60 per cent.) are functional in character and were first described by Leube. The chemism may be normal or show achlorhydria or hyperchlorhydria. The stomach is frequently hyperæsthetic. Anorexia is usually mental, as are polyphagia, polydipsia and adipsia (see Neuroses of Stomach). (*c*) *Intestinal symptoms.* Diarrhœa is less common than constipation, which, like previous sexual abuses, often occupies the patient's thoughts. They "would be perfectly well if they could have one natural movement," and when they do they complain of "exhaustion during the entire day afterward." Assimilation is often incomplete, as shown by the stools; a sense of pressure, Kussmaul's peristaltic unrest, hyperæsthesia, burning and flatulency torment the patient. Enteritis membranacea and Glénard's enteroptosis are not infrequent. (*d*) The amount of urine is often increased, or seemingly so, yet equally often the 24-hour quantity is decreased. The specific gravity is lowered, but the urates, uric acid and urea are increased. Phosphaturia and oxaluria occur; the latter is often attended by flatulent dyspepsia, melancholia and nervous depression. Vesical tenesmus is an occasional complaint. (*e*) *Genital symptoms.* Fear of the results of previous excesses or of self-abuse may dominate the patient's mind, but the trouble is more a matter of conception than of reality, since the sexual appetite is actually decreased as a rule. In most cases there is premature ejaculation and in some patients there is actual or psychical impotence. Emissions are frequent, but their importance is greatly overestimated.

Course and Prognosis.—*Acute cases* are rare and fatal cases extremely exceptional. The *usual chronic course* is one of slow onset, it is attended by evidences of nervous fatigue which are at first relieved by rest, but which later persist in spite of rest and incline the patient to stimulants or drug habits. Later the signs of cerebral irritability, disturbed sleep, vasomotor disorders, reduced mental and psychical capacity, pains, spinal tenderness, paræsthesia or paralgesia, sympathetic dyspnœa or palpitation develop, sometimes with temporary remissions. The outlook is poor in severe hereditary forms or when there are complicating psychoses or drug habits. In other types recovery is usual, although long deferred. Complication with other neuroses must naturally modify the prognosis.

Diagnosis.—The diagnosis is generally made with ease, but several rules must be borne in mind: (1) No single symptom is pathognomonic; (2) there must be no sign of organic disease; (3) neurasthenia may precede, follow or complicate organic nervous or visceral disease. The *subjective* signs, irritable weakness, emotivity, muscular weakness, headache, backache, insomnia, amnesia, impaired psy-

chical coördination and the phobias, together with *objective* findings, as asthenopia, insufficiency of the internal recti, tachycardia, epigastric pulsation, vasomotor alteration, as disturbed secretion of urine or sweat, increased reflexes, fibrillation and tender muscles cannot be mistaken. The neurasthenic prodromes of *progressive paralysis* (*q. v.*) are usually distinguished on careful search for organic disease. *Hysteria* in its stigmata and accidents is so typical that elaborate differentiation is superfluous; anæsthesia, persistent contraction of the fields of vision, dyschromatopsia, achromatopsia, monocular diplopia, convulsions, paralyses, contractures and anuria are foreign to neurasthenia. *Hypochondriasis* differs from neurasthenia in these essential points: it is a pure psychosis, its concepts are primary, ill-based, illogical or absurd and cannot be eradicated even for a time.

Treatment.—1. Prophylaxis.—Prevention is difficult, for the neurasthenic cannot control his ancestry, but even with hereditary stigmata much may be done. Treatment starts with the education of both child and adult. Children are mimics and the clearest of all observers. The inculcation of self-control, fearlessness, self-sacrifice, moderation in all things, truthfulness, prompt decision and constancy of purpose must be based on actual parental example, and in this way the child should also educate the parent. The child must learn to give up, to sleep alone, to stop at the right time, to finish what he has begun, to obey and to learn that he is not the centre of the family. He should be taught without being forced. Out-door living is more important than the school room. The English method of treating children with reserve has some advantages over American demonstrativeness. Simple varied food, without tea, coffee or alcohol, and long sleeps develop a strong nervous system. The sexual instinct requires attention. Parents or patients would do well to read Patrick's small classic, "How Not to be Nervous," *Jour. Amer. Med. Assn.,* Feb., 1903.

2. Therapy.—When once manifested, the disease should be treated as above outlined and causal factors should be investigated. Work alone is seldom injurious and while it is said that the modern complexity in the struggle for existence is increasing the number of neurasthenics, work should not bear the stigma nor should disappointment, for both make character. Work must be systematic but not slavish. Worry, hurry, failure to learn individual limitations, uncontrolled planning beyond the immediate day's work and worry (the irritable weakness of work) cause the damage, especially when work produces indifference to nature, family life, exercise, rest and the things higher and above one's own small life and ambition. The time for exercise should be taken from the work hours, it should not follow them, nor should it be enforced in every case, for some are hurt by it. Of course, work under stimulation is injurious. The chief element in treatment is psychical and the physician accomplishes more than do drugs. He carefully examines and reëxamines the patient and his assurance that

no organic disease exists has its weight for the time. Conversely, careless diagnoses, as of fatty heart, with no objective justification, work damage to the patient's mental balance. The patient's hygiene should be regulated. Strict dietaries and stimulants should be avoided, moderate out-of-door life or exercise enjoined and in very pronounced cases absolute rest from work, with sojourn near the sea or at a moderate altitude in a place not subject to sudden changes or high winds is recommended. Sexual matters should be left alone unless gross violations are found. The patients need sympathy, which they should receive—in moderation for their sake; but important as is the psychical element in therapy, the patient must be taught that he alone can cure his malady by self-control and by avoiding extremes in each and every thing.

Pronounced cases in thin women, especially those with gastric symptoms, are often helped by the Weir Mitchell *rest-cure,* which consists of absolute rest in bed, isolation, forced feeding, massage and faradization. The patient is not allowed to lift a hand and sees only the nurse, who is selected for her strength, optimism and lack of nerves. The patient is fed milk every three hours and the muscles are rubbed and faradized. Thus weight and strength are gained without muscular degeneration. Few men will tolerate this method and obese do not fare as well as lean women. In certain cases it is of distinct benefit. However, no greater mistake is made than to give all cases the rest-cure; they often want work and occupation, harmonized with rest.

3. Diet.—That no one dietary is essential is shown by the different recommendations; some physicians favor a meat diet, others avoid meat, especially in cardiac types and give well-cooked vegetable albumins, cooked fruit, fats, milk and eggs. In mental anorexia, feeding by mouth or rectum must be forced. Water in abundance is indicated. *Gastric hyperæsthesia* should be relieved by small doses of carbolic acid and gelsemium. Eructations must be controlled, since the air admitted exceeds the gas expelled; asafœtida is excellent because it makes as strong an impression when swallowed as when eructed. Electricity is largely suggestive in its effects. With massage and vegetable diet, endorectal faradization relieves *constipation.* In cardiac types alcohol and tobacco are interdicted and small doses of belladonna and bromide usually control *palpitation;* other vasomotor symptoms are relieved by these remedies or by ergotin or nitroglycerine, according as they are angioparetic or angiospastic. Arsenic is valuable in vasomotor troubles with anæmia.

Sexual symptoms are treated by psychical measures and by sitz-baths. *Insomnia* is often difficult to relieve. In the order named, the following measures are valuable: avoidance of evening work, stimulants or excitement; a cool bath, the wet pack or an ice-bag over the heart; small doses of bromide with valerian for the "expectant attention" which wards off sleep; 20 grains of sulphonal in hot milk, which should be slowly sipped; codeine with hyoscine; and chloral

(the continued use of which induces vasomotor disturbance). The habit of sleep must be cultivated and the patient must not get up to read. Local, urethral and uterine affections usually receive excessive therapeutic attention.

THE "TRAUMATIC NEUROSES."

This is a practical grouping of traumatic neurasthenia, hysteria, chorea or hypochondriasis. Hysterical forms were first described by Walton and Putnam; Erichsen (1866) wrote on the subject of trauma to the spine, on which the illogical name of railway spine was later conferred, and Oppenheim (1877) termed the group the "traumatic neuroses." All organic affections must be excluded, whether trauma may seem directly or indirectly causal, as vertebral caries, tumor, Bechterew's chronic ankylosis, Kummel's rarefying osteitis which Kocher refers to fracture; hæmatorrhachis, meningeal hæmatoma or pachymeningitis; and cord affections, as poliomyelitis, myelitis or lateral sclerosis. Experimental trauma may produce molecular nervous changes (Schmauss, Strümpell, Bruns, Vibert and Crocq), a fact which may be of clinical importance, and trauma is thought to hasten arteriosclerosis (Friedmann). Alcoholism and syphilis are predisposing factors. *"Traumatic neuroses" are purely functional affections caused by trauma,* which operates psychically rather than somatically. The symptoms are those of hysteria, neurasthenia and hypochondriasis, singly or in combination. The patient may feel well for a day or two and then pain develops at the seat of injury or in the spine; anxiety, fear for his financial future or for his family, a "fixed idea" of his incapacity and the development of "litigation symptoms" are typical of hypochondriacal neurasthenia.

Symptoms.—The *psychical symptoms* are usually prominent; they are accentuated by the effect on the patient of repeated, sometimes suggestive, physical examinations, visits of lawyers or adjustors, court room suspense, reversals of judgment or appeals. Vertigo, backache, headache, sleepiness by day and nocturnal insomnia may cause great distress. *Sensory symptoms.* Pain, which seems to be nervous, may prove organic on examination by the *x*-rays; it may be simulated, but is often real, as shown by persistence after award of damages. Tender areas are difficult to pass upon, because of exaggeration or simulation; Mann, Köpf and Rumpf found that pressure on areas which are actually tender increases the pulse rate by 10 to 30 beats; this sign is subject to several conditions: (*a*) it is not always present in real pain; (*b*) its absence is no proof of simulation; (*c*) in simulation, the agony and writhing which occur when certain areas are touched may increase the heart's action. Paræsthesia and hyperæsthesia of the skin or special senses are frequent. Anæsthesia indicates traumatic hysteria. Hysteric or neurasthenic contraction of the visual fields may remain the same from day to day and not vary with approach or withdrawal of the test object; this is not a sign of simula-

tion, as suggested by some writers; Förster's type cannot be assumed (*v.* NEURASTHENIA). In all cases the influence of suggestion must be borne in mind. The pupils may vary, especially by dull illumination; reflex immobility indicates organic disease. *Motor symptoms* may embrace all those of hysteria or neurasthenia, abasia, astasia, paralyses, contractures suggesting vertebral injury or joint disease (Brodie's joints), pseudo-spastic or -tabetic gaits, tremor (*forme trepidante*) or sometimes muscular atrophy (especially about the joints). Hysterical convulsions are rare; the author has twice seen choreiform affections in telephone girls who sustained slight shocks and once a genuine chorea. Patients must often be watched; one of the author's cases, with spastic gait, shuffled out of the office but walked perfectly well around the corner. The *reflexes* are often increased, perhaps asymmetrically; ankle clonus, which is probably spurious, is sometimes reported. Cardiac symptoms may be prominent and cannot be simulated. When there is albuminuria and glycosuria the question naturally arises whether they were present before the trauma.

Diagnosis.—The diagnosis concerns several points which are often difficult or impossible to establish, because objective findings are frequently few. (*a*) It is usually possible to state whether one of the neuroses is present or whether organic disease obtains. (*b*) Exaggeration and simulation are difficult to estimate, as shown by the change of professional opinion from the idea that all or most cases are simulated to the present statement of some writers that these factors are present in 4 (to 10) per cent. of cases only. (*c*) If disease is present is it directly due to injury? (*d*) If so, the most difficult of all questions is presented, namely, the degree and duration of the disease and the damages to be awarded.

Prognosis.—(*a*) *As to life,* the outlook is usually good, though some cases become insane or suicidal. (*b*) *As to complete recovery,* it is less favorable. In most cases it is surprising how soon symptoms disappear after satisfactory legal settlement is made; this is said without cynicism. Many persons do not then recover completely and symptoms may endure in those who persistently shrink from litigation. Hysterical symptoms are more favorable in prognosis than the neurasthenic or hypochondriacal. It is maintained by some that cardiac overaction may induce organic myocardial changes and that arteriosclerosis may develop into precocious senility (dementia præcox).

Treatment.—Treatment is similar to that of hysteria or neurasthenia and is therefore largely psychical. The physician, as a rule, dreads the effect of litigation on the patient, assures him that the symptoms are seldom severe or enduring, that there is no organic lesion and advises early settlement and speedy return to work.

EPILEPSY.

Definition.—Epilepsy, by derivation, means "being seized upon"; it has been called the "falling sickness" and morbus sacer. It does not include Jacksonian or symptomatic epilepsies, which will be considered in its differentiation. Though it is classified with hysteria and neurasthenia as one of the "three great diffuse neuroses," it will probably be finally shown to include more than one disease. It is a *disturbance of balance between the central exciting and inhibiting nervous processes* attended by (*a*) a typical *chronic convulsive disorder,* affecting all parts of the central nervous system, each attack being followed by *loss of consciousness,* (*b*) *partial or atypical manifestations* of these characteristics, or (*c*) concomitant or consecutive psychical or other symptoms. It occurs in from one to six persons per thousand.

Etiology.—The *ultimate causes* must be separated from the inciting causes (Nothnagel). Of the former, the *neuro-* or *psychopathic tendency* of Griesinger is the most important, being found in 37 per cent. of cases and lessening the nervous resistance. As in tuberculosis, only the tendency is transmitted by inheritance, rarely the disease itself. Neurotic or psychopathic antecedents, parental alcoholism (51 per cent.), morphinism, plumbism, syphilis, tuberculosis, gout, arthritism, diabetes, trauma during labor or ovarian and testicular diseases lessen the nervous resistance of the offspring. Though the disease may appear at any age, it rarely occurs after the thirtieth year. Seventy-five per cent. of cases (Gowers) develop before twenty; 46 per cent. between ten and twenty, mostly at puberty; 28 per cent. before ten. The sexes are about equally affected.

Inciting causes include (*a*) *infections,* as malaria or typhoid; syphilis may be hereditary, induce the Jacksonian type from cortical gumma, or it may be parasyphilitic; (*b*) *intoxications,* especially alcoholism; (*c*) *exhausting affections,* as rickets, and diseases of the blood or of metabolism; and (*d*) *trauma* or fright. Inciting causes are found in only 37 per cent. of cases (Gowers).

Reflex epilepsy is said to occur from painful scars, nerve tumors, or foreign bodies in the ear, nose, nasopharynx or larynx; diseases of the teeth, digestive tract, uterus and heart; onset of the menses or their monthly appearance, pregnancy, phimosis or onanism; from gall-stones, colic or abdominal paracentesis. The importance *per se* of reflex disturbances is daily lessening.

Pathology.—There is no characteristic macro- or microscopic change. The usual finding after death is that of any convulsion, *viz.,* venous engorgement and punctate ecchymoses. Induration of the cornu ammonis, induration in the medulla, neurogliar increase in the cortex, thickening, asymmetry or deformity of the skull and persistence of the thymus have not been proven causal.

Symptoms.—The symptoms are best studied from the *completely developed attack,* in which there are three component parts: (*a*) The

prodromal stage is not always present. Its "warnings" are either distant or immediate. The distant warnings (in 10 per cent. of cases) may precede the seizure by hours or days, are possibly due to metabolic changes in the cortex and take the form of restlessness, dispositional changes, depression, thoracic or cephalic oppression, insomnia, vertigo, nausea, grinding of the teeth, hyperæsthesia of the special senses, angioneurotic palpitation or cerebral congestion. The immediate warning is the *aura* proper, which is the beginning of the convulsion rather than a warning. It is a central cortical irritation; its frequency is 40 per cent.; it is rarer in sudden, stormy, generalized seizures. Its forms are varied. The psychical aura is rare and is manifested by emotional or intellectual alteration and anxiety. The sensory aura consists of paræsthesia and a sense of deadness in a limb or joint, attacks of migraine and epigastric sensations (from the pneumogastric nerve, pressure or irritation of which sometimes inhibits the convulsion). The special senses may be irritated or blunted; for instance, there may be flashes of light or colors, hallucinations, blackness before the eyes and less often anomalies of hearing, taste or smell. The motor auræ are circumscribed clonic or much less often tonic twitchings, as in Jacksonian epilepsy, from which they are distinguished by a rapid loss of consciousness; automatic movements, like stamping or buttoning the coat, are rarer and motor weakness and singultus, coughing or sneezing are least frequent. Vasomotor auræ may be localized or generalized, as rushing of blood to the head, palpitation, anæsthesia, increased temperature, increased arterial tension, pallor or chilling. The patient then falls, *pale and unconscious,* or is thrown with force, often on his face (Trousseau), when (*b*) the *convulsive stage* begins. There is first a *tonic* convulsion, which affects all voluntary muscles and is due to infracortical irritation. The initial *cry* is guttural or groaning, results from tonic abdominal, thoracic and laryngeal spasm and is heard in 50 per cent. of cases. The head is held back; the eyes are open and staring, the jaw set, the face flushed, the neck tense, the body often opisthotonic, breathing and pulse are suspended, the muscles are hard but slightly vibrating to the palpating hand, the arms are turned tetanically inward, the hands are clenched with the thumb adducted and under the fingers, the legs are extended, the thighs adducted and the toes flexed or extended and spread apart. In some cases the head, eyes and even trunk deviate and in a few instances the tetanic spasm spreads gradually, instead of the patient having the usual instantaneous tonic spasm. The tonic phase lasts ten, fifteen or thirty seconds, relaxing in inverse order from the limbs to the neck and face. The second phase of *clonic convulsion* occurs at once, appearing first in the limbs, which are not yet quite free of the tonic spasm. This stage is usually bilateral, highly irregular and violent, with very short remissions, leading perhaps to injury, fracture, luxation, breaking of teeth, laceration of skin, tongue and cheek, or rupture of muscles. It consists largely of alternating flexion and extension; the head strikes the

ground, the body turns violently, the eyes jerk and protrude, the face is distorted, the tongue is bitten as it is thrust out, bloody saliva is expressed by the masseters, inarticulate sounds arise from the movements of the diaphragm and respiratory muscles and gurgling results from intestinal movements; vomiting movements, filling of the stomach with air, cyanosis, swelling of the jugular veins, ecchymoses in the conjunctiva, skin, retina or larynx, involuntary and sometimes forcible evacuation of the bladder, less often of the rectum, and occasionally ejaculation, occur. The clonic phase lasts one-half to three minutes, ending in a fine generalized tremor. (*c*) The *comatose* stage often begins with a deep sigh; respiration and circulation become normal, the limbs relax and the condition resembles a sound sleep, from which the patient awakes in from fifteen minutes to several hours, sometimes suddenly, more often gradually and in a more or less dazed condition. He recalls nothing of the attack and the loss of memory may extend back of the seizure (retrograde amnesia). He experiences pain in the muscles, joints or head, nausea, mental irritability or depression and finally a group of exhaustion symptoms, to which Féré has especially directed attention; these are (i) sensory exhaustion, expressed by hypæsthesia, anæsthesia, hypalgesia or analgesia. (ii) Exhaustion of the special senses, as concentric limitation of the visual and color fields, especially in abortive attacks, dulling of hearing, taste or smell, and (iii) motor exhaustion which also is due to cortical fatigue.

The seizure has been described with special reference to the dominant motor changes, but during the attack other findings are observed. *Vasomotor* constriction often occurs, as shown by the initial pallor of the face, cardiac overaction or perhaps irregularity. The ecchymoses and retinal congestion are due to the convulsion. The *reflexes* are affected. The pupil is almost always reactionless, is often narrow at the onset, and usually dilates widely in the second stage; afterwards it may oscillate; the pupils are sometimes unequal (20 per cent., Féré). The tendon and skin reflexes are weak or lost in complete attacks and for hours afterwards, whence we may judge of the severity of the seizures. The temperature is usually considered normal, but may be elevated 2 or 3°. In the urine, Voisin found albumin in 50 per cent., though others consider it infrequent. The author has often noted transient albuminuria and cylindruria (granular casts) after marked paroxysms; glycosuria is uncommon. The phosphates and nitrogen are increased; polyuria is common in the third stage. The sweat is increased.

Types of Epilepsy.—1. The completely developed attack (*epilepsia gravior, grand mal*) consists of (*a*) the classic type (29 per cent.), as above described, with complete loss of consciousness, and tonic followed by clonic convulsions; or (*b*) the well-developed but slightly atypical type (19 per cent.), in which all the classic components vary somewhat; for instance, the spasm may not always be generalized.

2. *The rudimentary attack* (21 per cent.), consists of loss of con-

sciousness, plus tonic or clonic spasms. This includes the apoplectiform cases, especially the senile (arteriosclerotic) form; forms resembling myoclonus; cases which do not fall but stumble along at the onset (epilepsia procursiva) or turn (epilepsia rotatoria).

3. The third form is the *abortive attack* (epilepsia mitior, *petit mal,* 31 per cent.). Its importance is underestimated by the profession and the public; two main types, with endless variations, are distinguished: (*a*) one with loss of consciousness with few or no motor signs, and (*b*) motor and vasomotor signs with no loss of consciousness, or very slight clouding of the intellect. The loss of consciousness is often very slight or may last for a few seconds, so that a scarcely perceptible break occurs in the patient's occupation, reading, speaking, playing the piano or a game of cards; it is described by the French as "*absence.*" He may move his lips inarticulately or there is slight nystagmus, a chewing or swallowing movement, tremor or twitching of the face, monospasm or vertigo. In children, a cry may be the sole symptom. Some patients seem to fall suddenly asleep. Nothing, or everything, may be remembered. Auræ are very common and may constitute the whole attack, which aborts without unconsciousness or spasms. Vasomotor symptoms are very common.

4. The *psychical equivalent* of Sammt, also known as transformed or larvated epilepsy or the epileptoid condition is often mistaken. Mental symptoms may precede, succeed, alternate with or replace the ordinary convulsive type, which must be first recognized to establish this variety. The mental changes are slight (*petit mal intellectuel*) or marked (*grand mal intellectuel*) and include excitement, emotional outbursts, narcolepsy, incoherent conceptions, amnestic attacks and mania. The patient may act like an hypnotized subject; he may take sudden long journeys or commit assault, arson or murder, whence the great medico-legal significance of this variety. Milder forms are said to occur, as sweats, stupor or vacuity at the menstrual epoch.

The Interparoxysmal State.—If the patient is not seen during his seizures, evidences of epilepsy should be looked for carefully. Signs of psychical degeneration, poor differentiation of the ear, very large, small or prominent ears, hæmatoma of the ear, deformed teeth, lips or palate, coloboma, congenital strabismus, nystagmus, small eyes, irregular pigmentation of the iris, deformed genitals or fingers, disorders of speech or irregular facial innervation may indicate hereditary taint. Neurasthenic symptoms are not unusual. The expression is often dull. When idiocy occurs with epilepsy there are two possibilities; first, that both are coördinate results of a common cause, or second, that early, chronic and repeated attacks have initiated mental degeneration. However, many brilliant men have been epileptics, as Julius Cæsar, Napoleon, Carl V, Peter the Great, St. Paul, Mohamed, Petrarch and Rousseau. The importance of organic disease of the heart, lungs, stomach and kidney is difficult to estimate. Epileptics often sweat profusely, are usually constipated

and frequently become obese from inordinate eating and the bromide treatment.

Diagnosis.—The diagnosis concerns the convulsive seizures and the abortive types. Similar convulsions occur in numerous other conditions and must first be excluded. They occur in (*a*) *organic brain disease*, as syphilis, tumors, vascular lesions, paretic dementia, multiple sclerosis and infantile cerebral paralysis. These affections may produce generalized or Jacksonian fits. *Jacksonian epilepsy* is more clonic (from cortical irritation) than tonic (due to infracortical irritation), is often associated with paresis or sensory alteration, occurs less often with coma unless its generalization is unusually wide and in 50 per cent. of cases is due to small cortical tumors. Epilepsy rarely develops after thirty years of age (E. tarda) and 90 per cent. of cases which occur after thirty,—excluding alcoholism and uræmia,—are syphilitic (Fournier, H. C. Wood). After forty-five, vascular lesions are a very common cause. (*b*) Convulsions may occur in alcoholism, *uræmia* (eclampsia), plumbism or drug poisonings. (*c*) *Reflex epilepsy* results from dentition, worms or digestive disorders; in some cases genuine epilepsy seems to follow the reflex form (which perhaps is epilepsy in itself).

Simulation of epilepsy is impossible. In the complete type, the tongue biting, cry, stertor or involuntaries may be absent. Scars are often of diagnostic value. Nocturnal epilepsy occurs in 33 per cent., often coming on when the patient drops off to sleep or on awakening in the morning; it easily escapes recognition for years, but the depression, bitten tongue, ecchymoses and involuntary evacuations are unmistakable.

Differentiation from hysteria, which is usually easy, may be difficult, indeed the two affections sometimes co-exist; the following table is combined from Gowers and Biswanger:

Epilepsy.	*vs.*	Hysteria.
Distant warnings: few, mental.		Frequent, emotional.
Aura: more frequent, momentary, oftenest unilateral or epigastric.		Less frequent, longer, bilateral, foot aura, globus.
Cause: none.		Emotional.
Onset: always sudden, often with pallor, cry which is never verbal, immediate falling and complete unconsciousness, with rigidity, etc.		More gradual, rarely with pallor or cry, which may be repeated or verbal, and recurs later. Rarely brusque falling, slower and far less complete unconsciousness, and may react to suggestion or pressure on hysterogenous zones.
Tongue biting, frothing: very common.		Exceptional, or seemingly purposive.
Convulsions: generalized, tonic followed by clonic spasms, very rarely tonic alone; flexor spasm predominates.		Rigidity, to carry out semi-intentional movement; struggling; clonic, irregular spasms, seemingly fragments of partial movements of emotion; extensor spasm predominant "*arc de cercle,*" clownism, passional attitudes, etc.

EPILEPSY.	*vs.*	HYSTERIA.
Reflexes: pupils contracted at onset, dilated later, immobile (in clonic period). Tendon and skin reflexes absent during and after attack.		Pupils usually wide and react (may be immobile, Westphal, Karplus). Sometimes convergent strabismus. Reflexes rarely absent; or increased.
Involuntary evacuations: frequent.		Absent or most exceptional.
The urine: solids always increased, both nitrogen and phosphates.		All solids decreased and altered ratio between earthy and alkaline phosphates.
Temperature: sometimes elevated, 2–3°.		Practically never.
Coma: complete.		No real coma.
Duration of seizure: very short.		Longer, recurrent, relapsing.
Post-paroxysmal: sensory and motor exhaustion, ecchymoses, absent reflexes, mental incapacity, with no memory of transpired events.		Depression, fatigue, pain in head or stomach, nausea, some memory of attack.

The most difficult forms to differentiate are *petit mal* and transformed epilepsy, in which the history or observation of major attacks is important. *Petit mal* may be confused with cardiac syncope, labyrinthine vertigo, etc.

Course and Prognosis.—When one attack has occurred a second may be expected in 33 per cent. of cases in less than one month, in 33 per cent. in from one to twelve months, in 33 per cent. in over one year; and when epilepsy is once established, daily attacks occur in 10 per cent., in 50 per cent. they occur at intervals of less than two weeks, and in 75 per cent. they occur at intervals of less than one month (Gowers). The mode of life, stimulation, excitement, pregnancy and sexual excesses may increase their frequency and cessation during fevers is not uncommon. In some, fortunately rare, cases the attacks occur in quick succession (*status epilepticus, état de mal*), as many as 10 or over 100 occurring in one day. In one case 2,500 seizures occurred during a month; the stage of coma may last from two or three days to a week, with a temperature of 105–7° and death then results. The *outlook* (*a*) *as to recovery* is poor. Five to 10 per cent. of cases may recover spontaneously (Hufeland, Dana, Müller). The prospects are best in cases which begin before twenty in males, in cases with long intervals between attacks and, though it may seem strange, in hereditary forms. After two years duration the outlook is unfavorable. (*b*) *As to life,* the prognosis is more favorable. A few cases drown, suffocate from turning on the face or are fatally burned during the seizure. Some patients die during the insult from vascular lesions of the brain or less often from heart rupture, respiratory cramp, œdema of the lungs or vomiting into the larynx. Epileptics frequently become tuberculous. In 10 per cent. dementia occurs. The average duration of life is probably shorter than normal. Bodily health and intellectual vigor may, however, be remarkably preserved.

Treatment.—1. Treatment of Cause.—This has a limited scope. Syphilitic epilepsy, unless due to a gumma or to the secondary anæmia, does not respond to antisyphilitic remedies. Avoidance of alcohol may be almost curative in this form. Reflex factors should be removed. In Sweden epileptics cannot marry; 50 per cent. of hereditary epileptics transmit some transformation of the taint to their offspring.

2. Hygienic Therapy.—New cases in children should receive the general care outlined under the other neuroses. Work in school should be limited to a few hours and country is preferable to city life. For very poor subjects, institutional treatment is indicated. Most neurologists restrict the consumption of meat, although Gowers allows it once or twice a day. Toulouse and Richet withdraw salt and give a milk and cereal diet, which they think promotes the affinity of the bromides for the brain cells.

3. Bromide Treatment.—This was introduced by Laycock (1853) and was developed by Voisin. The bromides act directly on the nervous tissue; they depress the cortical cells, inhibit reflex action in the brain and cord and lessen centripetal conduction. *Untoward symptoms may result even within therapeutic limits.* Intellection may be dulled and muscular depression occur, but these may often be counteracted by administration of coffee; the frequent stomach symptoms can be avoided by free dilution of the bromide in water; diarrhœa, which results especially from potassium bromide, is relieved by opium if marked, or if less, by salicylate of bismuth and betanaphthol, āā gr. x, after meals; in its elimination by the skin, acne is common, which is lessened or removed by giving large quantities of water and small doses of arsenic and by washing the skin with green soap. Belladonna in small doses will modify the respiratory catarrh or salivation. The anaphrodisiac effects cannot be prevented. In some individuals there are *marked idiosyncrasies,* as mental excitement, hallucinations, rapid emaciation ("bromide cachexia") with lessened physiological resistance to acute diseases. *Toxic symptoms* may be *acute,* resembling those of paretic dementia, but more commonly are chronic; *chronic bromism* is most likely to develop in those with weak digestion or circulation and when the drug is given carelessly for a long time, because its elimination is slow. In the former, hydrotherapy, massage and tonics should be used as preliminary or concomitant measures. Cardiac symptoms are due almost wholly to the potassium salt, but especial care in the use of all bromides is indicated in cases of senile epilepsy (myocarditis, arteriosclerosis, endocarditis) or of great nutritive exhaustion, in which combination with digitalis or strychnia is indicated. In bromism the reflexes are decreased, particularly the pharyngeal reflex; Englehardt has shown that it is weak in 17 per cent. and absent in 25 per cent. of normal individuals, whence Ziehen watches the corneal reflex, which is never normally absent. In advanced bromism there are low temperature, bronzed skin, vasomotor relaxation, somnolence, apathy,

paralytic speech, muscular relaxation, ataxia, tremor, fœtor ex ore, anorexia, slow respiration (the usual cause of death), weak heart, abolition of sensation in the skin and mucous membranes and of all reflexes and decrease or suppression of urine. The drug therapeutically unites with the nerve cell and depresses its activity and metabolism and toxicologically it causes its degeneration or atrophy. The drug must be used for months or years and the symptoms above enumerated must be watched for; it seems that epileptics have a certain tolerance for bromides, which Voisin considered almost as a food in this affection.

Results.—Gowers noted immediate relief in 43 per cent., improvement in a short time in 47 per cent. and no effect in 10 per cent. of his cases. He gives a dram a day of the potassium salt, for two years, for he finds that larger doses are no more efficacious; in nocturnal epilepsy a large dose should be given at bed-time. Seguin's results were: cessation of attacks in 23 per cent., notable decrease in 40 per cent., no great effect in 26 per cent., no effect in 10 per cent. Charcot's method was to give a daily dose of one dram, which was gradually increased to nearly two, and was then slowly decreased to the original amount. Erlenmeyer's formula is extensively used and gives the best results (sodium and potassium bromide each two parts and ammon. bromide one part). The best results are obtained in cases treated early in their course and early in life and in those in which there are long intervals between attacks. Opium, belladonna, nitroglycerine, digitalis, thyroid extract and chloral are of but secondary importance, although with special symptoms they may reinforce the bromides. Nitroglycerine may be useful in *petit mal.* Children tolerate large doses of bromides.

4. Treatment of the Attack.—This is confined to cases in which auræ are present. The patient is placed on the bed or ground to avoid injury and a towel inserted between the teeth to prevent biting of the tongue. Compression or manipulation of the limb in which the aura is felt may in a few instances avert the spasm. Charcot placed an ice-bag over the head and one over the heart. Inhalations of amyl nitrite, ether or chloroform are sometimes beneficial.

5. Surgical Indications in Traumatic Epilepsy (Jacksonian or generalized).—Biswanger's conclusions are (*a*) that results are disappointing; only a small percentage of cases actually recover; operation is indicated (*b*) when convulsions emanate from a small cortical focus; (*c*) when the convulsions are of a type intermediate between the Jacksonian and generalized forms; (*d*) when, with generalized convulsions, there is a suggestive scar, bone defect or localized tenderness on percussion; (*e*) since the internal lesion does not always correspond with the external scar, the site of trephining should be governed by focal symptoms, motor or sensory auræ, or "exhaustion" signs; (*f*) cysts, tumors, bone, membrane or scars may be removed, but not the cortex (as recommended by Horsley); (*g*) operation must be followed by the bromide treatment; (*h*) operation is contra-indicated in ordinary epilepsy.

INFANTILE CONVULSIONS.

In young children the nervous tissues are incompletely developed, many axis cylinders are not yet clothed with myelin sheaths and the lower dominate the upper segments, thus predisposing the child to disproportionate nervous reaction.

Etiology.—(*a*) *Rickets* as a cause was especially emphasized by Wm. Jenner. Many cases of carpopedal spasm, tetany and laryngismus stridulus have a rhachitic basis; this form occurs in infants between the eighth and twenty-fourth months of life, is likely to recur and disappears after the second year. (*b*) *Reflex spasms* may be caused by peripheral irritation, as from dentition and worms, though the importance of reflex irritation has been greatly overestimated; gastro-intestinal irritation or auto-intoxication, phimosis and otitis media are possible causes. (*c*) They may be due to *gastro-intestinal intoxication,* which results in debility, malnutrition or a "hydrencephaloid" condition, which has been described above under various captions and is usually of most serious import. (*d*) *Nervous affections,* trauma during delivery, meningitis, tumor, sinus thrombosis, severe coughing, acute encephalitis (cerebral hemiplegia) or acute poliomyelitis are etiological factors. (*e*) They may result from *acute infections,* as scarlatina, measles or pneumonia, in which convulsions and vomiting rather replace the chill which is more common in adults. (*f*) Genuine *epilepsy* may begin in early life; 12 per cent., according to Gowers, and 40 per cent., according to Osler, occur in the first three years of life.

Symptoms.—The symptoms resemble those of epilepsy in most of its phases, but they are, however, less developed in the eclampsia of children. It usually begins in the hands and the seizure is partial, tonic, intermittent and carpopedal rather than generalized. Screaming and grinding of the teeth may occur. The laryngeal spasm has already been described under affections of the vagus.

Treatment.—The occurrence of the *convulsion* is the first indication for treatment and should be met symptomatically by inhalations of chloroform or nitrite of amyl, followed by grs. v to x of chloral and ʒss of bromide by rectum. The bromide should be given for several days, for its effects are cumulative rather than immediate. Opiates should generally be avoided in children under five years, especially in nurslings; if given to them at all the dosage should be as outlined under scarlatina. Opiates should not be used in weak or marantic infants. The hot bath is of little service.

The *cause is then determined* and treated, as (*a*) the rickets, and (*b*) the peripheral irritation; lancing of the gums is practiced less and less; (*c*) in gastro-intestinal intoxication the stomach should be emptied by ipecac or by means of the stomach tube; lavage of the bowel is indicated in acute cases and stimulants may be indicated. (*d*) The bromides should be administered as in epilepsy.

CHOREA AND CHOREIFORM AFFECTIONS.

The name is given to several convulsive maladies. Chorea or St. Vitus dance, known to Paracelsus, was endemic in the middle ages and is now often known as chorea major; it was hysteria or possibly epilepsy. Chorea in its usually accepted sense was described by Sydenham and is known as Sydenham's chorea, chorea minor or infective chorea.

Definition.—Sydenham's chorea is (*a*) a neurosis of late childhood; (*b*) it is characterized by subacute onset, limited duration and, usually, recovery; (*c*) it is probably cortical in localization, as indicated (*d*) by involuntary irregular spasmodic movements, and (*e*) increased incoördination on voluntary efforts; (*f*) it is always associated with some psychical weakness, irritability or alteration in character and (*g*) is frequently associated with rheumatism and (*h*) endocarditis.

Etiology.—(*a*) *Age:* 80 per cent. of cases occur in persons between the fifth and fifteenth years; 90 per cent. in those between the fifth and twentieth years; it is most frequent at the sixth and seventh years of life or at puberty. (*b*) *Sex:* Seventy per cent. of cases are in girls (Gowers, Osler). (*c*) *Rheumatism* has been considered a cause since Bright's time and especially by English writers. The French writers probably overestimate its importance and the Germans apparently underestimate it. Rheumatism is a factor in at least 20 to 25 per cent. (Hughes and Sée); some consider it causal in 45 or even 70 per cent. of cases. *Endocarditis, rheumatism* and *chorea* are too frequently associated to escape serious attention. Rheumatism occurs mostly before the chorea, but in 4 per cent. of cases do the two occur simultaneously. Their association is most frequent in the young, but their exact relation cannot be established until the cause of rheumatism is known. Rheumatism, endocarditis and chorea are probably correlated, *i. e.,* are due to a common virus. (*d*) *Other acute infections* are causes in 43 per cent. of cases (Neumann). Chorea has been observed after gonorrhœa, puerperal fever, sepsis, pertussis, measles, typhoid, etc. (*e*) *Pregnancy* is a predisposing cause in women, particularly before the twenty-fifth year. Chorea occurs in the first three months, especially in first pregnancies, and is most severe in character. It rarely occurs postpartum. Giles de la Tourette justly holds that many cases classified as chorea are really hysteria. (*f*) *Neuropathic heredity* is observed in 16 per cent. of choreic subjects, particularly among Hebrews. (*g*) Other causes are sometimes operative, as fright, trauma and reflex irritation from nasal or digestive disease. It occurs more largely in the lower classes, is sometimes due to iodoform or mercurial poisoning and is influenced by the poor hygiene, excitement, exhibition and competition of the school-room; this is called the "school-made chorea" by Sturges.

Pathology and Pathogenesis.—The disease is a provisional neurosis, since no constant pathological findings exist. The following changes

have been recorded; hyperæmia, exudation about the arteries, areas of softening, punctate hemorrhages, round hyaline bodies in the perivascular lymph sheaths and swelling and turbidity of the large pyramidal cells of the cortex; but none of these are characteristic or constant. In fatal cases endocarditis is the most frequent finding (90 per cent. Osler, 94 per cent. Sturges) and Osler notes that its frequency is greater even than in rheumatism. Kirke's theory of cerebral embolism has been abandoned. The bacteriological findings are negative or conflicting. It is probable that the irritation, whether the result of exhaustion or of toxæmia, is either cortical or in the basal ganglia, which are closely connected with the cortex or subcortex.

Symptoms.—1. Onset.—Prodromes are infrequent, but sometimes there is a subacute onset, with irritability, restlessness, depression, disturbed sleep, headache, anorexia, rheumatic pains or sensory irritation. The affection usually begins with motor disturbance in the form of *involuntary movements* and *incoördinate voluntary movements;* the latter are often misunderstood and punished, for the child cannot sit still or breaks or drops things.

2. Chorea Movements.—These are *involuntary*, although they may seem voluntary, as the thrusting out of the tongue or the reaching of the arm at an object. They are *"irregular in time, character* and *degree,"* are often long, are not usually attended by fatigue or pain, are incomplete and without motive, are increased by attention being drawn to them and by excitement and almost always cease during sleep. They usually begin in the arms and later extend to the face and legs. Chorea very often begins on one side, especially the right, whence it extends to the other side, but sometimes remains unilateral (*hemichorea*); both sides, however, are always involved to some extent. The face is contorted with varying expressions, now grimaces or again motiveless smiles; this is the *facies choreatica* of Sachs. The eyes roll, are disturbed, especially when attempts are made to fix them, and diplopia is not uncommon. The pupils are often wide and react slowly. The author recently saw with Dr. Halbert a case in which many of the teeth were broken. Speech is involved in 25 per cent. of cases; this is due rather to difficult articulation by the tongue and lips than to phonation. Speech is quicker than normal, like the movements of the arm, and may be aggravated by irregular movements of the glottis and respiratory muscles, resulting in panting, irregular inspiration or expiration or sobbing sounds in which the diaphragm participates. Aphasia is less frequent. The *arms are most involved;* they are flexed, extended, pronated and supinated in tireless succession. Bouillaud spoke of the movements as muscular insanity (*folie musculaire*). The *trunk* is sometimes involved in light cases and distinctly so in severe types. The movements are sometimes so violent as to throw the patient out of bed. The *legs* are usually much less involved; there is a zig-zag gait and walking may be impossible.

Besides hemichorea, other irregularities are at times encountered, as monochorea, chorea paraplegica or chorea alternans. The *muscular power* and endurance are often said to be normal. Some writers, as Wallenberg, insist that paresis is no part of chorea, but Gowers and Osler speak of marked muscular weakness. Two forms are distinguished: (*a*) paresis with chorea, in which the muscular power is reduced, as in the usual case, and (*b*) limp or paralytic chorea (Todd, West and the *chorée molle* of French writers). The paresis may be of the mono-, para- or hemiplegic type.

3. Psychical Changes.—These are constant and the undue irritability and ready fatigue resemble an acute cerebral neurasthenia. The subject is wilful, forgetful, emotional, irritable, unable to concentrate the attention or to remember perfectly. Marked alteration may occur, as complications, *viz.,* delirium, mania, melancholia or hallucinations; *chorea insaniens* has a most unfavorable prognosis. Combination with hysteria is frequent, with epilepsy infrequent. Hughlings Jackson found paroxysmal headache in 90 per cent. of cases.

4. Sensation.—Sensation is usually normal. Tenderness over the spine and peripheral nerves is not common, though S. Weir Mitchell and Triboulet described a painful chorea. Occasionally there is a tingling sensation and numbness. Blocq and others have insisted that sensory alteration is referable to hysteria, but Triboulet finds that there is usually some reduction of sensation.

5. The Reflexes.—These are difficult to study and are often said to be normal. They are normal in 50 per cent. and decreased or absent in 50 per cent. Oddo found the tendon reflexes (*a*) rarely increased, (*b*) normal in light cases only and (*c*) as a rule decreased or absent (absent on one side in 80 per cent., on both sides in 50 per cent.). The bladder and rectum are usually normal save in the severest forms.

6. Electrical Reactions.—The electrical reaction and mechanical irritability of the muscles are with few exceptions unaltered.

7. The Heart.—Irregularity is common and is often due to irregularity in breathing (pseudo-arrhythmia). A slight increase in rate is due to the general neurosis. It has been claimed that chorea may affect the "muscles of organic life"; Ollivier, Roger and Simon have written of cardiac chorea, which probably does not exist. *Functional murmurs* are frequent, especially in thin nervous children, from the altered condition of the blood, the toxæmia and increased cardiac rate. They may be confused with incipient endocarditis. Great conservatism must be exercised in their prognosis and diagnosis (see Diagnosis of Mitral Insufficiency).

Endocarditis has been mentioned in the definition of the disease and its causation and pathology (90 to 94 per cent. of fatal cases). It was found in 66 per cent. of Osler's cases without rheumatism. It is rarely malignant, but may produce embolism. Mitral lesions, especially insufficiency, are the most common form. The importance of

latent endocarditis, *i. e.,* without clinical symptoms, has been emphasized by Osler, Oxley and Grosse. Pericarditis is not frequent, though it is found in 20 per cent. of cases, in certain statistics. Heart disease is found in 50 per cent. of cases in which there have been two attacks of chorea, and in 100 per cent. of those having over three.

8. The Skin.—Herpetic eruptions and pigmentation result from medication by arsenic. Urticaria, erythema nodosum and purpura are sometimes observed, as well as the rheumatic nodes first described by Maynet and later by Barlow and Warner.

9. General Symptoms.—Emaciation occurs chiefly in the severer types. The urea and phosphates are often increased. Albuminuria is rare, except as a result of renal embolism. The temperature is not often elevated, save from cardiac and other complications; even in chorea insaniens it rarely passes 102°.

Course and Prognosis.—The average course of mild cases is from six to ten weeks, and six weeks to six months is the average duration of all varieties. In very rare instances the disease may last six (Gowers) or even sixty years (Meldner). Three main types are distinguished: (*a*) The *mild type,* in which the patient is nervous, irritable, has but slight choreic movements and is often dyspeptic and sometimes anæmic; (*b*) the *severe form* in which the spasmodic movements are severe and general; (*c*) the *maniacal type* (*chorea insaniens*), which is most often observed in adult or pregnant women, and sometimes at puberty. The movements are incessant and extreme and are often associated with fever, constant insomnia and great psychical disturbance. As a rule, the shorter the duration the more intense are the symptoms. The tendency to *recurrence* was noted by Sydenham. Relapses occur in 25 to 33 per cent. of cases. One year is the average interval and when there is no recurrence within one and one-half years it is probable that none will be observed (Gowers). Germain Sée held that relapses were most likely to occur at the sixty-ninth day. *Recovery* is the rule; the mortality averages between 2 and 3 per cent. Twenty to 25 per cent. of pregnant cases die. Fatality is greatest at the age of puberty and in adults. As Charcot remarked, death occurs less often from complications, as endocarditis, than from toxæmia, fever, mental changes, emaciation, delirium and coma. In all cases rise of temperature or obstinate insomnia is ominous. Though the immediate outlook is not influenced by endocarditis, it is a most important feature in the ultimate prognosis of a complete recovery. *Intercurrent fevers* may dissipate the spasms. While complete recovery is usual, in rare instances distinct psychical changes (Jolly) or permanent chorea remain (*v. s.*).

Diagnosis.—Recognition of the disease is usually most easy when one bears in mind the conception (definition, *q. v.*) of the disease.

Differentiation.—Simple tremor or the intention tremor of multiple sclerosis is easily separable. The hemichorea of hemiplegia (see Diagnosis of Cerebral Hemorrhage) or the bilateral chorea

of *infantile cerebral paralysis* (*q. v.*) is distinguished by the history and course. Friedreich's ataxia is differentiated by its slow, irregular, incoördinate movements, nystagmus, its frequent family incidence, the scoliosis, talipes and slow, scanning speech. *Hysterical chorea* (so-called) is rhythmic, more regular and frequently produces sudden ("electrical") movements, as bowing or salaaming, nodding the head, etc. *Chorea major* is easily distinguished. Chorea in the original sense is chiefly of historical interest; it was a pandemic or epidemic *hysteria* provoked by religious excitement. Its victims made pilgrimages to the shrine of St. Vitus, whence the name of the disease. Similar outbreaks occurred in Kentucky among the early settlers. This form closely resembles the saltatoric spasm described by Bamberger in 1859, in which the legs contract on attempting to stand, causing jumping or springing movements. It may be transitory or may last for years and occurs chiefly in neurotics. Beard and Thornton described a similar neurosis endemic in Canada among the "jumping Frenchmen." The "holy rollers," and jumpers of Russia and Java probably come under this head or under impulsive tic (*v. i.*).

Huntingdon's chorea has no connection with Sydenham's type. Chronic chorea comprises two forms: (*a*) The *senile form,* in which the mind is less involved, the course more benign than in (*b*) the *hereditary form* of Huntingdon. Although it was described by Walters (1842) and Lyons (1863), Huntingdon in 1872 brought out the cardinal features of the disease as observed on Long Island. These are (i) the late and gradual onset after the thirtieth year, more marked in males than in females; (ii) its hereditary character, sometimes involving 25 or 50 per cent. of the family, sometimes skipping generations or lasting for generations or even for two centuries in a family (Osborn); (iii) its progressive character, and (iv) the resulting mental degeneration, ending in slow dementia, often with epileptiform attacks or suicidal tendencies. The precise *pathology* of Huntingdon's type is not known. Pachymeningitis, hæmatoma of the dura, or less often chronic encephalomeningitis, vascular disease and atrophy of the convolutions have been present in the isolated autopsies made. The *etiology* is unknown. *Clinically* it is characterized by irregular movements, which usually begin in the hands, are somewhat controlled by voluntary effort and are more irregular, incoordinate and slow than in acute chorea. Slow spasmodic facial contortions are observed, and the speech becomes first slow and hesitating, and later quite indistinct. The gait is unsteady and swaying. Writing is interfered with, the reflexes are moderately increased, sensation is normal save that the muscular sense is somewhat blunted, and dementia develops. The disease is incurable, although somewhat benefited by opium and bromides.

Tic.—The term is now used to designate an "habitual, conscious, convulsive movement resulting in the involuntary contraction of one or more muscles, abruptly reproducing some reflex or automatic action of every-day life" (G. Guinon). There are three forms.

(*a*) In *simple tic,* (i) localized spasm, *habit spasm* or habit chorea, which is said by S. Weir Mitchell to occur in girls between seven and fourteen years of age, may result from lesions of the nose or throat or possibly from eye-strain, and includes movements such as winking, drawing of the mouth, sniffing, shaking the head and shrugging the shoulders. It usually ceases, but may last into adult life, and the "child's attention should not be drawn to the movements," although the writer believes that judicious reminders and discipline inhibit these habits. Arsenic may be beneficial. (ii) The *electric chorea* (Henoch and Bergeron) consists of short, lightning-like contractions in special muscles, as those of the neck or shoulders, as though the muscles were electrically stimulated. (iii) *Dubini's disease* (1846) consists of contractions of the arms and legs, followed by hyperæsthesia, pain in the head and back, muscular atrophy, paresis and sometimes fever and convulsions. It is endemic in Lombardy, its cause is unknown, and it is usually fatal from apoplexy or coma. (iv) *Paramyoclonus multiplex*, or *myoclonia,* described by Friedreich in 1881, is an affection of sudden onset, with symmetrical, involuntary, arrhythmic, quick, clonic contractions of the upper parts of the arms, legs and trunk, sometimes of the face and tongue, and occurs without loss of consciousness. The contractions number from 10 to 150 a minute and may terminate in a tetanic contraction. It affects male adults chiefly (75 per cent.) and follows mental or emotional agitation, so that some would classify it under hysteria. The myotatic irritability and reflexes are increased, the mind is usually normal, although sometimes fixed ideas are observed, hiccough is occasional from involvement of the glottis or diaphragm, voluntary movement is decreased or sometimes arrested, there are no sensory symptoms and the outlook is usually good, although the course may be long and relapsing. Treatment consists of administration of voltaic electricity, nervines and bromides. The family form, described by Unverricht, with epilepsy and spasms of the tongue and deglutition, is probably a subtype of hereditary chorea. C. Oddo gives the following general differentiation between

Tic	and	Chorea.
Form of movement: pseudo-intentional, with repetition.		Incoördination and great variability.
Rhythm: rhythmic, brusque, short, synchronistic.		Arrhythmic, slow, not synchronistic.
Will: suspends movements.		No action.
Accessory signs: muscular power preserved; normal tendon reflexes and sensation.		Myasthenia; modified reflexes and sensation.
Etiology: hereditary degeneration.		Neuropathic tendency.

(*b*) *Tic with explosive utterances,* impulsive tic or Gilles de la

Tourette's disease, is closely affiliated with hysteria and monomania. It occurs in neurotic individuals between the sixth year and puberty. The *cardinal features* of the psychosis are: (i) Involuntary movements in the face and arms or in all the muscles in severe types, when the movements become violent or explosive. (ii) Explosive utterances, resembling a bark, occur; the patient repeats words he hears (echolalia) or mimics movements (echopraxia); the child may use obscene or profane words (coprolalia), which was considered by de la Tourette to be most characteristic. (iii) The subject has fixed ideas. These include: arithmomania, in which every action is preceded by counting or doing a certain thing a given number of times; a fear of contamination (*délere du toucher*); *onomatomania,* or repetition of a given word or name; *folie pourquoi,* demanding the reason for everything. The prognosis is usually unfavorable.

(*c*) In *complex coördinate tic,* as thumb sucking, rocking in the bed and bumping the head, the outlook is favorable when it does not occur in feeble-minded children (Gee and Hadden).

Treatment of Acute Chorea.—1. Prophylaxis.—This may produce results; sensible methods of education should be adopted, the hours in the overheated, foul school-room should be short, and the useless forcing of children by marks, prizes and exhibition should be abandoned. Children can be taught more in an hour or two of judicious individual instruction than in the five or six confining hours which their wandering attention must endure. Prevention may concern other etiological factors (*q. v.*); for instance, the nose should be examined for sources of irritation, as adenoids.

2. Rest in Bed.—The results obtained by *rest in bed and isolation* are as rapid as those of any medicinal therapy. It not only saves tissue waste, but is the best prophylactic and symptomatic treatment of possible or actually established endocarditis.

3. Skin.—The skin is very often dry and harsh, whence it must be given special attention. Diaphoretics frequently produce beneficial effects. The stimulation of the skin by proper covering and hydrotherapy is the simplest and least injurious method. The refrigerant diaphoretics and full warm baths give the best results. The skin must be kept clean and in severe cases the water-bed must be used.

Trauma.—Trauma is prevented in severe types by padding the beds by mattresses and pillows, since the violent movements may even throw the patient to the floor. Sepsis is a frequent occurrence, and is most dangerous. The temperature must be taken by rectum.

5. Exhibition of Drugs.—(*a*) *Fowler's solution* is the most frequently used and the oldest remedy, and apparently gives the best results. It should be given in increasing doses (see Treatment of Pernicious Anæmia for detailed action); children tolerate large doses of arsenic exceptionally well. It may be administered hypodermatically or by clyster when the digestive tract is irritable. Arsenic is not without its dangers. C. Gamble, Jr., records an instance of fatal arsenical poisoning in a case to whom about 1½ ounces of

Fowler's solution was given in a month (including an intermission of one week). It appears that rest in bed and administration of a placebo, as nux vomica, has given equally good results. (*b*) *Salicylates* are of benefit only when rheumatism actually co-exists and acetanilide is indicated only in the initial stages. In other words, antirheumatic remedies are less important in the treatment than is rheumatism in the etiology. (*c*) *Depressomotors:* The *bromides* rarely cure the movements, although they often quiet the mild psychical manifestations. Chloral has been given in large and continued doses to produce sleep or continuous sleep. This method merely demonstrates the tolerance of the body for dangerous remedies and chloral cannot be given safely in amounts exceeding a dram for adults in three divided doses at intervals of an hour. Morphine is generally dangerous and inferior to chloral. Small doses often accentuate the mental agitation, but if combined with chloral, hyoscine and bromide, it may give at least temporary relief. (*d*) *Strychnine* is valuable (Trousseau), especially in the later stage, when recovery is slow. (*e*) In *chorea gravidarum,* abortion or premature delivery is indicated.

TETANY.

Definition.—Tetany, first described by Steinheim (1830) and Dance (1831) and named by Corvisart, consists chiefly of (*a*) peculiar tonic, intermittent, bilateral, painful spasms, mostly in the extremities, without involvement of the sensorium and (*b*) increased mechanical and electrical excitability of the nerves.

Etiology.—(*a*) It occurs *endemically* or epidemically, especially in Vienna, Heidelberg, Berlin and Syria, and it is prevalent during March and April. It is rare in America, England and Italy. Though once frequent (1830–1860), it is now uncommon in Paris. In the Vienna outbreaks 46 per cent. of cases occurred in shoemakers and 24 per cent. in tailors who were otherwise healthy. Tetany occurs among the poorer classes. Eighty-three per cent. occur in persons between sixteen and twenty-five years of age. It may develop in an entire family. (*d*) *Digestive affections* may produce it, as dilatation of the stomach (Kussmaul, 1871), dyspepsia, ulcer or cancer of the stomach or duodenum, often with pyloric obstruction; auto-intoxication is the most probable cause of this group. Only 80 cases of gastric tetany are on record. (*c*) *Rickets* is causal in 85 to 90 per cent. (Seeligmüller) and in 96 per cent. (Cassel) of the infantile types. Some writers—incorrectly, we think—consider that the infantile carpopedal spasm is tetany. (*d*) *Tetania strumipriva* is a type of tetany which follows removal of the thyroid in 15 per cent. of cases (Albert, Mikulicz, Kocher). It appears that the thyroid is less important than the parathyroid glands. (See pages 856 and 857.) (*e*) Tetany occurs also in the *acute infections,* sepsis, nephritis, puerperal or nursing women and rarely in intoxications with ergot, morphine or lead. There are recorded only 32 cases of the

puerperal form. Of the four cases which the author has seen in this country two occurred with florid secondary syphilis, one after an adenoid operation and one with gastrectasis. It may develop with other nervous disease, and French writers have attempted to class it under hysteria. No pathological changes have been observed in the brain or nerves; in a few cases there has been cloudiness with swelling of the anterior horns. Fifteen cases are reported in America (Howard, 1906).

Symptoms.—1. Muscular Contracture.—The earliest and chief symptom is the *muscular contracture,* which usually commences in the hand, with stiffness, numbness and pain, and culminates as a tonic, symmetrical, painful contracture. The muscles of the hand are hard; the attitude is that of the accoucheur's hand, the hand in writing or that observed in paralysis agitans; it is produced by contraction of the ulnar flexors; less often it resembles the clenched hand of old hemiplegic contracture, and the nails even enter the palm; rarely the fingers are tonically spread apart. Except in light forms, the spasm cannot be overcome by force. The wrist may be flexed. The foot is affected less often than the hand and the spasm draws the foot into the equino-varus position, with the toes flexed and the sole hollow. The spasm is usually limited to these parts, causes pain, lasts from minutes to hours or even days, disappears and usually recurs later. In rarer and more severe cases the parts above the elbows and knees may participate, with adduction of the arms and thighs, cramping of the face, neck, chest, tongue, diaphragm and ocular muscles, with diplopia, spasticity of the sphincters with retention, dysuria, etc.

Laryngismus stridulus (Clark, 1815) is common in infantile rickets, in which clonic contractures also occur. The muscles are somewhat weak and tremor is frequent. Pineles describes laryngospasm following parathyroid extirpation.

2. Trousseau's Sign.—This consists in producing the spasm by pressure over the nerves, for example, just above the elbow or over the peroneal nerve, for a few seconds or from one to five minutes. It is wholly due to nerve pressure, not to pressure on the vessels, and *is pathognomonic.*

3. Erb's Sign.—There is *increased electrical irritability* of the motor nerves to the galvanic current with early anodal closing and cathodal opening tetanus. Anodal opening tetanus is found in this disease alone. There is also increased electrical irritability of the sensory nerves (Hoffmann) and of the nerves of special sense (Chvostek, Jr.), to faradic and galvanic currents.

4. Chvostek's Sign.—*Increased mechanical irritability* of the motor nerves (*Chvostek's sign*) is observed on tapping or pressing on the facial, median, ulnar or other nerves, which produces contraction of the muscles supplied by them. It is quite constant but may be absent in children and is not pathognomonic, having been found in normal persons, in cases of bulbar paralysis, in the major neuroses

and in convalescing cases of facial paralysis. (Some of these may have been atypical tetany). The muscles themselves are not over-excitable.

5. Other Symptoms.—The sensorium is usually clear, except in rare cases with pyloric stenosis. The special senses are normal; optic neuritis is extremely rare. Headache and malaise are frequent. In contrast to the pain and paræsthesia, objective sensory findings are absent. In rare cases epileptiform seizures are seen, which are probably due to parathyroid changes. The reflexes are usually normal. The temperature is elevated in 15 per cent. of cases, but may be subnormal. In very rare chronic cases, trophic, vasomotor and secretory disturbances may occur, as œdema, polyuria, glycosuria or falling out of the hair.

Diagnosis.—The diagnosis in typical cases is most easy from the cardinal signs: (*a*) the peculiar spasms; (*b*) Trousseau's, (*c*) Erb's and (*d*) Chvostek's signs. In epilepsy these signs are absent and the aura, the cry, falling, tonic and clonic convulsions, biting of tongue and involuntary urination are characteristic. Hysteria mimics everything, but the cardinal symptoms, except the spasms, cannot be reproduced; it has its own stigmata; certain reported epidemics of tetany, as at Gentilly, were clearly hysterical. In adults atypical forms without spasm are recognized by the other cardinal signs, which easily separate tetany from the occupation neuroses and acroparæsthesia (*v. i.*). In children meningitis and brain tumor are distinguished on careful examination. The rhachitic relations are important even though the extreme statement that all tetany in children is rhachitis, cannot be admitted.

Prognosis.—The outlook is good in most cases and the course covers from two to four weeks as a rule. Recurrences are not infrequent. The etiological prognosis naturally varies, but generally speaking, death occurs not from tetany but from dilated stomach or ablation of the parathyroids. Cases may die from laryngospasm. In a recent review of his cases, Fränkl-Hochwart concluded that the prognosis is worse than is usually stated; 20 per cent. of his cases died, 20 remained well and the balance had recurrences and serious disabilities.

Treatment.—The treatment varies with the cause. (*a*) *Rest and quiet* are indicated on general principles, although cases of tetany are influenced very little by excitement. Massage, strychnia and electricity should of course be avoided. Change of occupation and location, at least temporarily, is advisable in the endemic form. (*b*) In the *digestive form,* washing out the stomach and bowels in suspected cases of auto-intoxication may induce severe spasms. These cases are both dangerous and obstinate. The spontaneous mortality in gastric tetany is 75 per cent.; with operation for pyloric stenosis, this figure is reduced to 37.5 per cent. (*c*) In the *rhachitic form,* dietary regulation and improved hygiene are necessary, and phosphorus and iron are indicated. Inhalations of chloroform in some cases irritate the sensitive larynx. (*d*) In so-called *tetania strumi-*

priva the mortality is 80 per cent.; parathyroid feeding is helpful. (*e*) Cases following *acute infections* usually have a spontaneously short course; those occurring in pregnancy rarely indicate abortion; in nursing women, weaning gives prompt relief; for intoxications there are obvious indications. In general, drugs are of little value; chloral is indicated *a priori,* to influence the motor nerves, but is inferior to the bromides, as is also morphine, except for pain. Sweating and digitalis in nocturnal forms of tetany have apparently helped some cases, but the disease usually runs its limited uninfluenced course.

OCCUPATION OR FATIGUE NEUROSES; WRITER'S CRAMP.

The continuous, hard, improper use of coördinate, functionally associated muscles may result in their cramping. *Writer's cramp* (graphospasmus, mogigraphia) was first described by Charles Bell (1830) and especially studied by Duchenne and Benedict. Neuropathic tendencies are noted in most cases; 83 per cent. occur in males, 93 per cent. occur in those between twenty and sixty years of age, and most of these cases develop between twenty-five and thirty-five. Worry is a frequent factor; toxæmia, cold, trauma, neuritis and neuralgia are possibly factors. The disease practically never occurs in those who write properly, *i. e.,* move the arm from the elbow or the shoulder, but develops in those who use the little finger or wrist as the basis of movement. The affection is located in various centres; some hold that it is in the coördinating cortical or subcortical centres; it is clearly not in the muscles or nerves, for their other movements are often normal; the occasional atrophy and paresis may suggest localization in the cord.

Symptoms.—The symptoms come on gradually, with fatigue, pain and cramping in the muscles of the forefingers and thumb, which may extend to the forearm or shoulder. There may be the "lock spasm" of Mitchell, in which the pen is spastically held or it may be thrown from the fingers. On rest, the tonic spasm disappears, but reappears with attempts to write. In Benedict's classification we find the spasmodic, paralytic, tremulous and neuralgic forms, which are of less interest as types than as showing the various symptoms, which are often combined in a single case. Vasomotor symptoms may occur, as flushing or glossy skin.

Differentiation.—Differentiation, according to the type, from hemiplegic weakness, paralysis agitans, musculospiral paralysis and neuralgia, is usually easy. *Similar cramps* are observed in telegraphists, milkmaids, weavers, cigarette rollers, tailors, shoemakers, watchmakers and violin and piano players. Public speakers may suffer from similar neuroses of the throat and Church mentions jaw spasms in gum chewers and of the lumbar muscles in physicians who drive much.

The *outlook* is poor, because the unfortunate sufferer is often com-

pelled to write until the affection is firmly fixed and when able, usually returns to the same work. In some cases the muscles may waste.

Treatment.—Rest is essential to recovery, because treatment for the usual nervous symptoms alone never succeeds. Plaster casts are sometimes successfully employed. Even when the subject learns to write with the other hand it often develops there. Prevention is possible by writing free-hand from the shoulder; holding the pen between the first and second fingers, use of pens with large handles, of quills and of forearm splints with the pen attached to the extremity, are also helpful. Vogouroux and Wolff use with success systematic gymnastics, including passive manipulations and active exercises.

MYOTONIA. THOMSEN'S DISEASE.

Myotonia is usually called myotonia congenita from its leading causal element, heredity. It was first described by Leyden, but more fully by Thomsen (1876), who was a victim among 20 cases in his own family, in five generations. This rare affection, of which there are about 100 reported cases, occurs chiefly in males and in Germany and Scandinavia. Its pathology is obscure, for the only necropsy which has been performed (Déjérine and Sottas) showed no nervous alteration. Muscle fibers, excised during life, show hypertrophy, decreased striation, nuclear multiplication and slight interstitial increase, leading Leyden and others after him to consider the process myopathic. Some offer the objection that these changes are artefacts.

Symptoms.—A peculiar *transient tonic muscular rigidity* develops when the muscles are first used, which limits or arrests movement, but wears off as their use is persisted in. It is intense when a new kind of movement is undertaken. The longer the muscles are rested the more severe is the initial spasm on exertion. Rigidity does not follow passive muscle movement, although firm pressure or a sharp blow initiates it. It appears early in life, perhaps "in the cradle," but generally between the ages of four and ten, it increases as the muscles develop and then becomes stationary for life. In one case improvement was observed. The legs are more affected than the arms, but in exceptional cases the face, tongue, bladder or the respiratory muscles may be affected. They are usually stronger and sometimes larger than normal, although some writers speak of muscular weakness.

Though the tendon reflexes and the mechanical and electrical excitability of the nerves are practically normal, application of the galvanic current to the muscles produces sluggish, prolonged closing contractures, which are easily and equally obtained with either pole and become tetanic with strong currents; this is the *myotonic reaction of Erb*. Undulatory contractures follow strong faradic stimulation. Subjects of myotonia are frequently irritable or hypochondriacal.

Treatment.—Treatment is of little value. Thomsen believed that exercise retarded the disease. Cold, dampness, excitement and fatigue are to be avoided.

The following are allied, if not identical, affections; the congenital *paramyotonia* of Eulenberg, consisting of tonic spasms, which last from fifteen minutes to several hours, occur especially in the arms, and often affect the eyes and mouth; it is excited by exposure to cold (vasomotor spasm in the muscles); and the *ataxic paramyotonia* of Gowers, in which tonic spasms may last the entire day, with muscular weakness, ataxia, especially in the hands and disturbed sensation or anæsthesia in the fingers.

PARALYSIS AGITANS.

This affection is also known as shaking palsy or Parkinson's disease; it was first described by Parkinson in 1817.

Etiology.—(*a*) Most cases occur in persons between the ages of *forty and sixty-five.* (*b*) Sixty-six per cent. of cases are *men.* (*c*) A *neuropathic tendency* is present in 15 per cent. (*d*) It is doubtful if trauma, acute infections or thyroid disease are causes.

Pathology.—It is classed among the neuroses, because of the absence of any constant pathological finding. It is not strange that arteriosclerosis and perivascular thickening are often found, since paralysis agitans occurs in "the early degenerative period." Gordinier's collection of cases examined by recent histological methods, shows that (*a*) general arteriosclerosis is less marked than that of the nervous tissue; (*b*) the arteries in the cord are markedly changed and around them are (*c*) increased neurogliar sclerosis and (*d*) pigmentation and atrophy of the nerve cells and fibers.

Symptoms.—The cardinal symptoms are (*a*) tremor; and (*b*) stiffness, slowness of movement, shortening and weakening of the muscles. (*a*) *Tremor* in 66 per cent. of cases is the first symptom; it may develop after the muscular rigidity and is sometimes absent (paralysis agitans sine agitatione). It usually appears first and is most marked in the *hands.* It affects first one hand and usually next reaches the leg of the same side, then the opposite hand, the opposite leg, and lastly the trunk, so that at different periods of development it is successively a monoplegic, hemiplegic or finally a diplegic tremor. It is slow (4 to 7 oscillations per second), rhythmic, fine at the onset, but somewhat coarser later and in the large muscles is more marked in peripheral than distal parts, *persists during rest, ceases during sleep* and ceases momentarily on voluntary action. In rare cases it appears only on movement, as intention tremor; Gerhardt found it increased by movement in 50 per cent. It is difficult to define precisely the muscles involved. In the hands it may affect chiefly the flexors and extensors at the metacarpal joints or the interossei, which give the *spinning or pill-rolling movement.* The wrists may be abducted and adducted, rarely supinated and pronated, and the arms and shoulders are implicated but little. The writing is tremulous. The feet are mostly involved at the ankles and in the toes but little. The thigh adductors often tremble. Trembling is infrequent in the face, neck and back, while the abdomen entirely escapes. The head

and body may of themselves tremble, but much of it is imparted to them from the limbs. In the rarest cases the tongue, palate, larynx and epiglottis tremble.

(*b*) *Muscular rigidity* and *shortening,* weakness and *slowness of movement* usually follow the tremor; slight symptoms are detected early and anticipate tremor or exist without it. These changes result in the following: (i) The *Parkinson facies,* which is *mask-like* and devoid of lines or expression; the eyelids seldom wink. The forehead shows transverse folds because of the bowed head and body. The eyes move tardily in reading, and the entire body must be turned to enable the patient to look laterally. There is also slow, monotonous, *monosyllabic speech,* with hesitation at first, but hurried at the finish. (ii) The *attitude* is characteristic, because of the flexor positions assumed, the head and the chin are held forward, the neck is extended and rigid, the body is inclined forward, the elbows are fixed and brought somewhat forward and away from the body; the forearms are brought toward the body, the trembling thumb and index finger are apposed, or the fingers deviate toward the ulnar side as in arthritis deformans, and in standing the knees are somewhat bent and at times closely apposed. The body expression, we may say, is like that of the face. (iii) The *gait* is equally characteristic. Like the speech, it is initially hesitant, and later hurried. The patient, when once started, may not be able to stop himself. Trousseau spoke of the patients as "chasing their centre of gravity." This *festination* or *propulsion* is probably purely mechanical from the forward bodily inclination, although interpreted by Charcot as a kind of forced movement. To counteract the throwing forward of the centre of gravity the arms may be carried behind the back. In some cases, if the patient is pushed backward or sideways, he may not be able to stop for some steps; this is *retro- and latero-pulsion.* (iv) The *palsy* is often slight and rarely extreme, although it may be simulated by the helplessness

FIG. 79.

Side view of a case of paralysis agitans, showing forward inclination of the trunk. Tendency to propulsion. (Dercum.)

caused by rigidity. The patient is not inclined to exertion, "moves like a piece of machinery," as Parkinson describes it, and may not be able to turn over in bed. Patients have drowned from falling on the face in a small puddle of water. Muscular reaction requires 40 per cent. more time than normal.

Other symptoms are subordinate. The mind is clear, but we may say *stiff* like the face and limbs, and a certain restlessness, irritability or apathy may be detected; insomnia is common and headache occasional. Sensation is essentially normal, although pains, pruritus, paræsthesia and hypæsthesia are seen in 35 per cent. of cases. Vasomotor disturbance most often assumes the form of a feeling of heat; indeed the peripheral temperature may be increased several degrees. Severe sweats are quite common. The pupillary, cutaneous, tendon and organic reflexes are normal.

Course and Prognosis.—The onset is usually gradual and the course covers several decades. The outlook is most favorable in atypical cases. Remissions may occur, but the disease progresses steadily. Death very rarely occurs from the affection itself, but usually from intercurrent pneumonia or advancing arteriosclerosis, cerebral softening, myocardial insufficiency, nephritis and kindred lesions.

Diagnosis.—When there is clear development of the cardinal symptoms the disease is unmistakable. Confusion is possible in the early hemiplegic forms, but the absence of a paralytic stroke and the later course are distinctive. Charcot and Ordenstein (1868) clearly differentiated the affection from *multiple sclerosis* (*q. v.*). The fine, *senile tremor,* the *hereditary tremor* of Dana, tremors due to overexertion or excitement, to toxic causes (alcohol, nicotine, lead, mercury, coffee, opium or chloral), weakness of convalescence from acute infections, paretic dementia or the neuroses (epilepsy, Graves' disease, hysteria, neurasthenia) are easily excluded by the absence of the Parkinson rigidity, facies and festination.

Treatment.—Treatment of the probably fundamental arteriosclerosis may possibly retard the disease, but prophylaxis is possible in the early stages only. All mental and physical fatigue should be avoided. The administration of full doses of iodide, chloral, opiates, bromides, hyoscine, ergotin, conium or duboisin may mitigate the tremor, but always lower vitality and disturb function.

PERIODIC FAMILY PARALYSIS.

This affection was first described by Cavaré (1853) and more fully by Westphal (1885); Holzapple collected 70 cases (1905). It is a family affection; Goldflam collected 11 cases in one family and E. W. Taylor 12 in another. Most cases occur in adolescence and nearly all of them in persons under twenty-five; it recurs every few weeks or months, possibly every day, or only at intervals of years. Its *symptoms* are almost wholly motor, producing a *flaccid paralysis* in the legs and arms, less often in the trunk, and exceptionally in the face,

neck, tongue and throat. It occurs without apparent cause, usually during sleep, from which the patient awakes with a sense of fatigue, weakness or dulness which develops into paralysis within twenty-four hours. Mental and sensory symptoms and those of the cranial nerves and special senses are rare. There is no fever, the pulse is often slow and cardiac dilatation has been observed. The deep reflexes are decreased or abolished, often also those of the skin, and the faradic excitability of the nerves and muscles is lessened or absent. The patient is well in a few hours or days, until another attack occurs. The disease usually subsides spontaneously after the fiftieth year of life. Its *nature* is not determined. Goldflam's theory of auto-intoxication is supported (*a*) by J. K. Mitchell, Flexner and Ebersoll, who found increased secretion of kreatin before and during the attack; (*b*) by Crafts and Irwin, who isolated toxins from the fæces; (*c*) possibly by the fact that potas. citrate sometimes lessens or aborts the seizure. Dana thought that the disease was hysterical, but Bernhardt and others consider it affiliated with myotonia and progressive muscular atrophy. Holzapple administers potas. bromide in doses of thirty grains.

MIGRAINE.

Migraine (hemicrania, megrim, sick headache) is an hereditary degeneration or psychoneurosis, with the paroxysmal occurrence (in a complete attack) of auræ, headache, nausea and vomiting.

Etiology.—(*a*) *Heredity* is a conspicuous cause (90 per cent. Möbius), either directly as migraine or indirectly from neurotic, insane or epileptic antecedents. Joint affections, as gout, are often noted in the family history. (*b*) Thirty-three per cent. of cases begin between the fifth and tenth, 40 per cent. between the tenth and twentieth years, and the remainder usually before the thirtieth year. Females are affected a little more frequently than males. (*c*) *Exciting causes* are acute infections, constipation, dyspepsia, overexertion, alcohol or sexual abuse, emotional factors, pelvic disorders and lactation. It is attributed, though probably incorrectly, to adenoids. Refractive errors may explain some cases.

Symptoms.—Preliminary symptoms are often absent, although the patient may feel depressed, or, in some cases, especially well and energetic. They occur in cases in which the sensory symptoms are least marked (see below). Sometimes the pupils are dilated. Most attacks develop during the night.

Auræ.—In over 50 per cent. of cases, auræ are (*a*) sensory, as visual auræ, a cloudiness before the eyes, sparks, lightning, zig-zag forms, etc.; they are most often bilateral and affect the homologous half-fields; taste and smell may be similarly affected; paræsthesia may occur, usually as hemiparæsthesia; (*b*) motor auræ are much less common, as aphasia, dysphasia or motor weakness on the contralateral side; in the very rare form, ophthalmoplegic migraine, there are fugitive ptosis, outward strabismus, double vision and a dilated im-

mobile pupil; mental auræ are uncommon and not usually pronounced, as restlessness, anxiety, confusion and double consciousness. The auræ last for ten or fifteen minutes.

The Attack.—(*a*) *Headache* is the cardinal symptom. At first it is usually intense and circumscribed, as over the eye, temple, occiput or forehead; then it spreads over one side (*hemicrania*), sometimes affects the opposite side or rarely reaches the neck or arm. The pain is deep, varies in character, being most often boring, and its intensity may attain that of the pain in brain tumor or meningitis. It is increased by movement, stooping or lying down. The special senses are hyperæsthetic but the nerve trunks are seldom tender. The mind is usually clear and often acutely active, sometimes there is double consciousness, and in rare cases stupor. (*b*) *Nausea* follows, often with vomiting of undigested food and finally of bile; this is the so-called "bilious headache." Feeding and medication are quite impossible in severe cases. Vomiting may be attended by hyperacidity or moderate collapse, as in sea-sickness. (*c*) *Vasomotor symptoms* may be present. The *spastic type* was described by Du Bois Raymond in his own case; with pallor, coldness, wide pupils and retraction of the eyeballs. Thoma found the temporal artery sclerotic on the side of lesion and de Giovanni noted a marked arcus senilis on the same side. The pulse may be slow and tense and the breathing superficial. In the *paretic type* observed by Möllendorf in himself, there are redness of the skin, small pupils, sweating and increased flow of saliva or tears. Very rarely is there erythromelalgia, œdema or ecchymosis. The paretic may follow the spastic symptoms. It is best to regard these sympathetic symptoms rather as a result than as a cause of the attack.

The *attack ends* in various ways. Relief may come with the vomiting, after which the patient frequently sleeps. Sometimes the ending is almost critical, with sweating, lachrymation, polyuria or epistaxis. The attack lasts from ten to twelve hours; the term *status hemicranicus* is used for cases of long duration or those in which the attacks overlap. Though the typical seizure comprises the aura, lasting fifteen minutes, and the headache, lasting ten or twelve hours with nausea and vomiting, *most attacks are atypical.* There is most often headache alone or headache with vomiting; in a few cases the attack aborts with the aura or vomiting exists alone. The auræ, pain, gastric and cardiac inhibition, vasomotor symptoms and vomiting prove that migraine is a *functional cerebral affection.*

Course and Prognosis.—When the disease has once been established in youth its attacks become more frequent in adolescence and usually last until the forty-fifth or fiftieth year. The periodicity, which was noted in early cases described by Tissot, Trousseau and Liveing, has not been explained. Overwork, worry, menstruation or change of routine may precipitate an attack at a regular or irregular interval. Attacks may cease during an intercurrent pregnancy, tabes or gout. The school of Charcot has emphasized the fortunately rare danger of transformation into epilepsy, labyrinthine vertigo or some psychosis.

Complete cases are more obstinate and dangerous than light forms.

Diagnosis.—When there is a history of heredity and development in childhood, the full attack is most characteristic. "Sick headache" is practically always migraine. In atypical forms there may be diagnostic confusion, chiefly with various forms of *headache,* which may be classified as follows:

1. Those which result *from nervous affections,* as (*a*) cerebral neurasthenia, in which there is a sense of pressure rather than actual headache, and which is often constant, without nausea, but with paræsthesia, and in location is frontal or usually general, but sometimes occipital or temporal; (*b*) hysteria, which is less common (clavus headache, "neuralgic headache"), and sometimes is precisely like migraine (Charcot); (*c*) epilepsy, which some consider as identical with migraine; in epilepsy the aura is shorter and loss of consciousness is the most constant symptom; (*d*) meningitis, which in all types and in all cases is early and severe; (*e*) hydrocephalus, which is often severe, especially in acquired forms; (*f*) brain tumor, which is constant, generalized, often very severe and sometimes typically hemicranic; (*g*) paretic dementia, in which migraine is frequent; care is always necessary when migraine occurs after syphilitic infection.

2. Those which result from affections of the *special senses,* as eye strain, hypermetropia; glaucoma, which most often produces frontal headache; nasal disease, which may cause sinus involvement with frontal headache; this form is not periodic and is often relieved by iodide, which has no effect in migraine.

3. Those which result *from toxæmia.* (*a*) Those due to acute infections are usually frontal and dull, but sometimes occur in other locations and are severe, deep-seated and increased by stooping; in typhoid or malaria they are often frontal or supra-orbital; (*b*) intoxication by alcohol, nicotine, lead, nitroglycerine and opium, and (*c*) constitutional states, as gout or arthritis. (*d*) In nephritis they are usually frontal, remittent or continuous, but sometimes sharply hemicranic. (*e*) In gastric affections they are exceedingly common, mostly occipital, but sometimes vertical or frontal.

4. Those resulting *from circulatory causes,* as from wearing a tight collar, repeated coughing, cardiac disease or arteriosclerosis (with vertigo), are usually increased by lying down.

5. Those which result *from anæmia,* especially from chlorosis, as a "tugging" sensation back of the eyes or frontal or generalized headache, are often relieved by lying down.

Treatment.—*Prophylaxis* is said to be of little value, but many subjects learn to recognize the warnings and avert the attacks by resting on noting unusual mental or physical capacity for work or an unusual appetite, which ushers in some attacks. Migraine subjects, as other neurotics, should observe *moderation* in eating, especially of meat, in work, exercise or recreation, narcotics or stimulants, as coffee or tobacco, and in sexual matters. Immoderation, indeed, is

almost a neurosis in itself. Overwork, hurry, worry and tension must be avoided and their first manifestations must be learned and treated. The extract of *cannabis indica* gr. ¼ to ½, given for weeks or months, after Seguin's plan, is almost the only remedy which helps the condition, and then only in some cases; the author knows of a dozen cases in which migraine has been benefited or wholly relieved under its use. Möbius finds that *salicylate of soda,* gr. xv, in black coffee, given the evening before the attack, often wards it off. Nitroglycerine in full doses sometimes aborts it (Gowers), as may washing out the stomach and following the lavage with a saline (Herter).

In the *treatment of the established attack,* bromides constitute the best remedy; they are given not only during the attack in the mild type, but continuously, as in epilepsy, when the type is severe and frequently repeated (Liveing and Charcot). Acetanilide often helps mild forms and may in combination with ergotin, lessen the throbbing pain, but soon loses its effect. Morphine should be carefully avoided. The stomach sometimes rejects all medication, which must then be given by rectum. Application of cold to the head, hot foot baths and sinapisms to the nape of the neck and epigastrium are subordinate helps.

NEURALGIA.

Pain is only a symptom, usually of an affection of a peripheral sensory nerve, although in rare cases centric disease may cause peripheral pain, as affections of the cortex, pons, medulla or parts near the thalamus. The pain of neuralgia, in which there is little or no organic change in the nerve, and the pains of neuritis may be confused.

Etiology.—(*a*) Neuralgia occurs mostly in *adult life* (twenty to sixty) and rarely before puberty or after sixty, in which case it is usually severe and intractable. (*b*) Most cases occur in *women.* (*c*) *Heredity* is an apparent factor in 25 per cent. of cases, especially in neuropathic, weakly or gouty families. (*d*) *Debility* from exhausting conditions, as lactation, anæmia, overwork, sensory fatigue, as from eye strain, sexual or other excesses, emotional conditions and trauma, may cause it. (*e*) It may result from *acute infections,* notably *influenza,* typhoid, especially at its onset, *malaria,* the causal importance of which has been much overestimated, rheumatism (from exposure to cold) particularly of the fascial type, and sometimes syphilis. (*f*) Alcoholism, *diabetes,* plumbism, gout and nephritis may produce it; most of these are also causes of neuritis, differentiation from which may be difficult. (*g*) *Nerve affections,* as sciatica and neuritis, herpes zoster, reflex irritation, as radiated from a carious tooth, compression of a nerve trunk by varices, aneurysms or neuromata or the root pains of tabes, may cause neuralgia. (*h*) Arteriosclerosis may affect the nutrient nerve vessels (Putnam, Dana, Thoma and Dehio).

General Symptoms.—The *pain* presents the following characteristics: (*a*) It is always *paroxysmal,* with intervals of total or relative

relief; intense pain is never constant. (*b*) It *recurs* irregularly at intervals varying from hours to months or with surprising periodicity on a given day or hour, perhaps at the menstrual period; periodicity is as frequent in non-malarial as in malarial cases. (*c*) It is usually *unilateral,* generally constant in its seat, at least for a time, and most often found over the distribution of a single nerve or several nerves, from which it sometimes radiates into other regions; bilateral pain is usually symmetrical. The pain may shift from place to place, especially when due to systemic causes. It is probable that the spinal cord may be the medium of radiation of pain from one region to another and that pain may follow the distribution of the spinal segments rather than that of the peripheral nerves, as in herpes zoster. However, the exact pathology of neuralgia is still uncertain. (*d*) Its *onset* and *cessation* are *sudden and spontaneous;* an imminent attack may be precipitated or existing pain may be aggravated by muscular movements, a breath of air or a simple touch; the character of the pain is *sharp,* shooting, *stabbing,* rarely pulsating; if it travels along the nerve trunk its shooting direction is usually centrifugal, rarely centripetal; if it is localized it is commonly burning or boring. (*e*) The pain is *usually deep,* but the skin is very hyperæsthetic. (*f*) The "*tender points*" of Valleix (1811), later called maximal points by Head, are found in the nerve (50 per cent.) both in the attack and the free interval, especially where the nerve emerges from the bone or fasciæ, lies on hard surfaces, divides or anastomoses. The diagnostic value of occasional tenderness over the spine at the origin of a nerve (Trousseau's *point apophysaire*) is overestimated, because it occurs in many other conditions. In neuritis the nerve trunk along its entire accessible course is tender. (*g*) *Vasomotor constriction,* followed by dilatation, is fairly common. Œdema, erythema, blanching or falling out of the hair, secretory disturbances, herpes, pigmentation, numbness or tingling, trophic alteration of the skin, localized hypertrophies and reflex twitchings of the muscles are much less frequent than in neuritis. Persistent pain or its etiological factors may decidedly reduce the general nutrition.

Local Clinical Varieties.—1. TRIFACIAL NEURALGIA (prosopalgia, *tic douloureux*) is the most important, severe and frequent type, because the fifth nerve is most exposed to trauma, compression and infection, by way of the eye, nose and ear. The entire nerve is not usually affected, though pain may radiate from one division into another; indeed only the secondary branches of a single division may be involved. The general features of neuralgia are present, as already described. (*a*) Involvement of the *first branch* is the most common form; pain occurs over the eye, forehead and frontal sinus (in which the familiar pain is experienced after eating ice-cream); it sometimes radiates into the second and third branches; there are "tender points" over the lower edge of the nasal bone, the supra-orbital notch, less often over the supra-orbital nerve, even to the vertex, sometimes over the occiput or over the cervical spines; cold,

carious teeth, disease of the sinus, glaucoma, hypermetropia, etc., are causal factors. (*b*) When the *second branch* is involved there is pain over the side of the nose and cheek from the infra-orbital region to the upper lip; there are "tender points" over the lower border of the nasal bone, the malar bone, the infra-orbital foramen, the gums over the canine teeth, and sometimes in the hard palate; among other causes is the dental alveolitis in the aged, due to sclerosis. Diseased teeth may cause neuralgia without local dental symptoms and in every severe case the dentist should be consulted. (*c*) Involvement of the *third branch* is the least frequent form; inframaxillary pain, along the lower teeth, tongue, chin, external ear and maxillary region; and tenderness over the auriculotemporal nerve and inferior dental foramen may occur. In old persons the loss of the teeth brings the jaws closer and therefore stretches the inf. dental nerve; it is relieved by proper plates. In severe forms vasomotor symptoms and increased secretion from the eyes, nose and salivary glands are usually present. Hyperalgesia is usual so that the face cannot endure simple contact, and drinking, mastication and wiping the nose cause great suffering. Herpes and trophic changes are probably neuritic rather than neuralgic symptoms. In rare instances facial spasm, transient reflex ptosis, internal strabismus or myosis co-exists. *Differentiation.* In headache and migraine there are no tender points; intracranial disease, as tumor or syphilis, is usually excluded by systematic examination. The *prognosis* is doubtful in severe forms, which are often lifelong in duration and agonizing in degree.

2. Cervico-occipital Neuralgia.—In cervico-occipital neuralgia there is pain in the first four cervical nerves, especially the inferior occipital and auricularis magnus; this pain sometimes radiates into the fifth nerve; there is tenderness half way between the mastoid and spine, over the parietal eminence and over the cervical plexus between the sternomastoid muscle and trapezius; this form is rarely primary, is often bilateral and is less intermittent than other neuralgias, which facts are explained by its frequent connection, especially with cervical caries and syphilis, and also with tumors, meningitis, root disease and affections of the ear.

3. Phrenic Neuralgia.—Phrenic neuralgia is rare, though it sometimes occurs in pericarditis and diaphragmatic pleurisy; it is most often left-sided, with pain and tenderness over the insertion of the diaphragm and nerve trunk in the neck; movements involving the diaphragm are painful. Diagnosis and differentiation are usually uncertain.

4. Cervicobrachial Neuralgia.—In cervicobrachial neuralgia any nerve may be involved, but especially the ulnar and circumflex. Pain may radiate over the entire plexus and into the chest; the tender points are most often in the axilla, shoulder and ulnar distribution; it is most often confused with neuritis from trauma, compression, rheumatic arthritis or other organic disease.

5. Intercostal neuralgia ranks third in frequency after tri-

facial and sciatic neuralgias (neuritis). Pain is felt along the intercostal nerve and tenderness over the *points* of emergence of *its three branches,* over the angle of the ribs, in midaxilla and near the mammary line. It is more easily distinguished in the upper than lower interspaces. Differentiation may be difficult because of muscular rheumatism in which movement is more painful. Peritoneal disease, gastric ulcer, spinal caries, aortic aneurysm and tabes must be distinguished. It is most common in gastrohepatic affections and in neurotic women.

Herpes zoster is probably an acute inflammation of the posterior root ganglia, as shown by Bärensprung and Charcot and later by Head and Campbell. It is mentioned in this connection because of the acute pain preceding and associated with the rash, which appears after three or four days; the pain is often protracted and intractable; the pain on the trunk follows the distribution of the spinal segments rather than that of the intercostal nerves. The ganglion is the seat of a cellulohemorrhagic exudation and its fibers to the posterior column of the cord degenerate.

6. Lumbar Neuralgia.—Lumbar neuralgia is infrequent; it occurs in the iliohypogastric, ilio-inguinal and genitocrural branches, sometimes in the ext. cutaneous, obturator and crural nerves. The pain is felt over the iliac crest, in the inguinal canal, labium or testis (irritable testicle).

7. Sacral Neuralgia.—Sacral neuralgia is infrequent. Coccygodynia is neuralgic and occurs mostly in women. The pain is increased by sitting, coitus and defecation; it is obstinate and resists even operative measures.

8. Pododynia.—In neuralgia *of the feet* there is pain in the heel, called pododynia by Gross; plantar neuralgia, which is mostly neuritic, occurs in acute infections and caisson disease. Morton's metatarsalgia, which was regarded by him as due to bony compression of the fourth metatarsophalangeal nerve, usually occurs in one foot and mostly in women; operation is usually indicated.

9. Visceral Neuralgias.—Visceral neuralgias are considered under diseases of the heart, stomach and kidneys and under hysteria and neurasthenia. Under this caption may be given *Head's areas of pain* referred to visceral disease: the organ is first given, and following it is the area to which its pain is reflected: *Heart:* 1st, 2d, 3d dorsal segments. *Lungs:* 1st, 2d, 3d, 4th, 5th dorsal segments. *Stomach:* 6th, 7th, 8th, 9th dorsal; cardiac end from 6th and 7th. Pyloric end from 9th. *Intestines:* (*a*) Down to upper part of rectum, 9th, 10th, 11th and 12th dorsal. (*b*) Rectum, 2d, 3d and 4th sacral. *Liver and gall-bladder:* 7th, 8th, 9th, 10th dorsal; perhaps 6th dorsal. *Kidney and ureter:* 10th, 11th and 12th. The nearer the lesion lies to the kidney the more is the pain and tenderness associated with the 10th dorsal. The lower the lesion in the ureter the more does the 1st lumbar tend to appear. *Bladder:* (*a*) Mucous membrane and neck of bladder, 1st, 2d, 3d, 4th sacral. (*b*) Overdistention and ineffectual contraction,

11th and 12th dorsal and 1st lumbar. *Prostate:* 10th, 11th, 12th dorsal, 1st, 2d, 3d sacral and 5th lumbar. *Epididymis:* 11th and 12th dorsal and 1st lumbar. *Testis* and *ovary:* 10th dorsal. *Appendages:* 11th and 12th dorsal and 1st lumbar. *Uterus:* (*a*) In contraction. 10th, 11th, 12th dorsal and 1st lumbar. (*b*) Os uteri. 1st, 2d, 3d and 4th sacral (5th lumbar very rarely).

Treatment.—Logically the treatment of the cause (*q. v.*) should precede that of the symptoms, but practically, pain is the prime indication for therapeutic measures.

1. Local Measures.—Pressure on tender points may diminish or abolish pain. As in sciatica, blisters and sinapisms (both of which may leave permanent pigmentation on the face), the cautery, local heat, acupuncture or injections of water or cocaine may give relief. Freezing the skin by ethyl chloride (Richardson, Doddard) often gives better results. Menthol, camphor-chloral (equal parts) and liniments are of some value (tr. aconiti, tr. chloroformi, tr. belladonnæ āā ʒij, linimentum saponis ʒiv). Injections of 1 per cent. solution of osmic acid into the nerve trunk are very successful; they were recommended by Bennett (1889) and later by Murphy; paralysis sometimes results. Schlösser (1900) recommended alcohol injections. Massage is often beneficial, for the muscles and nerves have common veins.

2. Nutrition.—A full diet, with milk and cod-liver oil and also with rest, the Mitchell cure, hydrotherapy and massage, is often beneficial, especially with change of climate or surroundings. Alcohol, which is always dangerous, is most often indicated in older subjects. The beneficial effects of castor oil are often great, probably because it promotes elimination. Gouty subjects may thrive on a vegetable diet.

3. General Drug Treatment.—There are numerous remedies. Iron and possibly phosphorus are valuable in anæmic subjects. Dana gives strychnine hypodermatically in doses of ⅓₀ gr. even up to ⅕ gr.). Arsenic is excellent in anæmic, malarial and degenerative types; quinine is beneficial in non-malarial as well as malarial types and in trifacial neuralgia especially, although its value is considerably overestimated. Valerian, cannabis indica and bromides should be given for irritability; salicylates for rheumatism and colchicum and cimicifuga for gout; iodides are sometimes of benefit to non-syphilitic subjects; full doses of nitroglycerine are frequently helpful.

4. Analgesics.—Acetanilide (gr. v) and the other coal-tars should be used in fresh cases. Extr. cannabis indicæ (gr. ¼) and fluidextr. gelsemii (♏ii to iv) should be administered every three to six hours in the less severe cases. In acute febrile or rheumatic cases, fluidextr. aconiti (♏i to ii) should be given every two hours until tingling in the mouth or extremities and slowing in the pulse intervene. Liebreich advocates croton-chloral, gr. v, or with care even gr. xx for several doses. Finally, in severe cases, morphine, as far as possible, should be avoided; sometimes its administration is inevitable; though some writers find that it tends to prevent recurrence,

the pain often develops again, intensified; it should be combined with atropine, which may be given alone, as it sometimes more permanently controls the pain than does the morphine.

5. Electrotherapy.—This sometimes relieves the pain, but it more often fails. Galvanization with a weak current, which should be just strong enough to produce a sensation of burning and should be applied with the sponges warm and with the anode to the painful area, may be beneficial, especially in newly developed cases.

6. Surgical Therapy.—This includes section, resection, stretching, twisting or divulsion of the nerves; resection should be tried first, but all these procedures may fail. In incoërcible trigeminal neuralgia the Gasserian ganglion may be extirpated (Hartley, Krause, Keen, Cushing), but this operation is severe and loss of the eye may result. Horsley opens the skull and divides the root of the fifth nerve above the ganglion, but this is dangerous; Spiller and Frazier therefore suggest its division without opening the cranial cavity.

VASOMOTOR AND TROPHIC NEUROSES.

Vasomotor centres exist in the cerebral cortex and medulla. The fibers probably run in the lateral columns of the cord and leave by the anterior roots. *Vasomotor paralysis* occurs independently or with functional or organic disease. Paralysis of the cervical sympathetic may result from trauma, aneurysm, tuberculous apical or glandular disease or disease of the cord. It is attended by redness, rise of temperature, pulsation and tortuosity of the vessels, sweating, unrest, ringing in the ears and palpitation. In paralysis of the cervical sympathetic there are pupillary narrowing from the unopposed action of the third nerve, slight ptosis, narrowing of the interpalpebral fissure from paresis of Müller's muscle, salivation, lachrymation and in cases of some standing, retraction of the eye and flattening of the cheek. The pupil reacts somewhat to light but not to stimulation by pain or pinching of the skin of the neck (Möbius); the pupil can be widened by atropine, but to a less degree than in the normal eye.

Vasomotor spasm presents converse symptoms,—pallor, coldness, small vessels and frequently a sense of tingling or arterial pain. Two examples are nervous angina pectoris and Raynaud's disease; spasm (contraction, irritation) of the cervical sympathetic produces wide pupils, an increased interpalpebral fissure and exophthalmos.

Little is known of the *trophic nerves,* if indeed separate nerves subserve nutrition. If they exist they are probably closely related to the vasomotor tracts. Trophic changes may be symptomatic of disease of the nerves or cord, as in neuritis or nerve trauma, facial hemiatrophy, tabes, syringomyelia and myelitis, under which their special features have been already discussed.

Erythromelalgia.—Erythromelalgia was described first by Graves (1843) and S. Weir Mitchell (1872); the term indicates the chief clinical characters, *redness and pain in the extremities.* It is rare,

though much more frequent than is shown by Voorhees' series of 70 cases (1907). It occurs mostly between the years of twenty-five and forty and is slightly more frequent in males. Its pathology is not wholly clear, although Mitchell and Spiller find neuritis and sclerosed arteries. The toes (less often the fingers) and balls of the toes are the seat of severe, usually paroxysmal pain, swelling and redness; the arteries throb and the veins are engorged; the color is sometimes blue. Pain is sometimes experienced in the head and neck and vertigo and muscular weakness may be noted. The symptoms occur mostly in warm weather and generally disappear in winter. The clinical course is long, the prognosis doubtful and the treatment consists of electrotherapy, cool baths, acetanilide and iodides.

Acroparæsthesia.—Acroparæsthesia is closely related to erythromelalgia. It was described first by Nothnagel, and F. Schultze (1890) named this very frequent affection; its derivation signifies *paræsthesia of the extremities.* More than 90 per cent. of cases occur in women between thirty and sixty years of age. The etiology is doubtful (stasis or arteriosclerosis). Its chief feature is an unpleasant crawling, tingling sensation, sometimes amounting to actual pain in the fingers or hands, less often in the toes or feet. The discomfort is greatest at night and in the early morning; it is peripheral and symmetrical but does not correspond with any nerve area. Generally there are no objective findings other than occasional vasomotor disturbance or slight sensory blunting. The outlook is favorable, the course is chronic and treatment is symptomatic; the faradic brush frequently affords relief.

Spontaneous Symmetrical Gangrene, or **Raynaud's Disease.**—Raynaud's disease was first described by Raynaud (1862). Sixty-six per cent. of cases occur in women, who are usually delicate, anæmic and neurotic, and most cases develop in persons between twenty and forty-five years of age. In 10 per cent. it is hereditary. Other causal factors are uncertain, as exposure to cold and dampness, exhaustion, worry and acute infections. Occasional co-existence is noted with hysteria, neurasthenia, epilepsy, tabes, syringomyelia, angioneurotic œdema, neuritis, acute mania, etc. Its pathology is not wholly established, but arterial spasm, endarteritis and neuritis are said to exist. There are *three stages:* (*a*) *Local ischæmia* (or syncope), begins with pallor and coldness of one or two fingers, perhaps of the entire hand or of the toes and is attended by numbness or severe pains. The "dead fingers" or toes appear on exposure to slight cold or on emotion and reaction occurs after a variable period. The process may not pass this stage. (*b*) *Local asphyxia* (or cyanosis) develops in the parts involved, at times with some swelling or anæsthesia. It is most severe in winter and chilblains are regarded as the mildest type of this stage. (*c*) *Local gangrene,* which is spontaneous, symmetrical and fortunately rare, is the last stage. It is dry, sometimes vesicular and its extent is always less than the area of ischæmia and asphyxia. The tips of the fingers or toes dry up, fall

off and scars mark successive attacks. Less often the tip of the nose, ear, buttock, heel, shoulder or abdomen is involved and very rarely the tongue, penis, elbow or knee. Fever is usually absent. In some instances there is severe general disturbance, as fever, chill, enlarged spleen, colic, hæmoglobinuria, uræmia, stupor, coma, convulsions, transient hemiplegia, neuritis and spastic retinal vessels. The severe or fatal cases occur especially in children. The *course* is chronic and consists of repeated attacks, which often abate in summer.

Differentiation.—Differentiation is usually easy from hysterical gangrene (self-injury), diabetic, arteriosclerotic or embolic gangrene, syringomyelia, leprosy, ergotism and acute arteritis, as in typhoid.

Treatment.—In treatment narcotics are given for pain; warmth and elevation promote venous return and nitroglycerine dilates the arteries. Cushing advises Esmarch's constrictor for a few minutes. after which the circulation is restored. In actual gangrene surgical interference should be postponed until complete demarkation develops, because the area is much more circumscribed than at first indicated.

Acute Angioneurotic Œdema.—Acute angioneurotic œdema was first fully described by Quincke (1882), although Milton (1876) described "giant urticaria." It occurs most frequently between the years of twenty and thirty-five and is slightly more frequent in women than in men. Hereditary forms are known; 22 cases occurred in 5 (Osler) and 22 cases in 6 (Milroy) generations of one family. It is allied to urticaria, erythema nodosum and possibly to some forms of purpura and consists of a circumscribed œdema, which is sudden in onset and in disappearance, is tense and pale, does not pit on pressure, sometimes causes burning and appears mostly on the eyelids, lips, cheek, tongue, hands or genitalia, sometimes on the buttocks, in the pharynx, larynx, lung or digestive tract. It sometimes attacks several areas simultaneously. Gastro-intestinal symptoms occur in 33 to 50 per cent. of cases, as epigastric pain, vomiting, colic or diarrhœa (see page 830). Hæmoglobinuria, albuminuria and polyuria are less frequent. The outlook is good, except for the dangerous laryngeal or pharyngeal localization which may cause sudden death. *Recurrence* is not uncommon and sometimes occurs with considerable periodicity.

Treatment.—Treatment is symptomatic and general; nitroglycerine has been recommended. Bloodgood employed deep incisions with great benefit.

Herz recently described a *pseudoperiostitis angioneurotica,* occurring especially on the thoracic bones; it is periodic, transitory, painful, tender and associated with paroxysmal, neurotic, cardiac disturbance.

Chronic Hereditary Trophœdema.—Twenty-two cases of chronic hereditary trophœdema in six generations were described by Milroy (1893) of Omaha, and 8 cases in four generations by Meige (1898). A firm, pale, painless œdema, chiefly below the hip or knee, develops toward puberty, without cardiac, renal or apparent local causes. It

may appear in the arm and leg of the same side, including probably some of the so-called local hemihypertrophies. The first cases recorded were thought to be phlebitis. Health and life are not threatened, although treatment produces no results.

Hydrops Articulorum Intermittens (hydrarthrosis intermittens).—Hydrops articulorum intermittens occurs with equal frequency in both sexes and 71 per cent. occur in persons between ten and forty years of age. Its etiology is not known. Some cases present malarial, gonorrhœal or traumatic antecedents. The knee is usually involved when several joints are affected and in 33 per cent. is alone involved. The effusion wholly disappears after two or three days, but it reappears obstinately every one to four weeks. There is no fever and usually no pain. *Treatment* is general.

Scleroderma.—Scleroderma was described by Lusitanus and Alibert, though Thirial (1845) gave the first full account of it. It is a chronic thickening and hardening of the skin, followed by atrophy. It is a rare affection, but is more common than is generally recognized. The *localized form* usually develops before and the *generalized form* after the twenty-fifth year. Seventy-five per cent. of cases occur in women. The *causation* is obscure; erysipelas, trauma, cold and neuropathic disposition are sometimes considered etiological and association with other neuroses, general and vasomotor, is frequent. *Anatomically* the skin and all the contained structures are fibrosed. In the localized form it occurs in spots or streaks, which often correspond to the course of given nerve trunks or spinal segments. In the general form the body may be literally "hide bound." Its distribution in order of frequency is (*a*) the upper extremities; (*b*) the trunk; (*c*) the head and face; (*d*) the lower extremities; (*e*) generalized. The skin is usually first painful and sensitive, then reddened and thickened, and finally pale, glossy, dry, atrophic and parchment-like. It is rigid and without folds, inhibiting the mimetic play of the features. The nose is peaked, the lids cannot close over the eyeball and may become everted. The mouth is so small as to sometimes necessitate extraction of some of the teeth and rigidity in the neck may render swallowing difficult. In the fingers it occurs in 5 per cent. of cases (*sclerodactylia,* Ball). The close skin produces decided deformity and loss of function. Sensation is usually preserved, the secretion of sweat is usually reduced and pigmentation and trophic changes in the hair and nails are occasionally seen. In some cases it occurs with herpes (the herpetiform morphea of Hutchinson). Circumscribed atrophy of the bones is said to occur. Only 30 per cent. of cases improve and 15 per cent. recover, whence the outlook is generally unfavorable.

Treatment.—Treatment is unsatisfactory. Improvement has followed administration of salol and inunctions with oil; thyroid extract (Osler, Lustgarten, Sachs and Bramwell), especially if used early in the disease, may promote recovery.

Ainhum.—Ainhum was first observed by Clark (1860) then by Da

Silva Lima (1866). It is a chronic affection, usually limited to the great toe, which at its plantodigital fold suffers a slow, painless, non-inflammatory circular constriction and spontaneous amputation. Ainhum is chiefly found in the colored races of Brazil, Africa, India and sometimes in this country. Its nature is disputed; an ingrowth of the epidermis and scleroderma have been thought to be causes. It is not amputating leprosy, as has been claimed.

DISEASES OF THE MUSCLES.

MYOSITIS.

Inflammation in the muscles may be single or multiple; it may be an independent affection or a secondary one. It may reach the muscles by contiguity from adjacent disease, may result from trauma, trichinosis, infection with cysticercus, echinococcus, myxo-, sarco- and microsporidia, sepsis, puerperal fever, rheumatism, scarlatina, gonorrhœa, syphilis or erythema multiforme. The muscles are the seat of serous or sometimes hemorrhagic or diffuse purulent exudation in septic forms and of fatty degeneration. There is a form, endemic in Japan, in which the Staphylococcus pyogenes aureus occurs in pure culture. The muscles are usually friable and sometimes fragmented. *Polymyositis,* first described by Wagner (1887), Hepp and Unverricht, occurs most often in youth and adolescence and is attended by pain, especially near the tendons, swelling and often œdema in the muscles, the electrical irritability of which is decreased as also are the tendon reflexes. The skin may participate, and herpes, erythema, bullæ or purpura may be present (*dermatomyositis*); the infection may involve the tissue in which the afferent nerves originate (muscle spindles) and topographically a neuritis and myositis may co-exist or coincide (*neuromyositis,* Senator). In some cases there is arthritis or phlebitis. Oppenheim describes a case of *dermatomucomyositis* with stomatitis, iritis and conjunctivitis. The inflammation occasionally reaches the tongue, pharynx, larynx or respiratory muscles and may cause difficult mastication or swallowing, ptosis or dyspnœa. The systemic reaction is severe, is marked by fever, chills, sweats, splenic tumor, sometimes by angina, bronchitis, albuminuria or leukocytosis. Death is the usual issue.

Differentiation.—Differentiation concerns *multiple neuritis* (tenderness over the nerve trunks, motor and sensory findings); trichinosis (trichinæ in the tender, swollen muscles, eosinophilia and digestive symptoms); and *periarteritis nodosa* (*q. v.*).

Treatment.—Treatment is symptomatic and is identical with that of sepsis. Sweats and salicylates are beneficial.

Myositis Ossificans.—Myositis ossificans is characterized by a myositis which usually develops acutely in the muscles of the neck or back before the fifteenth year and leaves residual foci of infiltration, partly in the periosteum and partly in the fasciæ and tendons, which gradually calcify. The thigh and arm are most severely and permanently affected. The process occurs in the strongest muscles and at an age when they develop most actively; it may affect many muscles and runs a chronic course, perhaps of several decades. One hundred and thirty-four cases are recorded (Cahier); there were 101 traumatic cases (Werner, 1904). Massage is sometimes beneficial.

MUSCULAR RHEUMATISM (MYALGIA).

This includes many vague but important affections. It occurs particularly in men, after exposure to cold and dampness, and is obviously promoted by certain occupations. Muscular overexertion, as straining and lifting, is frequently a part of the history. Rheumatic and gouty subjects are specially exposed and to recurrent seizures. Disorders of metabolism or mental as well as physical overwork mark some cases. Other forms are, as Leube insists, probably infectious, for fever occurs in 33 per cent. and is occasionally followed by endocarditis; muscular rheumatism in this sense does not generally involve many muscles, but usually one muscle or a single group. The pain is not paroxysmal, as in neuralgia, and is experienced in the muscle or reaches it from its tendons or fasciæ. In some cases it is apparently a mild neuromyositis. Its varieties are: *Myalgia capitis,* affecting the fascia of the head; *omalgia,* involving the muscles of the shoulder, or rheumatic *torticollis,* which is usually unilateral; *pleurodynia,* which affects the chest muscles, pectoralis, intercostals and serratus, limits respiratory movements and is likely to be confused with intercostal neuralgia, pleurisy, periostitis of the ribs or vertebral disease; *lumbago,* which involves the lumbar muscles, and is the most frequent, painful and obstinate form, causing great pain on movement or on efforts of the uninvolved muscles to maintain quiet; it is probably due in some cases to muscular rupture, as after lifting. In the abdominal muscles and glutei it may simulate pelvic disease. In *treatment,* rest is urgently indicated, for use of the affected muscles aggravates pain and protracts the condition. Involuntary movement is prevented by careful strapping with adhesive plaster, especially when there is pain in the chest. A hypodermic of morphine into the muscle is often indicated. Deep injection of water into the muscles of the back (or affected part) may relieve pain. Ringer's method of acupuncture with long needles which are pushed three inches into the back and left there for from five to ten minutes is beneficial, but is usually vigorously opposed by the patient. Very energetic massage, with the local application of heat, probably gives the best results. The actual cautery, the application of the constant current and the use of Tesla's coil give good results. The use of belladonna plasters

gives relief. Gelsemium, acetanilide and salicylates often produce no effect, but may instantly relieve certain cases.

Chronic cases, with vague, fugitive pains which come on with change of weather, are treated in the same way—usually without result. Confusion with chronic alcoholism, tabes, gout and diabetes is frequent.

SECTION X.

INTOXICATIONS. SUNSTROKE.

ALCOHOLISM.

I. Acute Alcoholism.—(*a*) The *highest brain centres* are first affected. The judgment is first involved (emotion predominating), motility next (as shown by the disordered speech, gait and vision), then the lower centres (respiratory, circulatory and spinal). (*b*) The *circulation* is increased and the pulse becomes more rapid, though alcohol does not stimulate the heart. Full doses may make the pulse slow and small. The vascular tone is lax and the face is flushed; for this reason alcoholics suffer little shock from coincident traumatism. (*c*) *Respiration* is slow, deep and seldom stertorous. (*d*) The *pupils* are variable, but more often dilated than narrow and reactionless. (*e*) The *temperature* may fall markedly, from vasoparesis, and usually with some cyanosis and failing of the bulbar centres; Reincke states that temperature has fallen as low as 75°! Involuntary evacuations occur in the severest cases and there may be subcutaneous hemorrhages.

Dipsomania is intermittent acute alcoholism, an hereditary psychosis in which, after intervals of abstinence and of no craving for liquor, the patient becomes intoxicated.

Diagnosis.—The alcoholic breath is usually suggestive but epileptics and uræmics may drink, and while drunk a man may have his skull broken or suffer an apoplexy. Many mistaken diagnoses result from carelessness, but many are unavoidable, and the apparently drunk patient should be treated for alcoholism, while other possibilities should be carefully considered. As a rule the patient can be aroused, but in "dead drunks" this is not the case and incomplete coma may occur from other causes. The condition of the pupils is not of certain diagnostic value, though they are usually dilated in alcoholic, contracted in opium and uræmic, and unequal in apoplectic, coma. Stertorous breathing is strongly indicative of apoplexy or uræmia. Convulsions following delirium and muscular twitchings are less common than in other similar conditions, but at the time of writing the author had a case which showed uni- and then bilateral convulsions. The urine rarely contains alcohol, as but 1 or at the most 5 per cent. of ingested alcohol is excreted by the lungs and kidneys. Sudden death may occur without adequate necropsy findings. One-

third of all the fatal poisonings in New York and Berlin are acute alcoholism.

Treatment.—Gastric lavage is sufficient in most cases. Even the convulsions require little attention, but when severe, yield to a few whiffs of chloroform. Acute mania yields to a hypodermic of apomorphinæ hydrochloridum (gr. ⅙). Collapse, cold extremities and cardiac and respiratory failure are relieved by hot coffee, strychnine, atropine, aromatic spirits of ammonia and local heat, as will be more fully outlined under acute opium poisoning (*v. i.*).

II. Chronic Alcoholism.—This is less an hereditary tendency toward drinking than a lack of character, though Plutarch said that drunkards beget drunkards. The constant use of alcohol has varying effects: some people can take beer, wine, spirits or patent medicines throughout life without apparent injury; in others, even the mildest alcoholic drinks may induce visceral alterations, so that as in liver cirrhosis, it would seem that there is some other etiological factor; however, alcohol in health is a bad food, an injurious narcotic or stimulant, a protoplasmic poison to the parenchymatous organs and arteries and a check on metabolism.

Symptoms.—(*a*) *Nervous manifestations.* Functional changes include the alcoholic tremor, which is a most common symptom; mental dulness and weakness; nervousness, jumping from slight stimuli, irritability, impaired judgment and feeble will power; neuralgias and muscular rheumatism. The alcoholic is suspicious, cruel, conceited and a degenerate. Quinquad's sign is elicited by having the patient place his fingers vertically to the examiner's hand or body, when a series of jerking shock-like movements is noted; Fürbringer decides that (i) if it is absent, alcoholism is absent; (ii) if it is moderate there is uncertainty and (iii) if it is marked, in 3 cases out of 5, the patient is an alcoholic. Of organic nervous manifestations, multiple neuritis is the most common; it may result from small amounts of alcohol, when there is a coincident metallic poisoning; alcoholism is much more often a promoting factor than an actual cause of paretic dementia; epilepsy may result from alcoholism; hemorrhagic pachymeningitis, chronic leptomeningitis, acute polioencephalitis, degeneration in the anterior horns or Clark's columns, degeneration of the cerebral nervous tissue and slight optic neuritis may be more or less direct sequences. (*b*) The *circulation.* Arteriosclerosis often develops with its train of sequences, cardiac hypertrophy and dilatation, angina pectoris, cerebral softening or hemorrhage. (*c*) *Chronic bronchitis,* emphysema, chronic pharyngitis and laryngeal thickening are frequent collateral findings. (*d*) The *digestive organs.* Alcoholic gastritis (*q. v.*) is common; the morning vomiting (vomitus matutinus) is quite characteristic and is quieted only by another dose of its cause. Beer drinkers usually have large stomachs. Alcoholic cirrhosis has been already discussed; fatty degeneration, arterial congestion and hypertrophy may antedate or accompany cirrhosis. Beer drinkers have lax bowels, while consumers of spirits are constipated. Hemor

rhoids are common. (*e*) The *kidneys* undoubtedly suffer. Dickinson, Formad and Pitt think that renal disease is less common than is usually believed; Formad and Pit found that the kidneys were hypertrophied without other change in 43 per cent. As in cirrhosis, several factors may be necessary to produce indurated kidneys. Sterility is common. (*f*) *Metabolic maladies,* as gout, obesity and, according to Strümpell, some forms of diabetes may result from chronic alcoholism. (*g*) The *skin* frequently presents the acne rosacea, a very common but not, as Trousseau says, an "indelible stigma." The vessels of the nose, cheeks and often of other parts are suggestively dilated; the conjunctivæ are injected and the eyes water.

Prognosis.—The prognosis is unfavorable, both from the standpoint of curability and complications. English life insurance companies estimate that the risk is 33 per cent. less in abstainers than in alcoholics. The general resistance is lowered, thus favoring infections, as tuberculosis, and raising greatly the death rate, most notably in pneumonia. Twenty per cent. of suicides are alcoholics. The offspring of inebriates are disposed to numerous nervous affections.

Treatment.—Treatment consists of correction of the habit and care of the complications, the latter of which have, in great part, already been considered. The cure of the habit is largely personal. Its prophylaxis is better than its cure. No person, however intellectual or strong-willed, can safely drink with any regularity. Institutional treatment is generally necessary. The vomiting should be treated in accordance with instructions given under acute and chronic gastritis; lavage is probably the most reasonable measure; a mixture of bismuth (see Typhoid, Vomiting) and small doses (♏i to ii) of Fowler's solution and large ones of tr. nucis vom. (♏x) are valuable just before the administration of hot milk with tr. capsici, ♏x. Hyoscinæ hydrobromidum (gr. 1/120) should be given two or three times at 3 hour intervals, chloral hydrate (gr. x) and sod. bromide (ʒss) for three or four doses, before bedtime, or sulphonal (gr. xx) in hot milk before bedtime, in order to induce sleep. Atropine (gr. 1/120) and strychnine (gr. 1/30) four or six times daily, hypodermatically, quiet the nervousness, which occurs in the first days of abstinence. Sipping ice-water, sucking lemons or drinking water with tr. quassiæ may satisfy the desire to "drink something." Ergot has little effect on the circulation (Sollmann and Brown). Cocaine and morphine should be avoided, lest a substitution or succession of habits develop. Relapse is very common.

III. Delirium Tremens.—Delirium tremens (*mania a potu*) is an acute accident of chronic alcoholism and almost never follows an acute debauch in an otherwise temperate individual. It was noted among sailors by Sutton of Greenwich in 1813, but was probably best described by Ware of Boston in 1831. Sixty-six per cent. of cases occur in men, while multiple neuritis is more common in women (Gowers); most victims are between thirty and forty years of age. The patient has been, on the average, an alcoholic for 8 years

before delirium tremens develops. The stronger spirits, rather than beer or wine, are the cause of delirium tremens, as they are of alcoholic convulsions. It may be precipitated by trauma to the chest or fracture of the limbs, mental shock, by sudden failure of the stomach to digest and assimilate food, acute withdrawal of alcohol or, in 70 per cent. of cases, the shock of onset of an acute infection.

Symptoms.—The symptoms develop with restlessness and insomnia, which renewed drinking fails to allay; for a day or two the mind wanders, but can be brought back to realities. The patient may be facetious, but is soon disturbed by horrible hallucinations. There is "great versatility of ideas" in delirium tremens. The patient talks volubly, turns suspiciously to the head of his bed, drives his horses vigorously and brushes away the vermin from the coverlet; hallucinations of hearing are also common. He at first knows that the bugs and snakes are unreal, but they soon become actual torments, so that unless watched he will leap from the window after waiting for his opportunity. The pulse is rapid and soft, the skin is flushed or slightly cyanotic and covered with sweat, the tongue is tremulous and is coated white, the urine is scanty and often albuminous and sleep is constantly absent. There may be moderate fever, 101 to 103°, which some attribute rather to complications.

Diagnosis and Prognosis.—The *diagnosis* is rarely uncertain. The chief danger of error is in overlooking coincident causal trauma and infection, as pneumonia. In heavy and constant drinkers a delirium may be delirium tremens or an "associated delirium," as from apical pneumonia.

The *outlook* is always uncertain. The early convulsions are of no great moment (Gowers). The prognosis is determined by the pulse, the general nutrition and complicating alcoholic or other lesions. Rigidity of the muscles of the neck is ominous (Putnam and Dana) and indicates œdema of the brain. The average mortality is 15 to 20 per cent., but varies as the affection is (*a*) uncomplicated (1 per cent. die, Lambert); (*b*) associated with other affections (grave outlook) or (*c*) occurs in the aged or debilitated (probably fatal outlook). Recurrence after recovery is frequent, and 27 attacks in one case have been recorded. In fatal cases death usually results from exhaustion and heart failure; the tongue becomes dry, the delirium constant and the skin cold and clammy. The autopsy shows œdema of the brain ("wet brain," Dana), degenerative changes in the brain and cord (Bonhöffer), arterial alteration (Collins) and hypostasis of the bases of the lungs.

Treatment.—(*a*) The patient should be *kept in bed* and watched constantly lest he escape. Strapping the ankles and wrists and confining him by a straight jacket is a harsh procedure and invites trauma to the hands and feet, but is inevitable when the family is indulgent or the nurses are negligent. (*b*) *Withdrawal of alcohol* is usually advocated but is a severe measure; the continuance of moderate doses seems to hold the pulse steady and favors the diges-

tion and is, in the author's belief, indicated. (*c*) *Sleep is induced* with great difficulty and as Ware first pointed out, comes when the disease runs its course. Excessive medication should be avoided. Sod. bromide (3ss), tr. capsici (ɱv) and whiskey (3ii), in hot peptonized milk should be given every three hours, by mouth if possible, or by the nasal catheter, for nutrient enemata are difficult to give and are seldom retained. Chloral is always dangerous in inveterate alcoholism. Hyoscine with strychnine may be beneficial (*v. s.*). Morphine should be given only by the hypodermic method, for its absorption can then be measured; it was formerly given to excess. Protracted warm baths may quiet the patient (Bonhöffer), but they are hard to administer. In 6 cases in which the violent delirium became ominous the writer has etherized the patient, and in 3 instances with excellent results, though he does not advocate its wide use. (*d*) The *circulation* should be treated as in pneumonia. Large doses of digitalis are not indicated; they are tolerated only because they are not absorbed; the original treatment by digitalis is based on the fact that a nurse by mistake gave a toxic dose of digitalis; the patient improved and the "digitalis treatment" was thus instituted. (*e*) *Cold affusions* upon the neck and chest are recommended by Broadbent.

OPIUM POISONING.

I. Acute Poisoning.—This is important chiefly from a diagnostic standpoint. In the second stage it is strongly characterized by profound sleep, dry, flushed skin, narrow pupils, slow, strong "digitalis pulse" and slow deep stertorous breathing. The third or terminal stage is different; the skin becomes cyanotic and clammy, the narrowed pupils may dilate, the pulse become weaker (though the heart centers live after the other bulbar centres have died), respiration halts and is very slow. Mistakes in diagnosis are made by attributing undue importance to the "pin point" condition of the pupils. Morphine may be detected in the stomach washings.

Treatment.—This consists of (*a*) *mechanical evacuation* by repeated gastric lavage. (*b*) *Chemical antidotes,* such as large amounts of tannic acid, should be given with the lavage and removed as the tannates formed may dissolve; 1 to 1,000 permanganate of potash (Moor, of New York) may be given by mouth or hypodermatically. (*c*) *Respiration* must be sustained by walking the patient (the ambulatory treatment), but *fatigue must be guarded against;* phrenic faradization is indicated, but care is necessary lest too strong a current produce paralysis instead of stimulation; strychnine plus atropine may be given hypodermatically, but the total dose of atropine should not exceed gr. $\frac{1}{30}$ to $\frac{1}{20}$; pulling slowly and rhythmically on the tongue may help respiration; Sylvester-Hall's artificial respiration and oxygen inhalation may be used. (*d*) *The heart* should be stimulated by coffee, strychnine, heat and digitalis.

II. Chronic Morphinism (Morphinomania).—The use of morphine

for pleasure only, so common in Asia, is rare with us; those taking morphine for pain and resisting its attractions are called morphinists; those using it for pleasure only are morphinomaniacs. The habit is contracted by its use for insomnia or for mental or physical pain. Forty-seven per cent. of morphine victims are said to be physicians; women, particularly prostitutes and nurses, druggists and—in Germany—officers rank next. It may be taken for years without mental or physical deterioration, but morphinists are usually less happy, as increase in the daily dose becomes necessary and the half-oxidized alkaloid produces symptoms (*v. i.* TREATMENT) which are only allayed by larger doses. Morphine acts as a nerve poison. The victim becomes restless and irritable without his dose. The mental and moral attributes are anæsthetized and there is a "moral insanity." The patients lie by choice, neurotic symptoms are common, muscular and mental asthenia develop, the pupils are contracted, the sweat and saliva are decreased, the teeth decay, the appetite fails, the skin becomes sallow, itchy and dry, the hair coarse and gray, constipation is usual and slight ataxia may develop.

Prophylaxis.—Physicians should not give opiates freely. When administered for severe pain the habit is less often contracted than when given to neurotics. At least 70 per cent. of cases relapse after a cure.

Treatment.—Treatment is almost impossible without (*a*) *institutional confinement,* which must of course be voluntary. (*b*) The *morphine* may be *withdrawn* abruptly, rather suddenly or very gradually; the second method is the best, stopping the morphine entirely in four to seven days. Its withdrawal is attended by awakening of dormant anæsthetized functions; there is at first paroxysmal yawning and sneezing; roughening of the voice, paræsthesia, neuralgias especially in the legs, clammy skin or rapid pulse, wide pupils, tremor, sexual excitement, extreme irritability, vomiting, colic and diarrhœa also occur. The symptoms were attributed by Marme to di-oxymorphine poisoning, the antidote to which is morphine; Hitzig believes that they are largely due to hyperchlorhydria, for the gastric nerves under the influence of morphine secrete little hydrochloric acid; relief is obtained by washing out the stomach with a solution of Carlsbad salts. The patient will obtain morphine if possible. (*c*) The *patient should be kept in bed* for a week to obviate vasoparetic cardiac collapse. (*d*) *Symptomatic treatment* is indicated (i) by the *collapse* (aromatic spts. of ammonia, ♏xx, strychnia gr. $\frac{1}{30}$, heat); (ii) by *nervousness* (warm baths, sodium bromide in doses of one dram every four hours); (iii) by the *insomnia* (hyoscine gr. $\frac{1}{120}$ every two hours for two to six doses, sulphonal or trional, gr. xx); (iv) by the *gastralgia* (gastric lavage with alkaline salts); (v) by *diarrhœa* and colic (as in enteritis, but vegetable astringents, large doses of bismuth, and no opiates should be used); (vi) by *neuralgia* (acetanilide, gr. v, fluidextr. gelsemii, ♏iij). Whatever treatment is instituted, alcoholism, chloralism, bromism or cocainism should not be substituted.

LEAD POISONING.

Etiology.—The importance of plumbism or saturnism is increasing. (*a*) *Industrial plumbism* is very common. Workers in white or red lead factories usually acquire the disease. Miners of the metal generally escape, but smelters of lead ore and even animals and birds near the furnaces are frequently affected. Painters, plumbers, glaziers, less often tinners, printers, cameo polishers, cartridge makers, etc., may be poisoned. (*b*) *Accidental contamination of food or drink.* Poisoning may result from the action of acids in canned foods on the tin or solder, as in cans of sardines; wine or cider may become poisonous in the same way, as happened in Devonshire and in Poitou (*colica pictonum*). Chromate foil, covering hams or candy, rubber nipples, bullets in pickled game, bread ground in lead vessels, chromate used to color buns (D. D. Stewart's report of the fatal Philadelphia epidemic), Seltzer or other charged waters, wine and *eau de vie* sweetened or cleared with litharge, are a few of the many subtle causes of saturnism. Drinking water conveyed through lead pipes is generally protected by the deposit of lime and other salts, but poisoning is possible when the water is exceptionally pure or soft, thereby depositing little lime in the pipes, or when it is saturated with organic matter or collected from lead roofs. (*c*) Less frequent sources of plumbism are in the environment, and in such things as hair-dyes, powders or cosmetics, linen, freshly painted walls, candles, sealing wax, brightly tinted toys, false teeth and thread. (*d*) *Therapeutic* and *criminal plumbism* is rare; it may result from the acetate of lead when given internally or externally, and from impure subnitrate of bismuth; its criminal use is very uncommon. (*e*) *Predisposing factors* are alcoholism, age (it occurs most often in persons from thirty to forty) and sex (75 per cent. of cases are females, Olivier). Susceptibility varies greatly; plumbism may cause death within a week or may not develop until after forty-two years of work in the metal (Tanquerel des Planches, 1838). *Absorption of lead* usually occurs (i) by the digestive mucosa; most of the metal is precipitated by the albumin of the food and eliminated without absorption; (ii) by the respiratory mucosa, by inspiration, as dust in white lead factories; or (iii) by the sound or diseased skin (cosmetics) or even the conjunctiva or vagina (lead douches). It is *eliminated* chiefly by the urine; Putnam stated that he found lead in 25 per cent. of 150 persons who had no evidence of plumbism; the liver and alimentary mucosa eliminate smaller quantities.

I. Acute Poisoning.—This is uncommon, though it may occur from therapeutic, criminal or professional causes. Its symptoms are (*a*) gastro-intestinal; a styptic burning taste occurs in the mouth, thirst is present, there is a lead line on the gums in some cases, though less than in chronic poisoning, nausea and vomiting sometimes of white lead chloride, abdominal colic, retraction of the abdomen, diarrhœa with black (lead sulphide) or bloody movements or sometimes con-

stipation. On postmortem examination, the catarrhal gastro-enteritis may be wrongly considered a postmortem change; white spots may be seen (Orfila's spots). The symptoms may be (*b*) circulatory; the pulse may be slow (50 to 40) and irregular; anæmia, cyanosis and collapse may occur; (*c*) they may be nervous; vertigo, stupor, delirium, neuralgia, cramps, convulsions and even cerebral and peripheral paralyses may develop; (*d*) nephritis with suppression of urine may develop.

Treatment.—Treatment consists of full doses of sodium and magnesium sulphate, in order to precipitate an insoluble lead sulphate, and to remove it, demulcent drinks (eggs and mucilages), opium for pain and cardiants.

II. Chronic Poisoning.—Symptoms.—(*a*) *Colic* is the most common symptom; it occurred in 88 per cent. of Tanquerel's 1,390 cases. It is due to local deposit of lead in the intestinal muscles or mucosa, spasm of the bowel, changes in the nerves or angiospasm. It may develop early or late; it may be preceded by malaise, lumbago or pain in the legs or may be brought on by alcoholic excesses. It is a colic accompanied by constipation (dry colic), rarely by diarrhœa; it is umbilical, whence it spreads over the abdomen, perhaps to the legs or scrotum; it is relieved by pressure, though sometimes the abdominal wall is hyperæsthetic; sometimes vomiting and absolute constipation may simulate obstruction, and in one case the author observed intussusception as a result of lead poisoning. There is no fever, the pulse is small and hard (Riegel) and the urine is scanty. Without treatment colic is protracted; its relief is usually marked by a bowel movement. Relapses are very frequent. (*b*) The *gingival lead line* (Burton, 1834) present in over 90 per cent., is a gray-black line, 2 to 3 mm. wide, which is seen most clearly on the lower gums and is due to deposition from the blood of lead sulphide, precipitated by the sulphuretted hydrogen of the tartar. The coincident anæmia brings it out very clearly. Similar deposits may occur under the buccal mucosa opposite the molars. It is very frequently mistaken for tartar, which occurs on the teeth, sometimes for the black carbon line seen in miners, or rarely for the silver line in argyria. (*c*) *Other digestive symptoms* are coated tongue, fœtid breath, parotitis, saturnine dyspepsia and rarely icterus. Test meals show lactic acid and no HCl (Sailer and Speese). (*d*) *Anæmia* is very frequent in the "lead cachexia." The hæmoglobin and erythrocytes decrease moderately; the red cells show basophile granules (Grawitz) and quite commonly nucleation, even in mild cases (Cadwalader, 1905). The leukocytes may be increased. (*e*) The *heart and vessels.* The heart is very often hypertrophied, as evidenced by a loud second aortic tone and aortic regurgitation of atheromatous origin; the arteries are often sclerosed, occasionally causing angina pectoris, frequently myocarditis, irregular heart action and perhaps some of the brain symptoms (*v. i.*). The asthma saturninum is probably cardiac or uræmic. (*f*) The *kidneys* are often sclerotic, as a result of the elimination of the metal. It is

said that the change does not begin around the vessels but in the tubules, which are the seat of a necrosing deposit of carbonate of lime. Nephritis, gout and plumbism may co-exist (*v. s.*). (*g*) *Nervous manifestations.* Peripheral degenerative *neuritis* occurs in 7 per cent., usually as a later symptom. About one-tenth of these cases have not had colic. It is characterized by the paralysis of muscles with the same function, by muscular atrophy and cramps, tremor, cutaneous hyperæsthesia, neuralgia, arthralgia (55 per cent.), myalgia and by varying degrees of reaction of degeneration (*v.* MULTIPLE NEURITIS). The musculospiral type, "*wrist drop,*" is the most common and is bilateral; the supinator longus (a flexor) and the small extensor of the thumb escape involvement; tenderness and anæsthesia are rare. Gubler's tumors are swellings of the extensor tendon sheaths and are due to backward dislocation of the carpus or perhaps to a trophic change; they are hard, ovoid, indolent, seldom painful or larger than a pigeon's egg and regress with the paralysis. A second but rare form is the *brachial,* involving the deltoid, biceps, brachialis anticus and supinator longus, rarely the pectorals. A third type is the *Aran-Duchenne,* which is especially observed in tailors (Möbius) and involves the small muscles of the hand, the thenar and hypothenar eminences; it resembles that of chronic anterior poliomyelitis (which indeed has been found in a few cases at autopsy). A fourth type, the *peroneal,* involves the peronei and toe extensors; according to Tanquerel, but 13 per cent. of lead palsies affect the leg. Lastly, rarer types may involve the larynx, vagus and face; generalized paralyses, either slow or rapid like Landry's paralysis, are very rare; primary muscular atrophy may occur, is most often of the Aran-Duchenne type and shows fatty and fibroid muscular changes.

Cerebral forms (*encephalopathia saturnina*) occur in 5 per cent.; they are often preceded by headache, colic and insomnia and are attended by amaurosis, limitation of the visual field, retinitis albuminurica or saturnina (with inflammation, engorgement and possibly ultimate atrophy) and paralysis of the eye muscles. The most frequent type is the *delirious* encephalopathy, which is variable or mobile in character, and sometimes resembles delirium tremens; the next type is the *convulsive* (epilepsia saturnina) which occurs without auræ, is usually generalized, often fatal and is always considered when epilepsy develops in an adult; the third type is the *comatose.* Hysteria frequently occurs with plumbic encephalopathy. Neglected or unrecognized cases may become dements (pseudodementia paralytica saturnina). Deposit of lead in the protoplasm of the brain is the usual cause; sometimes the cerebral symptoms are arteriosclerotic (as hemiplegia) or uræmic.

Lead-poisoning is usually *recognized* (*a*) by the etiology, (*b*) by the lead line, (*c*) by lead in the urine, (*d*) by colic and (*e*) by wrist-drop. In a case of long coma with choked disk, in the Cook County Hospital, no cause was found, until after a second examination, the lead line was seen. The man was a barber, who regularly dyed the

hair of a number of people, and probably absorbed the lead through his hands.

Prognosis.—The acute forms are more favorable. Vascular and renal changes are largely incurable. Atrophic paralyses are serious. The wrist-drop usually regresses, though it is sometimes permanent; 25 per cent. of cases with coma, convulsions and other cerebral symptoms die; after apparent recovery mental degeneration may develop.

Treatment.—(*a*) *Prophylaxis.* A mask should be worn in white-lead factories to avoid inhalation of lead dust; workers are careless and poisoning is frequent despite precautions. Painters and others may often avoid poisoning by carefully cleaning the hands and nails before eating. (*b*) *Lead colic* is treated by hypodermic injections of morphine and atropine to control pain and spasm. Riegel uses nitroglycerine for the supposedly causal spasm of the vessels. Spts. of chloroform (♏xx) with other aromatics may be given by mouth. (*c*) The *constipation* and removal of any lead salts unabsorbed in or excreted by the alimentary mucosa necessitate catharsis. Full doses of castor oil (℥iv or more) with paregoric (℥i) evacuate the bowels and relieve pain and spasm; sodium and magnesium sulphates purge and coincidently precipitate the lead as an insoluble sulphate. (*d*) In order to *eliminate the lead* from the tissues potassium iodide should be given. In acute or severe chronic cases it should be given with care, for it transforms the fixed tissue metal into free metal in the circulation. Nevertheless, *small doses of potassium iodide* (grs. iii to v) should be given, and some writers obtain even better effects from potassium bromide (grs. v to x). Warm baths and pilocarpine (gr. ⅛) promote excretion by diaphoresis and plenty of water stimulates the renal elimination. (*e*) *Iron* (not arsenic) should be given for anæmia. (*f*) For treatment of *neuritis,* see Multiple Neuritis.

ARSENICAL POISONING.

Acute poisoning has been considered under pernicious anæmia.

Chronic Poisoning.—(*a*) *Criminal poisoning* is of great medico-legal importance, as in the unfortunate Maybrick case. (*b*) Poisoning by reason of occupation is uncommon. (*c*) *Therapeutic poisoning* is not common, but may occur in chorea, leukæmia and pernicious anæmia. (*d*) *Most cases are accidental.* The dyes in garments, toys, glazed paper used in kindergartens, wrappers, artificial flowers and wall paper are important sources of intoxications. The action of moulds (penicillium and mucor) may liberate volatile arsenical gases. Contaminated glucose used in beer caused the Manchester epidemic. Arsenic is absorbed by the lungs and alimentary tract and is chiefly eliminated by the kidneys, which may degenerate. Putnam found arsenic in the urine of 30 per cent. of persons who showed no other symptoms of arsenical poisoning, but in this connection it may be remarked that arsenic is widely found in plants, sea water and springs. Smaller amounts are eliminated by the bowel, milk and other secretions.

Symptoms.—The symptoms are (*a*) *gastro-intestinal,* as dry throat, vomiting, purging or colic; the stools may be like "rice water" and contain flecks of mucus; and (*b*) *respiratory,* as dry cough or chronic bronchitis. (*c*) *Emaciation,* weakness, fever and anæmia may occur. (*d*) *Cutaneous* symptoms, as erythema, keratosis, pemphigus, herpes and brown pigmentation and (*e*) *nervous* manifestations, as weak memory, vertigo, headache and multiple neuritis may develop. The neuritis has the same general features as the saturnine form, save that sensory changes are more frequent and the legs are more often affected than the arms; paralysis of the peronei and foot extensors causes the "steppage" gait. The small muscles are less often affected than in plumbic or alcoholic neuritis; paralysis, ataxia, sensory and trophic disturbances and the reaction of degeneration occur as in other neuritides.

Treatment.—Treatment is that of lead poisoning or neuritis.

FOOD POISONING.

Bromatotoxismus (Vaughan) exists in several forms:

I. Meat Poisoning (Kreatoxismus).—Sausages and head cheese are more dangerous than beef or mutton. Sausage poisoning (botulism or allantiasis) was known in Germany a century ago. Van Ermingem has isolated an anaërobic organism, the *Bacillus botulinus;* it contains spores, is flagellated and mobile and grows only on alkaline media at a low temperature (18 to 25° C.); it is a saprophyte and causes symptoms by its toxins only, which are not destroyed by the gastric juice. Paratyphoid organisms were found by Durham (1900). Meat may seem to be normal to the taste and smell, and yet contain ptomaines; their nature is still undetermined, though Cobert considers them promatropin. Canned meat has often caused severe symptoms. In one instance botulism was caused by eating salad (Collatz) and beans (Fischer). The symptoms of botulism appear in twelve to forty-eight hours, with nausea, vomiting and sometimes diarrhœa. Paralysis of the soft palate, larynx and œsophagus rapidly develop and somewhat later paralysis of the bowel, mydriasis, ptosis, disordered vision, adynamia and weak heart action; the sensorium is clear and fever is unusual. The *mortality* ranges between 20 and 50 per cent.; death occurs in four to ten days and the autopsy shows parenchymatous degenerations and ecchymoses, but especially degeneration of the ganglionic cells, as those of the vagus or oculomotorius; if the patient survives, convalescence is established only after months. In the Wellback cases Ballard noted as early symptoms headache, chilliness or rigors and pains in the chest or back.

Symptoms.—The symptoms of other forms of meat poisoning are either those of *toxæmia* (the typhoid type) or *gastro-intestinal* inflammation (cholera type); the typhoidal symptoms, which appear after an incubation of four to six days, are fever, status typhosus and even roseolæ; the choleriform symptoms, which develop in two to twelve

hours, are vomiting, incessant purging, watery stools, clammy skin, collapse and cyanosis. Acute meningitis may be simulated exactly (A. D. Dunn). Mild or rudimentary forms occur in most epidemics.

Treatment.—Treatment is unsatisfactory. *Early gastric lavage, colonic flushings* and *purgation* are indicated; calomel followed by castor oil is the best aperient. Otherwise symptomatic medication, as in cholera or typhoid, is indicated; *champagne, strychnia,* hypodermatoclysis and opium may be given. The resemblance of botulism to the diphtheria toxins has suggested the use of antitoxine; its effects in animals have been remarkable, but to the author's knowledge it has not been used in man.

II. Poisoning by Milk (Galactotoxismus) and *Cheese* (Tyrotoxismus).—The former produces symptoms analogous to those of the infantile affections of the bowel, as vomiting, bloody diarrhœa, fever and collapse. In cheese poisoning Victor C. Vaughan has found tyrotoxicon and other more frequent toxins, which are probably albumoses. Similar ptomaines were found by him, Novy and Perkins in re-frozen ice-cream and custard. In Norway where tyrotoxismus is most prevalent, the colon bacillus has been repeatedly found. Home-made cheese is more dangerous than the factory product. Various forms of toxæmia result; some are cholera-like and others resemble belladonna poisoning. Treatment is that of kreatoxismus.

III. Poisoning by Fish (Ichthyotoxismus) and *Shell Fish* (Mytilotoxismus).—(*a*) Ichthyotoxismus, described by Comby (1827), may be caused by poisonous fish or poisonous glands in certain fish, as the roe or testicles, the muscular parts being harmless; the barbel in Europe and the tetrodon, diodon and fugu in Asia are poisonous (*v.* Beri Beri). The poisoning due to putrefaction is more common. Paralytic symptoms, like curare poisoning, are most frequent. (*b*) Mussel poisoning (mytilotoxismus) may cause dyspeptic symptoms in mild infections and cholera-like symptoms or symptoms of belladonna poisoning in severe forms. Rashes are common. Brieger isolated a mytilotoxin, which is found largely in the liver. The danger lies in placing mussel- or oyster-beds near river mouths, so that they feed on poisonous excreta. In restaurants certain glands near the heads of lobsters, considered toxic, are carefully excised.

IV. Grain Poisoning. (Sitotoxismus).—**1. Ergotism.**—Ergotism, which was described by Thuillier (1830) and carefully studied by Tüczek (1879), results from eating grain mixed with the ergot fungus (claviceps purpurea). Ergotized grain is found largely in bad harvests, resulting from wet springs and hot summers. Epidemics have occurred in Europe, and sporadic cases are not rare. *Acute ergotism,* with digestive symptoms, colic, cyanosis, weak heart and generally lethal outcome is less common than *chronic ergotism,* of which *two forms* exist; the first is the *convulsive* form, attended by paræsthesia, lightning pains, headache, delirium, melancholy, dementia, muscular relaxation and muscular spasms, either as generalized epileptiform attacks or as local contractures (flexion of the arms

and extension of the toes and feet); it is said to result from the alkaloid cornutin. The *second form* is the gangrenous, due to sphacelinic acid and marked by localized gangrene, usually in the phalanges and less often in the tips of the ears and nose. Chronic ergotism is characterized by a number of *symptoms which suggest tabes,* as lightning pains, ataxia of station and movement, and lost knee jerks; and in five autopsies Tüczek and Siemens found degeneration with, later, sclerosis of the posterior columns; degeneration in the anterior cornua, and minute foci of softening or hemorrhage also occur. The clinical course is chronic and death is frequent from convulsions or cachexia.

TREATMENT.—Treatment includes avoidance of diseased grain and symptomatic therapy; opium should be given for convulsions and nitroglycerine for gangrene.

2. Pellagra.—Pellagra or maidism is a chronic nutritional and toxic affection, due to the use of maize which has been improperly stored, or is decayed, unripe or infected with the aspergillus. An alkaloid (pellagazein) is thought to develop. Pellagra is endemic, chiefly in Italy, France, Spain, Hungary and Mexico. It occurs among poor farmers. There are *three stages;* the *first* consists of the *vernal erythema,* which is followed by exfoliation and sometimes by suppuration; dyspepsia and diarrhœa may develop; mild cases may recover or may pass into the *second stage,* in which headache, pain in the back and muscular spasms occur, but the most characteristic symptom is the *ataxic paraplegia;* autopsies reveal a posterolateral sclerosis, sometimes with atrophy of the anterior horns, leptomeningitis and variable brain findings. The *third stage* occurs after repeated attacks and is usually incurable; *cachexia, epilepsy* and *dementia* develop; it is said that 10 to 35 per cent. of Italian insanity is due to pellagra.

The rarer acute cases may resemble typhoid in symptoms and pathology (status typhosus and swelling with ulceration of the intestinal lymph structures), or meningitis (foci of acute meningomyelitis). Other findings at autopsy are atrophy of the digestive mucosa, parenchymatous degeneration and pigmentation, the latter of which is thought to occur from adrenal inflammation.

TREATMENT.—Treatment is prophylactic (proper storage of the grain); change of diet or of locality is indicated and arsenic should be given internally.

3. Lathyrism (lupinosis).—Lathyrism is produced by meal made from vetches, chiefly the *lathyris sativus* and *cicera.* Irving described the disease in India and it has been seen also in Algiers and Italy. Like pellagra, it probably produces a lateral and possibly some posterior sclerosis of the cord, though no autopsies are reported. Clinically there is a *spastic paraplegia* with increase of reflexes and some paræsthesia.

V. Potato Poisoning.—Solanin is contained in potatoes in small amounts (0.06 per cent.) but larger amounts (0.4 per cent.) occur in

potatoes which lie for some time on the ground or sprout in cellars. Fever, chills, general and cardiac weakness, alimentary irritation and jaundice may develop.

SUNSTROKE.

The difference between the two forms, (1) sunstroke, and (2) heat exhaustion, was first recognized by Dowler of New Orleans.

Sunstroke, also known as siriasis, insolation, *coup de soliel* and thermic fever, is due to exposure to the sun. A very frequent factor is alcoholism. There is thought to be a paralysis of the bulbar heat centres, leading to excessive production of heat and deficient elimination of it. The theory of infection has also been advanced.

Symptoms.—Symptoms may develop very abruptly and cause early or even immediate death from asphyxia and paralysis of the heart; this type was long confused with apoplexy. In most cases there is an interval lasting from a few minutes to an hour, in which there are headache, vertigo, visual disturbances or nausea, and perhaps vomiting.

During the coma which develops rapidly, and in which the physician usually sees the patient, the following signs are observed: the face is first flushed, later rather cyanotic, and sometimes stained with petechiæ. The preliminary pupillary dilatation passes into myosis and the skin has a peculiar odor. The pulse is rapid, usually over 100, and is bounding. The rectal temperature ranges from 107 to 112° and respiration is deep, labored and stertorous. Save for the laboring chest, the patient is usually quiet; in about 25 per cent. of the author's cases the patients struggled during treatment by the cold bath and in about 20 per cent. of the severe cases there were epileptiform convulsions. Lewis and Packard (1901) in 92 cases found convulsions and unconsciousness in all severe cases; immobile pupils and absent knee jerks were common. Involuntary evacuations are usual and the thin fæces have a peculiar odor. There is a leukocytosis in about 75 per cent. of the cases. Recovery is frequent; consciousness returns and the fever falls. Despite treatment, the Cheyne-Stokes' breathing may develop, the pulse may grow smaller and more rapid, and death may follow, usually in a little more than a day. The autopsy shows early rigor mortis, fluidity of the blood, great venous hyperæmia, particularly in the brain and lungs, and dilatation of the right and contraction of the left ventricle.

After sunstroke the patient becomes extremely susceptible to hot weather. Epilepsy, multiple neuritis and mental symptoms are among its after-effects. The author saw one case with a temperature of 109° in which the fever fell with hydrotherapy but rose to 104°; in a few days the trouble was recognized as typhoid.

Diagnosis.—The diagnosis is easily made. Practically, there is but one possible cause of confusion, pontine hemorrhage, but the latter never develops nearly as high a temperature and death is almost instantaneous; should the patient survive, paralysis, usually of the crossed type, is obvious.

Heat exhaustion may result from exposure to the sun or to high temperature with humidity; the cases which the author has seen came largely from laundries, basement stores or engine rooms; heat exhaustion occurs also among stokers on steamers. The symptoms are those of a centric vasomotor paralysis. The skin is cool and livid, rather than red, and the rectal temperature is often 3 or 4° subnormal; the pulse is rapid and weak and the breathing is rapid but not labored. In short, the symptoms of collapse are present. Restlessness, anxiety and sometimes delirium are noted, though the sensorium is practically normal in most cases.

Treatment.—In *sunstroke* the chief indication is reduction of the retained heat, by immersing the patient in a *bath gradually cooled* by large pieces of ice. Shock is averted by this means; cerebral congestion is lessened by placing a block of ice under the neck, an ice-bag on the vertex and an ice-bladder over the anterior neck; active friction of the body and limbs with the open hands brings to the surface the blood, which is cooled by the bath and the rubbing with pieces of ice. With this treatment, few more than one-third of the cases should die. The patient should be taken from the bath when his rectal temperature is 102°, for the fall usually continues after cessation of treatment. Laxatives are generally unnecessary, for the bowels move freely. Heart stimulants should be given *pro re nata.* For convulsions, a little chloroform by inhalation and a hypodermic of morphine are indicated. Asphyxia and distention of the right heart are relieved by phlebotomy.

In *heat prostration* the treatment is diametrically the opposite; a warm bath or hot bricks for subnormal registration, diffusive cardiants, as ammonia, camphor and strychnine, saline solutions under the skin and applications of mustard are indicated; cold and venesection are obviously contra-indicated.

SECTION XI.

DISEASES DUE TO ANIMAL PARASITES.

DISEASES CAUSED BY CESTODES.

Tapeworms were known to the Egyptians, Hindus and probably to the Jews. They may cause symptoms by the presence of the mature parasite in the bowels or of the larval form in the viscera.

I. Tænia Solium.—The Tænia solium or pork tapeworm (Linnæus, 1752) exists as the cysticercus cellulosæ in hogs and rarely in dogs, rats or deer, as "measles" (measle or bladder worms). The adult form is peculiar to man. Cysticerci (the larvæ) are found in the intermuscular tissue, particularly of the under surface of the tongue, in the masseters, shoulders, neck and diaphragm. They are easily seen with the naked eye and very easily with a low-power lens, as small opaque white bodies. They are vastly more frequent in Asiatic and German than in American pork. Man is infected by eating raw or partly cooked pork; the cysticerci develop in the upper ileum into the tænia ("band or ribbon-like") solium (referring to its usual solitary appearance, though 2 or 3, and even 20 or 41, have been found). The worm measures 2 to 8 metres in length. Its head (*scolex*) is round, often gray or black in its upper part, is smaller than a pin head, and is attached to the upper ileum; it has 4 sucking disks and 20 to 30 hooklets, arranged in an anterior more numerous row, and a posterior row, whence the name "armed" tapeworm; it has no real mouth. (See figs. 80, 81, 82, 83.) Its neck is thin and not jointed, to which the segments ("*proglottides*") are attached. These number 800 or 900; the segments measure 10 mm. in length by 5 or 6 mm. in width; those near the neck are narrower and shorter. About a yard from the neck they become squarer, and lower down they are smaller again. Each proglottis is hermaphroditic, containing male and female reproductive elements. The uterus is central and vertical, with 7 to 15 lateral branches; only a few of them mature, chiefly those in the lower links; the ova (31 to 36μ) are very numerous in each maturing segment and each one shows a shell and an embryo armed with six hooklets. The tænia reaches its full length of two to four yards in three to four months, when the lower

FIG. 80.

Ova of *T. solium: a*, with yolk; *b*, without yolk, as in mature segments. The hard brown shell is indicated. (Leuckart.)

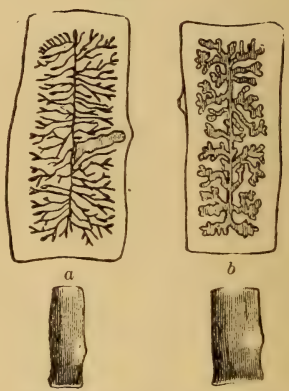
segments are detached and voided; if the worm should break and a yard or so appear in the fæces the segments will not reappear in the fæces until three or four months later. An ovum ingested by a hog or man loses its shell in the stomach and the freed embryo passes into the tissues, where, encysted, it becomes again the cysticercus cellulosæ (or larva).

Symptoms of the Tænia Solium in the Human Intestine (Intestinal Tæniasis).—Many, possibly most, cases present no symptoms; they may first develop after passing segments are found in neurotic subjects. In other cases headache, itching, pupillary inequality, emacia-

FIG. 81.

Head of *T. solium:* × 45.

FIG. 82.



Proglottides of (*a*) ***Tænia saginata*** and (*b*) *T. solium*. natural size, and enlarged three times to show arrangement of uterus. (After Leuckart.)

tion and dyspeptic disturbances arise, as anorexia (or less often increased or ravenous appetite), nausea, abdominal pains, constipation or diarrhœa. Stiles, in an experimental infection on himself, thought that he could feel the parasite move. Vomiting of segments is rare, though several feet of tapeworm have been vomited; in these instances the ova (embryos) may enter the tissues as cysticerci; this is a rare condition in which one individual suffers from both the intestinal and the larval stages. Anæmia is rather uncommon. In children reflex convulsions are possible. The parasite may live for even 10 to 15 years. The *diagnosis* is determined by the segments and ova and the *prognosis* is practically always good.

Treatment.—As to *prophylaxis;* segments of the tapeworm should be burned; meat inspection should be more general than it is, though "interstate" meat is well inspected; pork should always be thoroughly cooked. Hogs obtain ova from human fæces.

1. To cure the disease a light and chiefly *fluid diet* should be

given for two days and then food should be withheld for 12 to 18 hours; this often causes the parasite to release its hold on the mucous membrane; some writers advise that a meal of onions and herring be given just before drugs are administered. 2. A *preliminary cathartic* is given to clean the bowel to allow more direct access of the vermifuge to the parasite and to prevent its regaining its hold when once detached. 3. *Anthelmintic therapy.* Of these *pomegranate* (granatum) ranks first; a decoction is made of 3 ounces of the root with 8 ounces of water; this is reduced to 4 ounces by evaporation; it is taken in divided doses within 3 to 4 hours; its sole active principle is the expensive pelletierine, which is given in doses of 3, 5 or 10 grains and is followed by a purge in an hour (Pelletierinæ tannas). *Male fern* (felix mas) is almost equally effective; it is given as the oleoresina aspidii, or extr. felicis maris æther., 3iss to ijss, may be given in syrup; both are followed by a saline purge in three to four hours. Castor oil is never used, because it promotes absorption of the male fern, which causes toxic symptoms, as icterus or amaurosis; Sidler collected 78 such cases of poisoning with 15 per cent. mortality and lasting blindness in 25 per cent.; Leichtenstern advises (*a*) that more than 10 gm. (3iiss) of the ethereal extract should never be given to adults (3j or less to children); (*b*) that it never be given on an empty stomach and (*c*) that it never be given two days in succession. The drug decomposes easily, whence reliable preparations must be obtained. Combinations may be made by giving with or after the decoction of pomegranate, spts. chloroformi (♏xv) for four doses or croton oil (♏ii) for one dose, but pomegranate and male fern produce the best results. Search should the made for the head of the parasite, without removal of which treatment is unsuccessful. If it is not found another trial should be made after a number of days.

FIG. 83.

a *b* *c*

Eggs of (*a*) *T. saginata;* (*b*) *T. solium;* (*c*) *Dibothriocephalus latus:* × 300. (After Eichhorst.)

℞ Oleoresinæ aspidii .....
Tr. vanillæ ..... āā gtt. xlv.
Acaciæ .....
Sacchari albi ..... āā 3i.
Aquæ ..... 3i.
M. et ft. emulsum.
S.—Take in one dose.

Symptoms of the cysticercus cellulosæ (somatic tæniasis). These result when ova of the tænia solium are taken into the stomach; ova may reach the stomach from the intestines by antiperistaltic movements during vomiting or indirectly by handling worms voided from the bowels. Cysticerci in the hog produce few symptoms. In man, symptoms depend largely on the number and localization of the

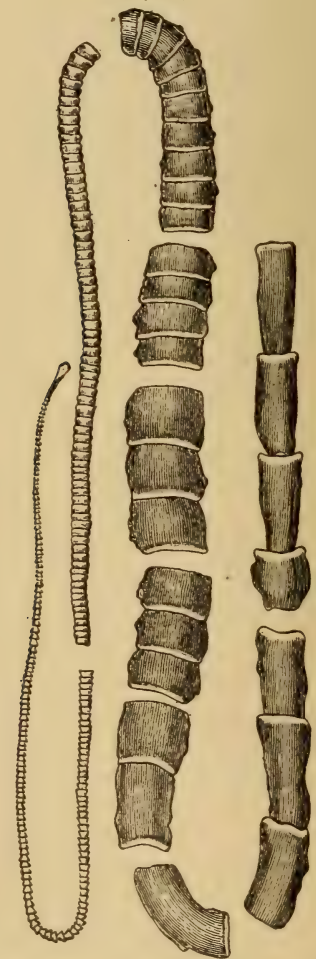
larvæ. (*a*) In *the muscles and skin,* a few cysticerci cause no symptoms; numerous cysticerci cause pain, tenderness and difficult movement. Diagnosis is possible only on excision of the subcutaneous nodules. In Stiles' collection of 155 cases the localization was muscular in 20 per cent. and cutaneous in 3 per cent. (*b*) If they are in the *brain* (in 75 per cent. of Stiles' series) symptoms depend on their location; they are often absent; if the location is in the cortex, Jacksonian epilepsy is frequent, and if in the medulla, diabetes may develop. (*c*) *Other localizations.* They may be found clinically in the eye (v. Graefe) and at autopsy in the lungs, kidneys or liver. There is no *treatment* except surgery.

II. Tænia Saginata (Mediocanellata).—This form (Goeze, 1782) is the most frequent of large tapeworms in America and Europe.

FIG. 84.

Head of *Tænia saginata.* (Eichhorst.)

FIG. 85.



Tænia saginata: natural size. (After Leuckart.)

It is the "fat tapeworm," the "unarmed" or beef tapeworm. Its *cysticercus bovis* is found in masseters of beef nearly seven times as frequently as in other muscles or in the heart or brain. They are not so readily seen with the naked eye as the cysticerci in pork. Ingestion of rare beef containing cysticerci produces the tænia saginata in man, in whom alone the adult worm is found.

The intestinal parasite differs from the T. solium in the following points: (*a*) its *head* is larger, measuring 2 mm. and is square. It has no hooklets but four sucking disks, which are larger, more forward and are surrounded by pigment. (See figs. 82, 83, 84 and 85.) (*b*) Its neck is much shorter. (*c*) Its *proglottides* are longer (16 to 22 mm.) and broader (5 to 10 mm.); even 1,000 segments have been found. (*d*) The *uterus* contains 15 to 35 branches, which are less dendritic than pronged or forked; in diagnosis the uterus is more important than the ova or size of the segments, which are hard for the practitioner to distinguish (Stiles). (*e*) The *ova* measure 30–40 by 20–30μ. (*f*) The *parasite* is larger and longer, measuring 4 to 10 meters. Its symptoms and treatment are those of the tænia solium. Ingestion of the ova or segments from human fæces produces the cysticercus mediocanellata, which is common in beef but extremely rare in man.

III. Other Tæniæ Occurring In Man.—The *Dibothriocephalus latus* (Linnæus, 1782), or the Russian tapeworm, is found chiefly in Russia, Poland, Norway, Sweden, Switzerland and Japan. It is rarely found in this country unless imported (30 cases reported, Stiles). Its cysticerci are found in the muscles and peritoneum of the pike, trout, salmon and perch and in man's intestines develop into the dibothriocephalus. Its head is flat or ovoid, possesses no hooklets and attaches itself to the intestines by two lateral grooves (suckers). Its neck is long and its segments broad, square and may number 3,000 to 4,200. It is long, measuring 8 to 10 yards. It may produce a profound anæmia which in severe cases closely resembles the pernicious type in its general symptoms and blood findings. (See figs. 83, 86.)

The *Tænia elliptica* or cucumerina (Dipylidium caninum) is a form, the larvæ of which develop in the lice of dogs and the adult form exists in the intestines of dogs, sometimes in those of cats and rarely in man. (See fig. 87.)

The *Tænia nana* (Hymenolepsis nana) is the dwarf tapeworm. It measures but ⅕ to 2 inches in length. Its cysticercus stage is in the intestinal wall of the rat and its adult stage in the rat's intestine. Stiles decided that it is much more frequent in man than previous accounts would indicate and in some places is the most common type. Its head contains four suckers and one row of hooklets. It may occur singly or in myriads. Aspidium alone is of therapeutic benefit. (See figs. 88 and 89.)

Tænia Echinococcus (Echinococcus Disease) (Zeder, 1803).—We may first consider the *adult parasite as inhabiting the intestine of the dog*

chiefly and rarely of the wolf, fox or jackal. The head is small and is provided with four suckers, and from 30 to 50 hooklets arranged in a double row. The segments number only three or four, and only

FIG. 86.

Proglottis of *Dibothriocephalus latus: natural size,* and enlarged three times. (After Eichhorst.)

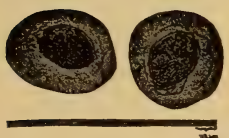
FIG. 87.

Tænia cucumerina: natural size. (After Leuckart.)

FIG. 88.

Hymenolepsis nana: × 12 (After Leuckart.)

FIG. 89.



Ova of *Hymenolepsis nana.* (Smith.)

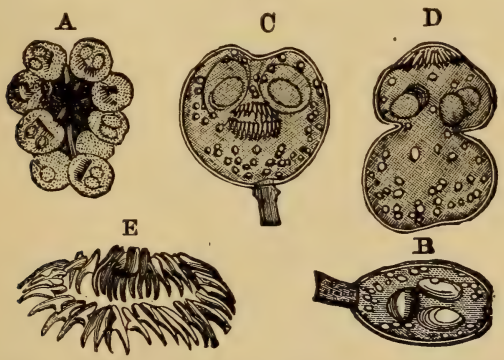
the end one matures; it measures but 0.6 by 2 mm. but contains even 5,000 ova. The worm is small, measuring but four to five mm. in length, is very delicate and white, so that it readily escapes detection. When the ovum reaches the stomach of another animal, the hog and

ox chiefly, and less often the horse, sheep or man, it loses its shell and migrates to the various organs (*v.* SYMPTOMS). The *geography* of echinococcus disease and mode of infection is as follows: In Iceland, where dogs and men live together, the disease in man is common, every seventh person becoming infected. In Australia both men and sheep are often infected. In Europe the malady is much more common than in America, where the reported cases in 1901 numbered 241 (Lyon); most of these were foreigners. The Icelanders brought the disease to Manitoba in 1874.

Infection occurs by licking of the hands by the dog, the tongue and anus of which often come in contact. Infection by contaminated drinking water or vegetables is less common.

General Symptoms and Pathology.—The small embryo, freed of its shell, penetrates the intestinal wall and wanders in various directions, into the muscles or peritoneum or into the radicles of the portal vein or cava, so that almost any tissue or organ may be infected, and the embryo merely stops when it reaches vessels too small for it. Once lodged, its hooklets disappear and a cyst develops, which is small at first but gradually reaches large or enormous proportions; there are records of cysts containing 30 and 70 pints of fluid. Its layers consist of an inner endocyst which is granular and an outer finely lamellated structureless capsule; a fibrous layer develops outside from reaction of the tissues to the foreign body. After a while a number of "daughter" cysts arise from the endocyst; these are first "buds"

FIG. 90.



Human echinococci. (From Finlayson, after Davaine.)

A, a group of echinococci, still adhering to the germinal membrane by their pedicles. ×40.
B, an echinococcus with head invaginated in the body. ×107.
C, the same compressed, showing the suckers and hooks of the retracted head.
D, echinococcus with head protruded.
E, crown of hooks, showing the two circles. ×350.

and then miniatures of the original mother cyst; from these daughter cysts, which number about a dozen, "granddaughter" cysts arise by an identical process. Allen in one case found 8,000 daughter cysts. Finally from the endocyst of the maternal and daughter cysts there develop "brood capsules," in which small buds arise, which gradually

become scolices (the heads of which will be intestinal tæniæ in the animal which ingests them). Some cysts do not develop scolices and are therefore called sterile. In animals the daughter cysts may grow outward (exogenous cysts). The well-grown cyst contains fluid (*v.* Liver and Lung Echinococcus); it is clear and neutral, has a specific gravity of 1005–10–15 and contains succinic acid, inosite (possibly sugar) and much sodium chloride but no albumin, unless the parasite dies and cystic inflammation develops. Then the fluid may become cloudy, buttery, gelatinous, purulent, brownish or grumous; the cysts disappear as well as the membrane, though the hooklets of the scolices remain a long time. When the parasites are alive a

Fig. 91.

Hooks from Tænia echinococcus. × 350. (Corti.)

toxin seems to be present, for collapse often develops when the cyst ruptures or is punctured (see Lung Echinococcus).

The parasite is said to live for years, in one instance it survived for 20 years; after a variable time, unless suppuration intervenes, the cysts become cheesy and perhaps calcified or ossified.

The parasite may rupture into the serous sacs, bloodvessels (causing embolism), into the air passages, urinary tracts or externally (see Echinococcus of the Lung, Liver and Kidney); in 50 per cent. it is fatal within five years.

Visceral Localization.—In 1,912 cases (the total reported by Davaine, Finsen, Mosler, Neisser and Cobbold) the liver was involved in 51 per cent., the genito-urinary organs in 10 per cent., the intestinal canal in 9 per cent., the lungs or pleura in 9 per cent., the brain or cord in 7 per cent., the bones in 3 per cent., the heart and vessels in 3 per cent., and other organs in 8 per cent. Vegas and Cranwell (1902) reported 970 Argentine cases of which 64 per cent. occurred in the liver and 7 per cent. in the lungs. In statistics from single observers, the percentage of cases with localization in the liver often runs higher (69 to 72 per cent.). Thomas, in 809 Australian cases of single hydatid cyst, found the lungs involved in 16.5 per cent. Hooklets have been obtained by lumbar puncture (Jacobs). There are reported 142 cases of renal echinococcus, 78 cases with localization in the bones and 23 in the pelvic bones (Baradulin, 1906). Grulee in 1905 found 55 cases with cardiac or pericardial localization.

Echinococcus cyst of the thyroid has been reported 23 times (Ehrhart, 1905).

There is an unusual form of echinococcus, the *multilocular form,* probably due to a separate worm, the clinical features of which are described under hydatid disease of the liver, to which it is almost exclusively confined. Virchow first described it in 1856. It occurs in Bavaria, Würtemburg, Switzerland, Austria, Austrian Tyrol and Russia. Few imported cases have occurred in America. It is unknown where the ordinary form is most common. It is rare, few more than 100 cases being recorded. Sixty per cent. occur in males between 20 and 50 years of age. The ova are spheroidal and measure 100 to 170 micro-millimeters. The great difference from the ordinary form is the outward (exogenous) growth of the cysts. The irregular and diffuse cystic proliferation is an unfavorable feature. The cysts contain a gelatinous material (which sometimes causes confusion with colloid cancer), surrounded by intersecting fibrous strands, which gives the liver a porous appearance, like a sponge or cheese with air cavities. In some vesicles there is cheesy material, with some resemblance to the granulomata. The vesicles sometimes suppurate.

Echinococcus cysts which cause symptoms belong largely to surgery, though, as described elsewhere, spontaneous recovery is not infrequent.

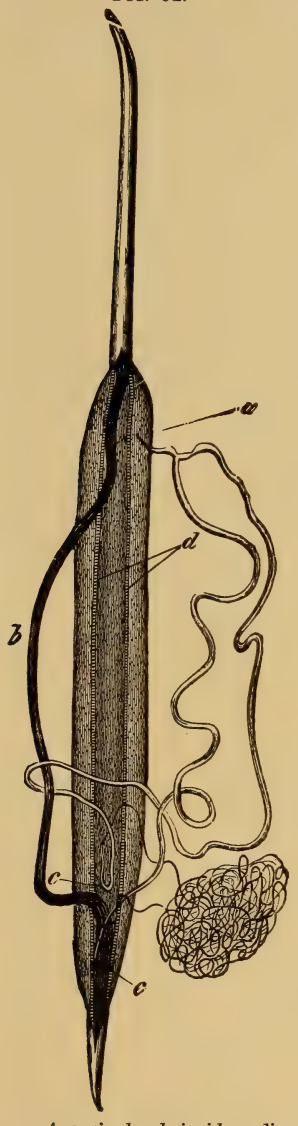
DISEASES CAUSED BY NEMATODES.

I. The Ascaris Lumbricoides.—This is the most common intestinal parasite in man. It was described by Linnæus, 1758. It occurs oftenest in children and with relative frequency in the insane. Unlike the cestodes, no intermediate host is required. The female measures 7 to 12 and the male 4 to 8 inches in length; it is smooth, pointed at both ends and has transverse rings and four longitudinal ridges, a white one dorsally and ventrally and two brownish ones laterally. (See fig. 92.) The ova measure 0.075 x 0.058 mm., are oval and reddish and have a thick capsule. It is said that 60,000,000 eggs may develop in a single female. It lives in the upper small intestine; its mode of infection is obscure. Though they usually occur singly or in pairs, they may exceptionally develop in such numbers that by intertwining, they may obstruct the intestine. The ascaris may rarely rupture through an intestinal ulcer or perforate the sound gut. It has passed through a perforated appendix. With or without vomiting, ascarides may pass from the stomach into the œsophagus, nose, bronchi, lungs or middle ear, causing asphyxia, gangrene of the lung, etc. They may migrate into the common or hepatic duct.

Symptoms are not usually pronounced, though anorexia, salivation, offensive breath, constipation, colic and diarrhœa occur in some cases. Malnutrition is sometimes marked. The parasite secretes an irritating substance which is often noted to emanate from it. It is suggested that this secretion, if absorbed, may cause constitutional symptoms (Peiper).

Such manifestations as irritability, grinding the teeth, muscular twitchings and convulsions may develop in nervous children; they are said even to resemble meningitis. The author always doubted the existence of the *typholumbricosis* of Chaffard, Marie and Tauchon, until in one adult case, in which there were fever and nervous toxæmia, with otherwise negative findings, instantaneous subsidence of all symptoms occurred when the parasites were removed; this strongly suggested a relation between the fever and other symptoms and the lumbricosis.

FIG. 92.



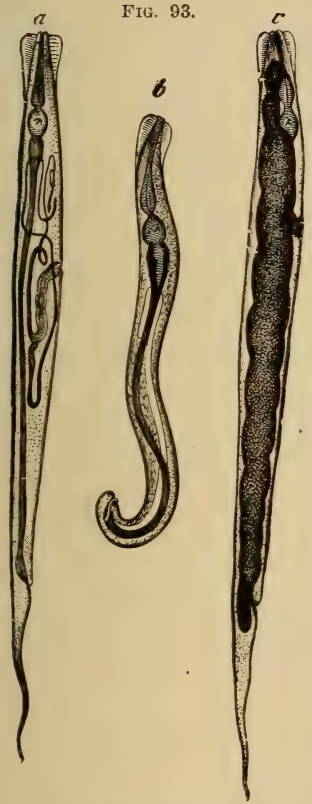
Ascaris lumbricoides, dissected and walls thrown back; *a*, genital orifice; *b*, intestine; *c*, oviducts; *d*, longitudinal band; *e*, ovaries. (Heller.)

Treatment.—After a fast of one-half to one day, *santonin* should be given in doses of gr. ss–j, for a child, and gr. ij, iij or v, for an adult; it should be followed by a saline cathartic; if unsuccessful it may be repeated on two or three successive days, though toxic symptoms sometimes develop, as xanthopsia (yellow vision), urticaria (which may result from the parasite itself), vomiting or convulsions. The *fluidextractum spigeliæ* (3j) may be given with or without the santonin or calomel. These remedies do not kill the parasite, but merely render it uncomfortable.

II. Oxyuris Vermicularis (Seat Worm, Pin or Thread Worm).—Children are more often affected than adults, and women more often than men. They are relatively frequent in the insane. The female in length measures 10 and the male 4 mm. (See figs. 93, 94 and 95.) Ingested ova develop in the small intestine, where the worms mature and cohabit; afterward they exist chiefly in the lower colon and rectum. The eggs remain in the mother until the worm is expelled, though she occasionally "aborts" in the bowel. Infection occurs in those who are the most uncleanly, as in children and the insane. Infection may occur from water and green vegetables, and reinfection is not uncommon from scratching the anus during sleep and thus crushing the parasite; Eichhorst frequently found ova in the minute fæcal particles about the anus and Zenker demonstrated repeatedly their presence under the finger nails. As the parasites migrate at night,

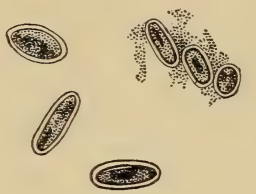
the symptoms are greatest at that time; they may be found on the bed sheet. The anal itching and rectal irritation are often excessive; periproctal suppuration may be excited. If any vaginal discharge moistens the perineum (and only then) they can wander into the

FIG. 93.



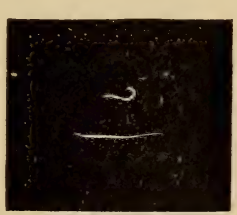
Oxyuris vermicularis, magnified: *a*, young female; *b*, male; *c*, mature female, full of eggs. (Payne.)

FIG. 94.



Eggs of *Oxyuris vermicularis:* ×275. (After Eichhorst.)

FIG. 95.



Oxyuris vermicularis: natural size. (Eichhorst.)

vagina, causing irritation, sexual stimulation or masturbation. Disturbance of the sleep and appetite, nervous symptoms and anæmia may result. Diarrhœa is due to large numbers of parasites.

Treatment.—Santonin may be given in obstinate cases, but local measures are usually sufficient, as high enemata containing small proportions of carbolic acid, quassia and turpentine. They should be repeated daily for two weeks. Itching is palliated by application of ½ per cent. carbolic salve to or within the anus.

III. Trichina (Trichinella) Spiralis; Trichinosis.—Though Tiedemann (1822), Hilton (1832) and Paget (1835) saw the parasite, Owen (1835) first fully described it. Leidy saw it in the hog in 1847. Zenker (1855–60) first described its pathological and clinical bearings; he found 4 cases in 136 autopsies.

Swine are infected in a manner which is not wholly clear, but infection most probably results from eating offal. The disease is rare in some countries, as France, and more common in others, as Germany. The hog is more frequently diseased in America than in Germany (a ratio of 1 to 17 (or 48) in the former and but 1 to 18,000 in the latter). From the clinical and etiological standpoint, the (*a*) *muscular or larval stage in hogs* should be considered first. The small encapsulated, "hair-like" (trichina) worm lies in the abdominal, diaphragmatic, psoas, laryngeal and other muscles. They live for a long while, even twenty to twenty-five years, without apparently causing any symptoms; calcification rarely occurs in swine. *Man is infected* by eating smoked, "cured" or insufficiently cooked pork; animals may be accidentally or experimentally inoculated, especially the rat, guinea-pig, rabbit, less often the cat and rarely the dog. In much of the literature relating to its alleged presence in fish or worms, the trichina has been confused with other parasites. (*b*) The *adult or intestinal stage in man* is the next phase. When it is eaten by man, the small capsules (larval form in swine) are digested and the worms liberated; in the small intestine they mature (the females measure 3 to 4 mm., and the males are half as long) and the females are fecundated in two to three days. The female trichinæ produce one to two thousand embryos at once or possibly in succession, which stage requires another week (from fecundation of the female to birth of the embryos). At the end of the second week the embryos commence to migrate; they enter the lymph vessels and then the veins, by which they reach the voluntary muscles; some writers, as Graham, maintain that there is a primary direct entrance into the venous system. They lodge between the muscle fibers, enter them and attain (*c*) *the mature larval form,* which is identical with the larval stage (*a*) in hogs, and differing only in that man is their host. They measure 0.5 to 1 mm. in length. As a result of tissue reaction against the foreign bodies, a capsule develops in about six weeks, which surrounds one or more parasites. The capsules are at first transparent, but grow more opaque and, after months, calcify and later the trichinæ calcify also.

Frequency.—Though trichinæ are apparently about 40 times as frequent in American as in German swine, the disease is more common in North Germany where raw sausage, Westphalian ham and smoked ham are freely eaten. Fränkel denies that German trichinosis results from American pork. Williams of Buffalo, in 505 autopsies, found trichinosis in 5 per cent. Müller of Dresden found it in 0.98 per cent. of 1,939 postmortem examinations. Other statistics usually show lower figures than Müller's, and like the above, are taken from accidental postmortem findings. The disease occurs sporadically or epidemically. Perhaps a thousand cases have occurred in America. In Germany the epidemic at Hedersleben numbered 337, and that at Emmersleben 250 cases.

Symptoms.—Symptoms may be absent or slight, particularly in

sporadic cases, and with moderately severe infection, as evidenced by accidental autopsy finding of encapsulated parasites.

In clear cases the symptoms are divisible into *two stages;* (*a*) the preliminary or *gastro-intestinal* stage, which begins in two to three days after eating the diseased meat; the symptoms are not constant either in sporadic or severe epidemic cases; they are anorexia, nausea, vomiting, colic and sometimes diarrhœa, which may resemble the evacuations of cholera. (*b*) The *invasion symptoms* begin in one or two weeks and correspond to the parasitic migration. (i) *Fever* is usual, but its onset seldom begins with rigors. It results from the parasites and probably from some toxin which they elaborate; it may rise to 102, 104 or 106° and its course is remittent and intermittent. Symptoms attendant on any fever may be present, as headache, febrile urine or quickened pulse. In severe cases, as in Zenker's, typhoid may be suggested by delirium, epistaxis, dry tongue, status typhosus, diazo reaction (in 80 per cent.), bronchitis, albuminuria and kindred toxæmic symptoms. In two cases typhoid and trichinosis co-existed (McCrae). (ii) *Acute diffuse myositis* is always most suggestive. Its intensity varies from mild forms of "muscular rheumatism" to the most severe and characteristic involvement; there are pain, tenderness, swelling, flexion to relieve muscular tension, and *œdema* in the face and other locations. The worms most curiously select only the striated muscles, so that the œdema observed in the abdomen and thighs, for example, never invades the scrotum or vulva. The eyelids are often affected greatly and early. Invasion of the eye muscles causes pain on moving the eyes; pain during mastication, phonation and deglutition mark penetration of the masseteric, laryngeal and pharyngeal muscles by the parasite; dyspnœa results from involvement of the diaphragm and intercostal muscles. The tendon reflexes may disappear. (*c*) *The blood* presents two striking features: first, the *leukocytosis* (15,000 to 30,000) and second, the *eosinophilia* (T. R. Brown); the eosinophiles, normally 0.3 to 4 per cent. of the leukocytes, rise as high as 50 or 68 per cent. and though they increase in the other parasitic diseases, this rise is most peculiar if not absolutely pathognomonic. The eosinophilia is greatest at the time when the trichinæ enter the muscles. (See Plate XVI, Fig. 5.) On the 24th day a second œdema appears. (*d*) *Profuse sweats,* polyuria, paræsthesia, urticaria and anæmia are inconstant manifestations.

Diagnosis.—This is based on (*a*) the possibility of infection, as by eating poorly cooked pork, (*b*) the discovery of trichinæ in the uneaten portions of suspected meat, (*c*) the detection by removal of small slivers of the biceps or pectorals for microscopic examination, especially the parts near the muscular insertion, (*d*) the myositis with œdema, (*e*) signs of insufficiency, *e. g.,* in the ocular or diaphragmatic muscles and (*f*) leukocytosis with eosinophilia.

Prognosis.—This depends on the number of parasites ingested. Choleraic and typhoidal symptoms, dyspnœa and pneumonia are most ominous. In moderately severe early diarrhœa it is favorable.

Children die less often than adults. Convalescence is prompt or tardy as the infection is light or severe. According to Stiles, the German mortality averages 5.6 per cent. In the epidemic at Weimar there were no fatalities in 108 cases. In the epidemic at Hedersleben 30 per cent. died. Epidemics have occurred where the mortality was as high as 70 to 100 per cent.

Treatment.—(*a*) *Prevention* comprises the feeding of hogs with clean food, government inspection of meat, and thorough cooking. (*b*) In the *preliminary stage,* active and repeated purgation should be given, for moderate and early spontaneous diarrhœa is prognostically favorable; calomel should be followed by salts and castor oil; male fern, santonin (*v. s.*), thymol gr. v, t. i. d., benzene ♏ij to v, t. i. d. and oil of turpentine ♏v to x are recommended; glycerine is now discarded. (*c*) In the stage of *muscular invasion,* the fever is treated as in other infections, the myositis by local applications of ice and by narcotics, and the general nutrition by food and stimulants.

Fig. 96.

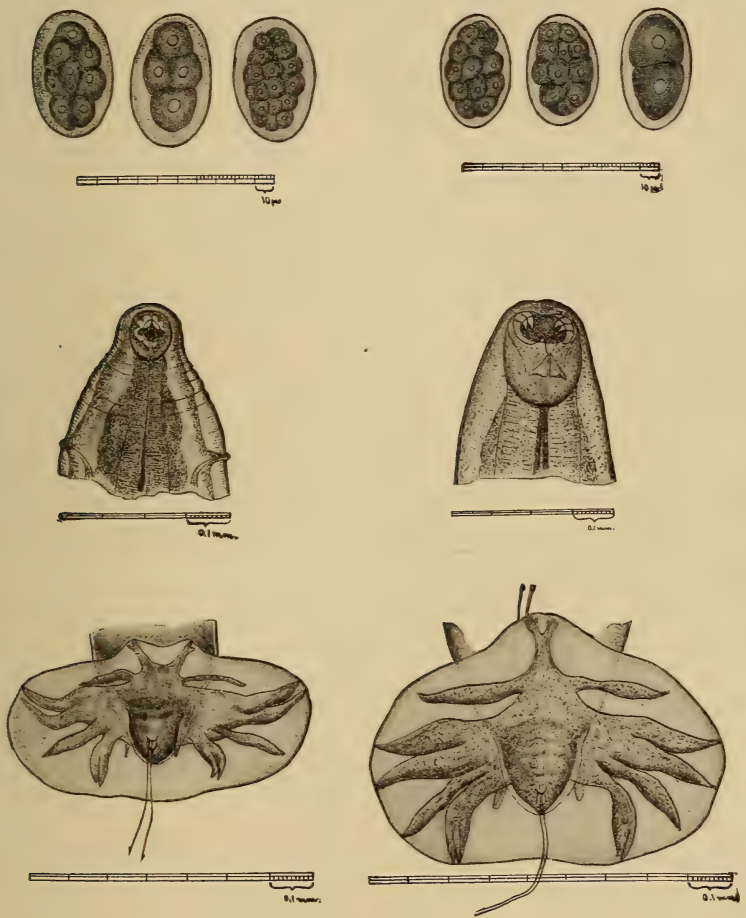
Uncinaria duodenalis magnified: *a*, female; *b*, male. (Bristow.)

IV. Ankylostoma (Uncinaria).—The uncinaria duodenalis (ankylostoma duodenale) exists in two forms, that of the old and that of the new world. Ankylostomiasis was first well described by Piso (1648) in Brazil and the parasite was recognized in Milan by Dubini (1838). It occurs chiefly in the tropics and sub-tropics; according to Thornton, it is the most dangerous of all tropical diseases. In Egyptian chlorosis it was found by Griesinger, and is said to be present in nearly every autopsy made there. It is epidemic in Italy ("bricklayers' anæmia"). In India it is found in even 80 per cent. of the healthy coolies and in 300 autopsies in Assam it was present 299 times. Stiles (1902) described the new-world type, caused by the Uncinaria (or Necator) Americana. Thirty per cent. of Porto Rican deaths result from uncinariasis, and Ashford and King state that 90 per cent. of the rural and 50 per cent. of the urban population have the disease; during six months in 1905, 18,865 cases were treated in Porto Rico, of which 33 per cent. died. It prevails in our southern states. Capps in 1903 reported one case of the European type and collected 50 other American cases. More than twenty years ago, Belfield recognized the parasite in cats.

In the Westphalian mines, Tenholt himself treated over 4,000 cases. It was the cause of the "tunnel anæmia," which was observed in building the St. Gothard tunnel. It is disseminated by Italian and Polish workmen. Infection occurs from ingestion of the larvæ

and not of the ova. Infection comes from the food, drinking water and earth which are infected by dejections from persons suffering from the disease. Loos has proven that it may enter by the skin, producing the "ground itch" which is common in the Southern States. The exact role of the uncinaria in clay-eaters is not yet

FIG. 97.



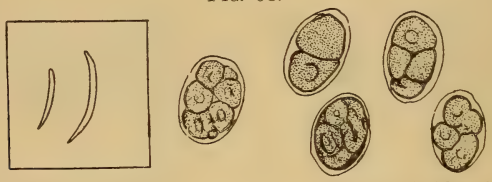
Above: to the left, three ova of *Uncinaria Americana* in different stages of segmentation, from 66 μ to 68 μ in long diameter; to the right, three ova of *Uncinaria Americana* in varying stages of segmentation, long diameter from 58 μ to 62 μ.

Middle: to the left, the head of *Uncinaria Americana* (dorsal view), exhibiting the smaller size of the head and more tapering anterior extremity of the worm in comparison with *U. duodenalis,* and its buccal armature consisting of lips; to the right, the head of *Uncinaria duodenalis* (dorsal view), drawn to same scale, showing its greater size and the hook armature of the buccal border.

Below: to the left the caudal border of male *Uncinaria Americana* (dorsal view, dorsal lobe of bursa thrown forward), showing comparative size of the worm in relation to that of *U. duodenalis,* partly divided dorsal lobe and suggestion of a ventral lobe, the dorsal ray divided nearly to base, each of its divisions having a bipartite tip, showing also the tip of tail within bursa, the long barbed sexual spicules, and the arrangement of the rays of the bursa; to the right, caudal bursa of male *U. duodenalis,* showing larger size of bursa, entire dorsal lobe, absence of ventral lobe, division of dorsal ray limited to distal third and tripartite tips of each division, with other features in general similar to *U. Americana.* (Smith.)

determined; the parasite certainly exists in some cases, though it is claimed that the dirt eaters instinctively eat clay to remove the parasite. The male is 8 to 10 and the female 10 to 18 mm. in length; the mouth has hooks by which it adheres to the mucosa of the duodenum. (See figs. 96, 97 and 98.) The ova measure 30 to 60

FIG. 98.



Uncinaria duodenalis, with eggs. (After Blickhan.)

micromillimeters; they are oval, are covered with a translucent shell and differ from the flat oxyuris ova. They show 1 to 6 yolk segments and may number over four millions. The larvæ develop in moist earth, whence they infect the water or the fingers or enter through the skin.

Symptoms.—Symptoms may be absent or of a mildly dyspeptic character. In pronounced infections (*a*) an *anæmia* develops which is first of the secondary type and later of the progressive pernicious type; anæmia results not only from sucking of blood by parasites, but from hæmolysis due to some toxin which they elaborate (in their salivary glands); normoblasts are much more common than megaloblasts. Poikilocytosis is less marked than in the usual pernicious type, and the eosinophiles are increased, and sometimes reach 30 per cent. of the total number of leukocytes. (*b*) *Gastrointestinal symptoms* are usual, as ink-like patches on the tongue, diarrhœa and colic; epigastric pain and tenderness are the most constant and suggestive symptoms. In Herrick's Panama case (reported by Capps) which the author saw in the County Hospital, there was great nausea, pain and exquisite abdominal tenderness; the stools contained blood, eosinophile cells, Charcot-Leyden crystals and ova. Stiles suggests a ready test for blood, by which parts of the stool are placed upon blotting paper; after an hour or so a rusty stain develops about the stool. Gastro-intestinal catarrh and small ecchymoses are found at autopsy. (*c*) *Nutrition suffers;* as in pernicious anæmia, œdema develops. Clay-eaters are emaciated and apathetic; debility is frequent. In young children puberty is retarded; development is poor and the abdomen is greatly distended. Cardiac atrophy and dilatation are common. The prognosis is grave, though less serious in the new world than in the old world type.

Treatment.—1. PREVENTION.—Drinking water should be boiled and the hands should be cleansed before eating. The stools should be received in water-tight closets and disinfected; careless defecation in mines has caused wide dissemination of the disease. Mines should be disinfected with the milk of lime.

2. Medication.—*Thymol* is the most efficacious remedy. After a light diet, 3ss should be given, and this should be repeated in two hours and followed by a saline purge two hours afterward. As toxic symptoms (nervous excitation or smoky urine) may develop, no solvents of thymol should be given, as alcohol, chloroform, ether or castor oil. This treatment should be repeated once a week, as long as ova or Charcot-Leyden crystals appear in the stools. The most severe cases do not respond to any treatment. *Male fern* is considered the best remedy by Tenholt and other Westphalian writers.

V. Filaria.—Demarquai (1863) first found the embryos of the filaria; Wücherer (1866) found them in the urine and Lewis (1872) in the blood.

1. **Filaria Nocturna.**—Its distribution is largely tropical and subtropical. It occurs in 30 per cent. of the inhabitants of the Samoan and Friendly Islands. In the United States it has been found in the South and also in New York, Illinois and Pennsylvania. (*a*) *Infection of man* occurs by mosquito bites or possibly by water which the mosquito contaminates with its eggs. In the mosquito, the embryos change to the young adult form. (*b*) The *next stage* is the presence of *adult filaria in the lymph channels,* where they cause lymph stasis and lymphangitis. The female filaria measures 155 x 0.7 mm. and the male 83 x 0.4 mm. (*c*) The *embryos enter the blood current* from their birth place, the lymph channels; they are very numerous, measure ⅛ inch in length and have the same diameter as a red blood cell. Under a low-power lens their active movements are easily seen. They appear in the blood only at night, usually near midnight, and during the day retire to internal organs, as the lungs, but if the patient sleeps by day, they appear in the day time only.

Symptoms are absent in the majority of cases of human and animal infection and are due to the parent worm, not to the embryos. (*a*) *Chyluria* or hæmatochyluria is due to plugging by the worm of the lymphatic vessels in the peritoneum, pelvis, vesical and perirenal tissues. Lymph stasis, ectasia and lymphangitis result, with intermittent rupture into some part of the urinary tract, and the passage of milky, chylous urine, containing molecular albumin and fat. The amount of the urine varies; it may be normal or increased. There is usually also some blood. If the blood clots, vesical disturbance results. The embryos are often found in the blood and sometimes in the urine; there is also a non-filarial chyluria (page 774). The affection may last for years without impairment of health. The very rare rupture into the peritoneum causes chylous ascites. (*b*) The *lymph scrotum* is due to lymphatic obstruction by the adult worm. The scrotum may weigh 50 to 200 pounds. Rupture into the tunica vaginalis causes chylocele. The labia may be similarly involved. The embryos are often missing from the blood, being apparently confined to the local involvement by the lymphangitic thickening, whence the difficulty in stating the relative percentage of parasitic and non-parasitic cases of lymph scrotum and elephantiasis. (*c*) *Elephantia-*

sis usually develops in the legs, scrotum and labia, less often in the arms, breasts or trunk. In forms (*b*) and (*c*) there occur intermittent exacerbations with fever and nervous disturbance, which end with critical sweats.

2. Other Forms of Filaria.—The exact status of the *filaria diurna* is not known; *f. perstans,* which causes pustular eruptions (craw-craw); *f. loa* (the adult stage of filaria perstans) which inhabits the conjunctiva; *f. labialis;* and *f. oris* and *bronchialis* are other forms.

Treatment.—(*a*) For *prevention,* mosquito bites and infected water should be avoided. (*b*) No parasiticide is known. (*c*) *Chyluria* is treated expectantly; a dry and fat-free diet is recommended but it is difficult to understand how it can influence lymph stasis. (*d*) *Lymph scrotum* and *elephantiasis* may necessitate castration, amputation or less radical measures to eradicate the obstructing parasite.

VI. Dracunculus (Filaria) Medinensis.—This is found on the West African coast, the Gold Coast, in Abyssinia and South Egypt; on the Caspian Sea and Persian Gulf; in the East Indies, parts of India and South America. Guinea-worm disease (drachontiasis) developed in a person who had always lived in Philadelphia. Man is infected by a small water flea (cyclops) which contaminates the water with the larvæ (embryos). These embryos, measuring 0.6 mm., enter the stomach, bowel and probably the mesentery, where they copulate; the male, of which little is known, is either discharged from the bowel or dies in the mesentery. The female is cylindrical in form, measures 50 to 80 x 2 mm., has a triangular mouth, eight papillæ and a blunt tail; its uterus occupies almost its entire length. The female, which is usually solitary, wanders downward in the subcutaneous and intermuscular tissues to the thighs, legs and in 66 per cent. of cases reaches the feet where, near its head, vesicles, abscesses and ulcers develop. Its downward course occupies a year, during which it may be felt subcutaneously. When the skin breaks the parasite discharges her embryos and then leaves the body, to die.

Treatment.—Treatment by excision, or by 1 to 1,000 bichloride injections, seems most rational. If the worm is ruptured in efforts to extract it, high temperature, alarming nervous symptoms and sepsis are said to result from migration of the embryos which are liberated in the tissues.

VII. The Trichocephalus Dispar or Whip Worm.—This nematode is found most often in southern Italy and France and but seldom in this country; one case occurred in the Cook County Hospital. It is also called trichuris trichura. It requires no intermediate host, enters with the food and drink and is found in the cæcum and large gut. The male is slightly shorter than the female, which is 4 to 5 cm. in length. The anterior portion of the worm is thin; the posterior two-fifths is thick, in the male round and in the female pointed. It bores into the mucosa and is nourished by the blood and not by the alimentary contents. The eggs measure 0.05 mm., are oval and are marked by a button-like projection. It causes few symptoms; occa-

sionally there is severe abdominal pain. Three fatal cases are recorded. Male fern and thymol internally and benzene 3j to Oij of water as an enema are recommended. (See figs. 99 and 100.)

VIII. The Strongyloides Intestinalis.—This includes a number of nematodes, as the anguillula intestinalis or stercoralis, rhabdonema, etc. They are found in the endemic diarrhœa which occurs in Cochin

FIG. 99.

Eggs of *Trichuris trichura:* × 275. (After Eichhorst.)

FIG. 100.

Trichuris trichura, male and female: natural size. (Eichhorst.)

China. Strong reports cases from Manila and W. F. Thayer reported the first case in America. A few parasites cause no symptoms. Thymol is almost specific.

DISEASES CAUSED BY TREMATODES (DISTOMIASIS).

1. *Bilharzia disease* (endemic hæmaturia) is due to the blood fluke (Schistosoma hæmatobium). Bilharz described the disease in 1851. It is an African and Japanese affection; it has prevailed for many centuries in Egypt, where Bilharz considered that half of the lower classes were affected. It is equally frequent in Uganda. Importation has occurred into India, the Mediterranean Islands and America. Only six cases have occurred in the United States (Gunn, 1906).

Children are more predisposed than adults to infection, which occurs through the drinking water or possibly through the skin. The embryos enter the stomach, penetrate the alimentary mucosa and mature in the veins of the pelvis, spleen, mesentery, liver, kidney and bladder. The male worm measures 4 to 15 x 0.6 mm., the female 15 to 20 x 0.3 mm. The male is rolled up like a leaf, forming a groove in which the female lies during copulation. Many ova are found in the urine; they are oval, carry a spine and contain a ciliated embryo. They die in the urine in a day but live for some time in water, where their life history is still imperfectly understood.

Hæmaturia is the most common and often the only symptom; it may be macro- or microscopic and passes chiefly at the end of urination; examination shows many ova and eosinophiles. Cystitis occurs in severe infection. Stones are not uncommon, particularly in Canton, China. Suppuration may develop fistulæ. These clinical symptoms result from massive accumulation of ova in the submucosa,

which leads to papillomatous hyperplasia, hemorrhage, suppuration, necrosis, salt deposits (calculi) and rarely to malignant degeneration. Similar changes in the rectum cause pile-like papillomata, dysenteric evacuations and tenesmus. As complications, pyelitis, prostatitis, urinary fistulæ and changes in the uterus, vagina, seminal vesicles and renal pelvis, resembling those in the bladder, may develop. The eosinophiles are increased to 10 to 50 per cent.

Treatment.—Change of climate is advisable to avoid infection. The parasites usually die, though slowly, and as no local or systemic remedies modify their life history, treatment must be expectant.

2. *Lung flukes* cause the *endemic hæmoptysis* which is observed chiefly in North China, Corea, Japan, the Philippines and Formosa. The parasite is named Distoma pulmonale (Paragonimus Westermani); it measures 10 x 6 mm. and is found largely in the lungs, but sometimes in the brain and skin. In young males dyspnœa, anæmia, lung induration and a chronic cough develop with bloody or prune-juice sputum, in which are found blood, dark brown ova, eosinophile cells and Charcot-Leyden crystals. The hemorrhages, which are sometimes profuse, result from small infarcts and cysts of ova in the subpleural lung tissue. Epilepsy and hemiplegia may result from brain embolism. The course is usually favorable, though chronic, and is not influenced by treatment.

3. *Liver flukes* (Fasciola hepatica, Opisthorchis siensis and at least four other species) enter like the above forms through contaminated water. The fluke is found in the upper small intestine and the biliary or pancreatic duct. It is found in Japan and certain provinces show a mortality of 20 to 72 per cent. (Bälz and Inouye). The liver is enlarged and tender; icterus results from biliary stasis, for the parasites lie in the walls of the biliary ducts and gall-bladder; diarrhœa develops, and the dejections contain ova and blood; anæmia, ascites, anasarca, adynamia and emaciation develop and death invariably results. There is no treatment beyond the symptomatic support of the heart. Prophylaxis: avoidance of drinking or of swimming in canal water, and thorough cooking of fish and mussels have stamped out the plague in one Japanese province (Inouye).

DISEASES CAUSED BY PROTOZOA.

I. Malaria and Amœbic Dysentery (*v.* Acute Infections).

II. The Trypanosoma.—This is a protozoön found by Gruby (1843) in frogs, Döflein (1845) in rats, and later in many other animals and in man (Neprue, 1890–98). There are many species. It is leech-shaped; its body is granular, measures 13 to 25, by 2 to 4μ and contains a nucleus and micronucleus (centrosome); on one side and attached to an "undulating membrane" is a solitary flagellum by which the parasite moves. In cool and moist hanging-drop preparations they may live for a month and a half. Novy and McNeal cultivated the T. Lewis and Brucei on agar and defibrinated blood.

Infection is carried by the tsetse-fly, flea, louse, mosquito and ticks. In man the two clinical forms (*v. i.*) of trypanosomiasis are conveyed by the tsetse-fly (Glossina palpalis) or possibly by bites from infected rats. Animals may be infected during coitus, by subcutaneous inoculation or by the stomach.

1. *Trypanosoma fever* was first described by Nepreu. The atrium may be present as an inflamed bite or the history of a bite may be obtained. Parasites in the *blood* may cause no symptoms; they occur in the plasma and not in the corpuscles; some anæmia is present and the large lymphocytes are increased in number (up to 20 per cent.), but the eosinophiles show no increase. Most parasites are found in the *lymph glands* which *are constantly enlarged. Fever* is irregular, subcontinuous or remittent, and lasts from three days to three weeks. An erythematous eruption is almost constant. Œdema, particularly of the lower lids, weak and rapid pulse, prostration, splenic tumor and enlargement of the lymph glands which contain the parasite usually occur. In *treatment,* arsenic and trypan red are apparently most helpful, while quinine is without effect. Careful feeding and cardiac stimulation are indicated. The patient, as is the case in yellow fever, should be so screened that flies can neither convey fresh infection to him nor bite him and thus infect others.

2. *Sleeping sickness* or *African lethargy,* which is endemic in Central Africa, is due to localization of the same trypanosoma parasite in the nervous system, causing a diffuse meningo-encephalomyelitis (Mott). Negroes are chiefly affected but Caucasians may contract the disease.

The incubation is long and may possibly cover years. Incipient symptoms are those of the trypanosoma fever (*v. s.*) or less often there are preliminary psychical phenomena, as epileptiform convulsions, melancholia or mania. Somnolence develops, from which at first the patient can be aroused, but which later develops into profound lethargy. The parasite is found in the blood in 92 per cent. (Bruce) and in 60 per cent. of cases in the cerebrospinal fluid when withdrawn by lumbar puncture (Castellani), or in 100 per cent. (Bruce). Malnutrition, decubitus and sometimes secondary infections develop, and after the convulsions deepen into coma the patient dies. The course lasts from months to several years. Treatment is unavailing. More than a quarter of a million people died in Uganda in the last few years from this affection.

III. Tropical Splenomegaly.—Kala Azar, Assam Fever or Dumdum Fever, endemic in the eastern hemisphere and India, is caused by a protozoön and is characterized by fever, great splenomegaly and emaciation, usually terminating fatally.

Etiology.—In 1903 Leischman described the parasite, found in the blood, spleen, liver and bone-marrow. It is 2 to 4μ in diameter, oval and contains two characteristic chromatin masses. It is not certain whether the organism is a piroplasma, trypanosome or flagellate. Rogers cultivated it and Patton found it in bedbugs.

Symptoms.—The disease begins with *fever,* which is oftenest remittent, rising two or three times daily; it recurs. Marked *emaciation* develops with abdominal tenderness, nervous toxæmia, neuritic or arthralgic pains and various hemorrhages. The *skin* is *dark,* even icteric, and pigmented. The *spleen* is greatly enlarged, tender and is crowded with parasites. The *liver* becomes swollen and tender from perihepatitis, is packed with parasites and frequently becomes cirrhotic. There is *secondary anæmia;* there is relative lymphocytosis, usually with leucopœnia. The *course* averages 6 to 9 months and the mortality is 96 per cent.

Diagnosis.—The blood findings are positive in 75 per cent. and the parasites are found constantly and in all stages by splenic puncture (which, however, may entail hemorrhage or rupture).

Therapy.—Quinine seems to reduce the mortality to 75 per cent.

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